THE SCIENCE *of* FIRST AID

THE THEORETICAL AND EXPERIMENTAL BASIS

OF MODERN FIRST AID PRACTICE

ST JOHN AMBULANCE AUSTRALIA

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Canberra Avenue, Forrest ACT 2603

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FOREWORD

St John Ambulance is a charitable organisation, with branches in many countries, dedicated to the relief of persons in sickness, distress, suffering or danger. For many of us, the familiar image of St John is that of the uniformed volunteer attending a football match or other public event to offer first aid to the injured, free of charge. St John also provides first aid training to the public, publishes first aid manuals, sells first aid kits, conducts an ophthalmic hospital for a deprived community overseas, and provides a volunteer community care service for the elderly, the housebound and others who need the kind of special help that a dedicated volunteer can give.

St John has always prided itself on the technical standards of its first aid procedures and courses. These standards have been derived from, and maintained through, the expertise of its highly qualified medical advisers. This book, reflecting as it does the intellectual standards that underpin the work of St John, is a new venture of particular significance for all who are interested in the advancement of scientific thought as it relates to the principles and practice of pre-hospital emergency care.

The subject of the book is not first aid itself, but rather its theoretical and experimental foundations. Each chapter deals with a different aspect of first aid, and each is written by an expert in the appropriate field of medicine or science. Thus the chapters on heat stress and cold injury include coverage of the relevant physics and biochemistry, and that on lightning strike and electrocution includes coverage of the physics of electricity as well as the pathophysiology of its effect on the human body.

While uncompromising in its intellectual standards, and containing much that will interest and inform academic and medical readers, *The Science of First Aid* is at the same time very approachable by all with an interest in the subject, whether they be paramedics, ambulance officers, volunteer first aiders, or for that matter anyone who has an interest in knowing how their body works and exactly why it makes sense to care for it in certain ways in an emergency.

P.G.F. Henderson, AC
Chancellor
St John Ambulance Australia

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PREFACE

First aid is both a science and an art. The injured victim and the acutely ill patient present themselves to the first aider in an infinite variety of guises, in an infinite variety of circumstances, and with an infinite variety of potential outcomes. No first aid course of instruction and no first aid manual can ever hope to cover this great variation of countless different scenarios in which first aiders find themselves in real life.

For these reasons, the doctrines of first aid are encapsulated into a series of drills. These drills comprise standard operating procedures which are the official policies of the world's great teaching and co-ordinating bodies, such as the Order of St John, the American Heart Association, the European Resuscitation Council, the Royal Life Saving Society and the World Life Saving Association. It is in the interests of all to have common doctrines and common techniques, universally agreed upon, with their basis built on a core of common underlying science. At an international level, such common standards have not yet been achieved but may well prove to be within reach by the beginning of the twenty-first century.

First aid drills, or standard operating procedures, are designed so that the training of large numbers of people can be achieved. "Every parent and every workmate a first aider!" could well be seen as a basic rallying call for St John Ambulance internationally. At present, very few countries in the world have more than 5 per cent of their population trained in basic first aid skills. To reach out to a wider group in the national and international community necessitates this absolute adherence to drills and standard procedures, taught in common by all first aid teaching bodies. Of necessity, both the technical "hands-on" skills themselves and the teaching methods used must be didactic and uncomplicated. This approach is inescapable, but it brings with it a simplicity and a dogmatism which is unsatisfying for the experienced first aider, paramedic, doctor and nurse. A corollary is that many professionals – volunteers and paid ambulance officers alike – desire to know and understand the clinical science which underpins both the skill and the art of first aid.

Why do we compress the chest of the apparently dead heart attack victim at a certain rate? Why do we replace the avulsed tooth of a footballer back into its socket immediately? Why do we remove gravel and dirt from a skin wound? Why do we sometimes use ipecacuanha syrup to induce vomiting in the poisoned child, rather than tickle their throat? Why do we douse a box jellyfish sting with vinegar? Effective first aid doctrine needs always to be based upon soundly researched biological and clinical fact and, for successful "hands-on" first aid practice the science of first aid must remain an indispensable "elder partner" to the art.

A simple first aid technique, taught in the classroom and practised in real life, is often the end result of many years of clinical research, sometimes the product of high-quality experimentation and always the outcome of decades of published field experience. It is this background which is so helpful to the skilled first aider, and which makes the discipline of first aid so exciting and the practice of first aid a true "profession", in the best sense of the word.

This book is a compilation of "the story behind the drill". It will help to explain why current doctrine enshrines certain principles and promotes specific techniques to the exclusion of others. It will provide the experienced volunteer and the professional alike with new insights into the underlying science on which current practice is based.

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Too much of the doctrine of first aid has come in the past from the distillation of personal experience and personal belief, rather than from objective experiment. Much of the dogmatic treatment of the past, and some of the present, is of necessity still majority opinion or dogma. For this reason, it is not surprising that controversies abound, and that there exist quite significant and sometimes fiercely held differences of professional opinion about the options available to the first aider. Major international differences in policy also exist. This book, by directing attention to some of the underlying science, may help to reduce these differences.

First aid is not a static subject but a constantly evolving discipline. As new research is undertaken and as new experience is published, so the doctrines of the future will change. Advances in physiology, pathology, medicine, surgery, technology and retrieval medicine underpin the changing theory of first aid; and the disciplines of systems analysis, psychology, educational theory and managerial expertise weld the clinical sciences into a practical drill which becomes the art of the first aider.

All experienced paramedics and first aiders would wish to understand the rationale for the drills which they employ in real life situations. Indeed they must if innovative advances in the art and practice of first aid are to occur. They also need a source of reference for this purpose. That is the essence of this book.

Professor John Pearn
Director of Training
St John Ambulance Australia
1996

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PART

I

THE LOGISTICS *of* FIRST AID

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CHAPTER ONE

ACCIDENT SITE CONTROL

Ole Juul and John Field

Life-threatening injury involving an individual, or trauma involving multiple victims, necessitates control of the accident site itself. Such control protects the victim or victims from further injury, and protects the first aider and other care givers from injury. It also facilitates access to the victim(s), and allows the processes of rescue, extraction, on-site resuscitation and stabilisation to be carried out by the best available experts. It facilitates triage and the transport of the victim(s) to hospital, and preserves the dignity of helpless victims at a time when they are most vulnerable.

Control of the accident scene is the first of the basic skills which first aiders must master. Accident site control is of particular importance in multiple or mass casualty situations.

With good accident site control, much can be achieved, and lives saved, even in the face of apparently impossible odds. The worst catastrophe in this century was the Tangshan earthquake, on the north east coast of China, on 28 July, 1976.

> *Tangshan was an industrial city with a population of 1.06 million before the catastrophe. The epicentre was within the city proper. All the houses fell down in a few seconds. In total, 242,769 persons died, an additional 164,851 were injured, and more than four thousand children orphaned. Immediately after the earthquake, the city was paralysed. All the city's facilities were destroyed and transportation, post-office, telecommunications and water supplies were completely immobilised. However, a local command was organised in the city. One hundred thousand troops, about twenty thousand medical workers, and four thousand electricians, engineers and skilled workers were sent to the city. Then a planned and well organised disaster relief organisation was carried out in an orderly manner. Nearly one half of about 600,000 people who were buried underneath the rubble were rescued by mutual help. (Dr C.Y. Sheng, Postgraduate Medical College, Beijing, in* Journal of Trauma 1987; **27:** *1130–1135.)*

Accident sites involving two or more victims quickly become congested. Those involving multiple casualties and true mass disasters require the presence of large numbers of trained first aiders, people trained in resuscitation skills, and doctors who can work as a team; and it must be kept in mind that most of the medical staff who are summoned to a multiple trauma site have never attended a major accident before.

Irrespective of the training or experience of the specialists involved, all who find themselves at an accident site must adhere to one primary rule. That rule, encompassed in all first aid training, teaches that "Danger" is the first theme that must be addressed in the assessment of any casualty victim. Further risk to the casualty and superimposed danger to both rescuers and first aiders have been the cause of many tragedies.

Most secondary threats to the victim and risks to the rescuer come from high-energy sources, and most involve a repetition of the primary incident. A road-accident victim on the road is occasionally run over a second time. A first aider leaning over a prostrate victim on the edge of the carriageway is occasionally run down, especially if no sentinels have been placed to warn oncoming traffic, if visibility is bad, or if the speed of

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vehicles is such that they cannot stop in time. Electrical hazards which have killed one victim also tragically kill would-be rescuers if the accident site is not controlled as a first step in management. Snakebite victims are occasionally joined by their would-be first aiders if the primacy of accident site control, as the first step in first aid intervention, is not honoured.

Some examples of practical risks, taken from the international literature on the subject, include those listed in Table 1.1.

In the management of the overall situation at the accident site, the protection of patients from natural environmental hazards is also easily overlooked. This may result in further injury or exacerbation of existing injuries. Table 1.2 summarises some of these environmental situations and risks.

Many of the action-plan checklists of the world's first aid teaching bodies use the St John Ambulance acronym "DRABC" or something very similar. This is a mnemonic, or simple memory aid, for the sequential assessment and management of the victim. Its components are:

Table 1.1 Examples of practical risks to first aiders and rescuers

Hazard	Risk	Protection
Motor vehicles	Casualty run over a second or multiple times. Rescuers and first aiders run over.	Move the victim from the carriageway. Post sentinels with flags. Spotlight accident site.
Motor cycles, machinery, factory chemicals and equipment	Secondary burns from exhaust pipe as victim extracted from wreck. Burns to first aiders.	Awareness of risk.
Industrial	Secondary poisoning or burns to rescuers or first aiders.	Awareness of risk. Protective clothing.
Confined spaces, sewers, pipes, industrial tanks	Asphyxiation of rescuers.	Awareness of risk. Safety ropes on rescuers. "Buddy" systems for rescue teams.
Terrorist bombs	Secondary detonation with first blast as the lure.	Awareness of risk. Co-ordinated site assessment for hiding places.
Agricultural or industrial chemicals	Contamination of rescuer from victim's clothes or body.	Awareness of risk. Removal of contaminated clothing. Masks and respirators.
Dangerous animals, violent men and women	Rescuer or first aider also attacked. Gunshot wounds to police, firemen, first aiders.	Awareness of risk. Physical protection. Multiple co-ordinated personnel.
Water	Rescuers also drowned.	Use of safety lines, "throw and reach".
Fire	Rescuers and first aiders burnt, trapped, overcome by smoke and fumes, crushed by falling roofs and walls.	Safety ropes on rescuers. Breathing apparatus. Protective clothing. "Buddy" systems.

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- *Danger* (to the casualty or the first aider),
- *Response* (of the casualty to stimulation challenge),
- *Airway* (its patency, ie openness),
- *Breathing*, and
- *Circulation*

The first of these, the "D" for Danger, is the fundamental aide-memoire for accident site control. This implies

- taking control of the accident site,
- assessing the danger, and
- protecting victim, rescuer and first aiders.

Many accident scenes progress to disarray, and valuable time is lost, because nobody takes the time to organise the situation. Too many people think only of first assisting the injured without realising that this may in fact retard definitive treatment downstream. The first person at an incident must take charge. Do not make the mistake of automatically rushing to the assistance of some of the injured and hope that someone else notifies the emergency services. Time wasted in making initial contact may delay professional assistance or the arrival of specialised services which in the long term may do the most good for the most victims.

PRINCIPLES OF SITE CONTROL

The general principles for controlling an accident site are the same, irrespective of the nature or extent of the incident. The following principles are axiomatic.

- All activity should centre upon or relate to the injured person(s).
- At the site of an incident *all* have an important role in the prevention of further injury and promotion of recovery. There is no room for professional jealousy, competition, or demarcation issues if this fundamental aim is to be achieved. One's role may change from controller to slave as time passes. The ability to function in either role, sequentially, is an essential skill of the effective first aider. Give and expect co-operation.
- Do not take unnecessary risks which may injure you or others.
- If road trauma is involved, move unnecessary vehicles well away to allow room for essential vehicles, equipment and personnel.

The principles of site control can be portrayed in a flowsheet called a Site Control Plan (Figure 1.1). You, as

Table 1.2 Some environmental situations and risks

Environment	Risk	Protection
Rain	Hypothermia. Loss of morale.	Raincoat, plastic sheets, tarpaulins, tents, caves, rescue vehicles.
Wind	Hypothermia. Dehydration. Loss of morale.	Windproof material, eg plastic sheets, survival holes, caves, rescue vehicles.
Sun	Sunburn. Dehydration. Hyperthermia.	Shade, reflective awnings, eg white sheets. Do not forget face and eyes.
Cold	Hypothermia.	Blankets, towels, extra clothing, hot water bottles, survival holes, caves, vehicles.
Bystanders	Dignity, modesty.	Move bystanders and protect patient, however, do not cover wounds with sheets until they have been assessed and dressed.

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the first professional on the accident scene, can achieve the co-operation of all involved and thus facilitate treatment. Good organisation and adherence to the general plan can save lives and prevent further injury. Whether one or one thousand people are involved, the principles remain the same. The differences lie in the available time and resources.

With respect to this general plan, most volunteer first aiders will be involved with Steps 1 to 6. Steps 7 to 10 are mainly applicable to salaried emergency service workers, although the first aider may be required to assist.

BASIC STEPS IN ACCIDENT SITE CONTROL

1 Taking control

Being able to take control is a skill which is natural for some, and can be learned by all. It is as important a skill as cardiopulmonary resuscitation or bandaging. Taking control can be difficult because of the confusion at the scene of an accident. There may be people screaming for help who are not injured. Others who are silent may need considerable assistance. Bystanders may mill around not knowing what to do and there will be others in a state of panic. Therefore, it is essential to establish

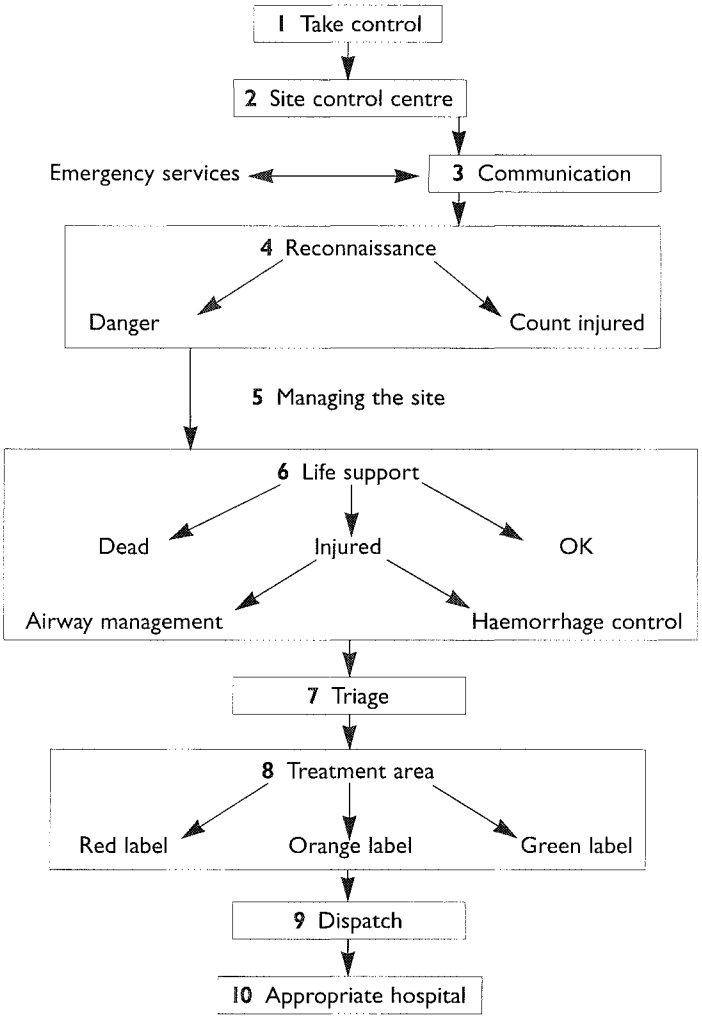


Figure 1.1 *Site control plan*

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control. As a trained first aider, whether you be a doctor or ambulance officer, volunteer first aider or fireman, you must assume control of the situation and organise others to assist you. Remember that you may be much more effective by managing the entire situation than by helping only one person.

After taking control of the situation you will need to enlist the help of bystanders. This involves assignment of tasks in the following areas to those available.

- Communication
- Reconnaissance
- Field triage and urgent treatment particularly for haemorrhage and airway problems
- Securing the area from onlookers
- Clearing the road access for emergency vehicles

2 Establishing a site control centre

In the initial stages of an incident, establish a site control centre as near as possible to the incident, taking due regard of safety aspects. The important things to remember are as follows.

- Do not become a victim yourself.
- If the scene happens to involve hazardous fumes or the possibility of gas or petrol spillage, establish the control site upwind. This is particularly the case where smoke is involved.
- There should be no smoking anywhere near the scene.
- The site control centre should be on a vantage point which should
  - i be elevated for best communication,
  - ii have convenient access for emergency services,
  - iii provide visual access to the whole site, and
  - iv have easy access to and from the scene.
- Alert emergency services.
- Establish a communication base.
- Move all onlookers and their vehicles away from the scene; erect perimeter tapes if practical.

Site control is the key to success in handling an incident (Figures 1.2–1.5). The site controller must remain in position as a focus for activity and co-ordination. All bystanders and helpers must know exactly where the site control centre is located. As soon as the emergency services arrive they may utilise the same control point.

If at all possible use a vehicle which has some form of communication. This may be a bystander's vehicle with a mobile phone. Again if possible, mark the vehicle as "Site Control". Some form of flashing light is probably the best form of identification. As soon as the emergency services arrive they will establish site identification and thus facilitate site control reporting.

When emergency services do arrive they will almost certainly take over control. That is their job. But they may require assistance from you, as you will have a more intimate knowledge of what has gone before. If you have put into effect a manageable plan, little time will be lost in effecting an efficient change of control.

3 Communication

Communication with emergency services is essential. The sooner this can be effected the greater the chance of saving time. Remember, time saved means lives saved.

Many private cars have mobile telephones or have two-way radios, the majority of interstate transport vehicles have CB (Citizen Band) radios, taxi cabs quite often have both mobile phones and radios, and all courier vehicles have two-way radios. If a bystander has a vehicle fitted with some form of communication, use it immediately. Otherwise send someone to the nearest phone to contact emergency services. Take any opportunity to hail a vehicle with some form of communication. Once communication has been established from the site keep the lines of communication open to the emergency services. Proper communication and precise information can reduce risks to responding emergency services. For example, there may or may not be immediate danger from fire. This should be communicated so that the fire services may respond appropriately.

4 Reconnaissance

This phase of site control is one of the most important and yet frustrating. The first and natural reaction of a first aider is to rush to the assistance of the injured. Unfortunately, this response may in fact exacerbate problems rather than alleviate them. Careful reconnaissance allows the first responder to provide an initial

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Figure 1.2 *Accident site control. The principles of accident site control apply whether first aiders, ambulance officers or police officers are helping with one victim or many. This scene shows some principles of site control where eight severely injured patients have been extracted from a two-vehicle high speed collision. The ambulance and response vehicles are strategically parked to protect the incident scene. Selected bystanders are pressed into service (far left). The general public is kept clear, and professional officers of three services (ambulance, fire and police) are identified by uniform or tabard. (Photograph courtesy of the Ambulance Service of New South Wales.)*

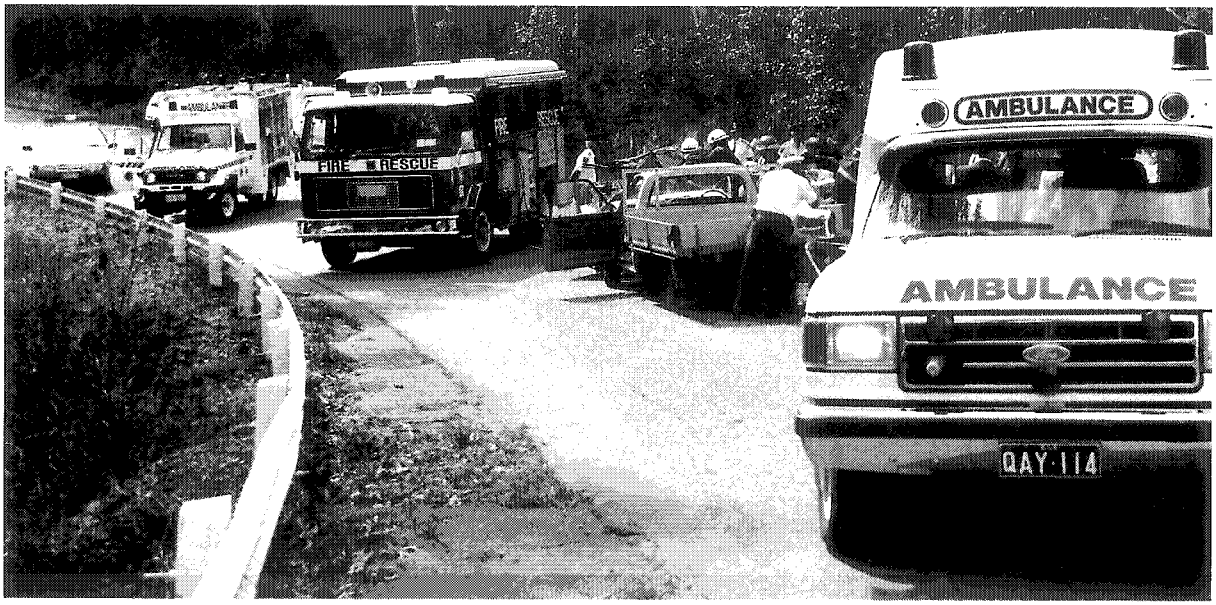


Figure 1.3 *Accident site control. This scene portrays some correct principles of accident site control. The emergency vehicles have been sited to protect the victims trapped in a wrecked motor vehicle. All accident site activity is centred upon the injured persons and their welfare. (Photograph courtesy of Mr Miles Kemp of the Queensland Ambulance Service.)*

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Figure 1.4 *Accident site control. This scene is re-enacted every minute somewhere on the roads of the world. A gravely injured victim is being extracted from his wrecked motor vehicle by the most highly trained rescuers available. Other steps (out of picture) have been taken. The risk of sudden fuel explosion has been lessened. The site controller has kept crowds back to give the rescuers clear space in which to work. The victim and the first aiders are protected from further injury, and the dignity of the helpless victim is preserved. (Photograph courtesy of Mr Miles Kemp of the Queensland Ambulance Service.)*

survey of the situation and subsequent continued reconnaissance will make sure that nothing is overlooked. This continued survey is of particular significance as sometimes new dangers to helpers can develop. A number of bystanders may be required for this task depending upon the severity of the incident. Remember that, for example, in the case of a multiple motor vehicle accident people may be thrown from a vehicle, another vehicle may be a hundred metres down an embankment and another may be further on down the road. The task of reconnaissance requires that nothing is overlooked and that as far as possible the situation should be monitored constantly.

The reconnaissance phase of an incident may be divided into two parts.

a *Danger* Identifying any danger(s) inherent in the situation.

b *Count* Making a count of all involved in the incident, both injured and uninjured.

Each incident has its own specific problems and requirements. Some examples follow.

Road accident

a Make the scene as safe as possible.

b Position bystanders to warn approaching vehicles. These sentinels must be far enough away to enable a speeding vehicle to stop.

c If a vehicle is parked on the wrong side of the road, use emergency accident lights to warn other vehicles, and make sure that its headlights are turned off.

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Figure 1.5 *Accident site control. Accident site control at this two-vehicle collision has ensured that the victims have an optimal chance of survival. Rescue vehicles are positioned to protect the victims and ambulance officers. Sentinels are posted (out of picture). Firemen are standing by (left foreground). Senior police and ambulance officers give the hands-on rescue team room to operate. (Photograph courtesy of Mr Miles Kemp of the Queensland Ambulance Service.)*

d Fumes from leaking petrol and battery acid can cause an explosion. Keep naked flames, smokers and bystanders whose services are not being used well clear of the scene.

Electricity

Electricity is particularly dangerous because it cannot be smelled, seen or heard. Quick reflexes are in no way protective if a live cable is touched. Whenever a person has an electric shock from wires outside the house call the electricity authorities immediately. Remember that all wires are live until the electricity authority has arrived and turned the power off. Only after the electricity authority has declared wires safe should the area be approached. Domestic electricity supplies are usually low voltage, 110 volts or 240 volts, but external supply lines are high voltage.

Low-voltage electric shock usually occurs when various electrical appliances in the home become faulty or are used in dangerous situations. It is quite often the case that electrical appliances are used in wet conditions – heaters, hair dryers, and so on have fallen into the bath and electrocuted people. People have been known to push a knife into the electric toaster when it is on.

a With appliances, turn off the switch and immediately disconnect the lead.

b If it is not possible to turn off the switch without danger, turn off the main power supply at the meter board and remove all fuses (or turn off each circuit breaker).

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c Pull the victim away by grasping clothes, providing they are dry. Take great care to avoid skin contact. Approved rubber insulation gloves should be worn if at all possible.

We have to assume that all electrical wires outside the house are high voltage, simply because we do not know what impact an accident may have had on the electrical systems. For example, wires may have been dislodged and fallen across domestic wiring. Under no circumstances should you touch electrical wiring. All electrical wires are live until the electricity authorities declare them safe.

a Where there is any involvement with electrical mains call the electricity authority immediately in all cases.

b When high-voltage lines are lying across a vehicle this is a very dangerous situation, especially when the vehicle is still resting on the tyres. Within minutes it may catch fire. If equipment and trained officers are available, the electrical cable may be removed. Alternatively, a trapped occupant may jump clear making sure that in so doing they do not touch both car and ground simultaneously. Unless there are other life-threatening aspects of the situation, tell the occupant to remain in the vehicle and wait for the electrical authority to disconnect power and declare the situation safe.

If a special insulated rod with a hook is available and the operator is trained in its use, an attempt may be made to move the casualty or the cable appropriately. Exercise extreme caution in moving the cable as it may flick and touch the operator.

Dangerous goods

Many trucks carry dangerous goods such as flammable liquids, corrosives and explosives. Wherever dangerous goods are stored or transported and an accident occurs, the nature of hazardous materials will always complicate the situation.

Any vehicle or storage facility which has dangerous goods is obliged to display appropriate signs. In many countries where vehicles are using LP (liquified petroleum) gas an appropriate label on the number plate must be displayed. Larger vehicles, such as tankers, will display an emergency information panel (Table 1.3).

If an accident has occurred involving a vehicle carrying dangerous goods, the golden rule is to stay clear of the scene. However, if the first aider on the scene has an opportunity to read the emergency information panel, it will indicate the nature of any danger and provide useful information to be passed on to the emergency services.

Home and industry

a Identify the nature and extent of the problem.

b Check that all occupants are accounted for.

c In gas or smoke-filled rooms, make sure of adequate ventilation before entering and then enter only if you are certain there is someone who requires assistance.

d Remember that modern furnishing materials give off toxic fumes when burning.

Water safety

a Check the nature of the water or waterway.

b Check for safe access to the persons involved.

c Check for accessibility of any life saving aids such as floats or ropes.

d Remember that lives have been lost because an inexperienced swimmer or in some cases a non-swimmer has gone to the assistance of another person. Unskilled rescuers should reach with a stick or throw a rope or inflatable object.

e If a power boat is involved, remember that it is just as important to avoid a second accident as it is in the case of motor vehicle accidents.

Violent situations

a Where violence is involved in a domestic situation, a street brawl or a general assault, take care to identify who has what weapons.

b Remember that you may easily become the next victim in any violent situation.

c Try to anticipate any renewal of violence and if assisting an injured person adopt a position where you can watch the attacker and give yourself an escape route.

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Count the injured

Remember in the first instance *only* count. There are three types of patients.

a The silent – seriously injured and require urgent treatment.

b Those able to talk or even screaming – may be injured, but generally not in a life-threatening way.

c Those whose injuries are incompatible with life.

At the reconnaissance stage it is important to report findings quickly and accurately to the site controller. If enough bystanders are present and can be used, establish a field triage system with some of the bystanders appointed as markers. This will assist by developing an accurate base for organising subsequent treatment. Inaccurate information is wasteful of time and resources.

5 Managing road accident sites

Probably the most common serious incident with which the first aider will be confronted is a motor vehicle accident (Figure 1.6). It is important to understand that following a motor vehicle accident many other vehicles will arrive on the scene within the space of about ten minutes. Consider an incident where an accident has occurred involving two cars each with two occupants. One of the vehicles has veered off the road and struck a power pole after the initial collision. The resources

Table 1.3 Interpretation of Emergency Information Panel

Panel item	Action
a Chemical name	Pass on to fire service.
b United Nations number	If you pass this number to the fire service, they will be able to make the correct resources available much more quickly.
c Hazchem Code	This code also is useful for the fire service, though the information it conveys may also be derived from the United Nations number. It is important for the first aider to know that if the Hazchem Code consists of one number and two letters of the alphabet, and if the second of these letters is "E", then this "E" stands for "Evacuation". This code is used for loads such as flammable fuels, gases and explosives. If there is fire or danger of fire, urgent evacuation to at least 700 metres away is necessary.
d Emergency Services telephone number	Ring this number.
e Dangerous goods class labels include the following: Class 1 – Explosives Class 2 – Poison Gas Class 3 – Flammable Liquid Class 4 – Flammable Solids Class 5 – Oxidising Agent Class 6 – Poisons Class 7 – Radioactive Class 8 – Corrosive Class 9 – Mixed Load	Inform emergency services accordingly.
f Specialist advice	For information of emergency services.

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which may respond to such an incident could include:

- Ambulance Service — 1–3 vehicles plus a supervisor
- Fire Service — 1–2 vehicles plus a supervisor
- Police — 2–3 vehicles plus a supervisor
- Rescue — 1 vehicle
- Tow trucks — At least 2 vehicles
- Electricity — 2 vehicles
- Medical team — 1 vehicle
- Aeromedical — 1 helicopter

This crowded scenario does not include the variety of normal traffic that very soon banks up at an accident site. For accommodation of the response vehicles, it is essential to have approximately 200 metres of clear road with ready access. Hence, as far as possible have bystander vehicles cleared from the site.

6 Life support

Following the assessment for danger at the accident site, and subsequent site control, triage follows if there are multiple victims. This will lead to the placement of victims in different treatment areas.



Figure 1.6 *Managing a road accident site. This scene illustrates several aspects of the management of a road accident site. An authority figure (in uniform, left) directs traffic; bystanders provide physical protection for the victim; police (obscured, right) control the scene with other bystanders kept back (far right). (Photograph of a single motor-cycle accident, Darwin, courtesy of St John Ambulance Australia (NT) and NT News Services Ltd.)*



Figure 1.7 *Accident site control. At an accident site, the dignity and privacy of the casualty are important. The use of uniforms as symbols of authority help accident site control, impart confidence in concerned or frightened bystanders and help with the control of crowds. This photograph shows the scene at an Anzac Day ceremony in Australia. An old soldier has fainted and injured his right hand. The crowd has complete confidence in the skill of the attendant first aiders.*

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Emergency treatments on site may involve:

- Continued cardiopulmonary resuscitation
- Defibrillation
- Continued airway control
- Continued haemorrhage control
- Intra-venous fluids
- Oxygenation
- Suction
- Pain relief
- Splinting, bandaging

At a large incident the treatment area should be conducted as a field hospital, with an exit loading point.

7 Triage

(See Chapter 2.)

8 Treatment area

(See Chapters 2 and 3.)

9 Dispatch

It is possible that the site controller may have to organise patient transport to a hospital at a considerable distance from the site of the incident. In some countries this may even be in another State. In general terms, road transport is appropriate and quickest up to 50 kilometres, rotary wing transport from 50 to 150 kilometres and fixed wing response greater than 150 kilometres. Naturally, there are often specific circumstances which modify such decisions.

10 Appropriate hospital

All major cities around the world have introduced designated Trauma Centres. This service has greatly reduced the delay time which a victim must endure before they receive definitive care. It is recognised that at least 80 per cent of serious trauma cases should arrive at the Trauma Centre within an hour, for the highest long-term survival and the lowest morbidity results.

CONCLUSION

After the incident has been controlled, it is important that first responders contact the emergency services for debriefing. This may involve them in giving vital information for future actions, or it may result in their gaining deeper insights to assist in any subsequent incidents that they may attend. Accident site control is a very important skill. The responsibility borne by the controller is great. For most, even experienced health professionals, properly controlling an accident site makes great personal demands. The controller rising to the occasion in the face of such demands will lead to the saving of life and limb. They may not be aware of the personal effect of the incident on them; and appropriate debriefing can assist them in coming to terms with possible problems and putting them in touch with counselling services. Whatever the cost, good accident site control always pays off for the victims who are in the site controller's care.

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Chapter Two

TRIAGE

Gerard Fitzgerald

Making judgements about the priorities of treatment, at multi-victim accident scenes, is an inescapable part of first aid and emergency care. Wherever two or three victims are gathered together in one place, there is triage necessary. "Triage" means "sorting" (from the French verb *trier*, to sift or sort); and the prioritisation of individual victims is a special skill demanded of all first aiders. (Figure 2.1)

Triage normally applies to the victims of physical trauma, whether the scene be that of a roadside following a multi-vehicle accident, a paddock following an aircraft crash, or a beach where multiple victims have been stung by jellyfish. However, the principles of triage apply equally to acute medical situations such as a sudden outburst of food poisoning or the not infrequent eventuality of an outbreak of mass hysteria where two, twenty or even a hundred victims may be rendered helpless in the one place at the one time.

The philosophical basis for triage arises from the utilitarian philosophy of ensuring the greatest good for the greatest number.

The concept of triage is well established in the agricultural, mining and railway industries. The term was originally adopted into the English language in the eighteenth century, to describe the sorting of woollen goods, and was taken up in the nineteenth century in the coffee industry.

In first aid and emergency care, triage is the sorting of patients on the basis of their injury or illness, and other factors, into categories which determine the degree of priority for their on-going care. This involves giving scores, or affixing labels, to individual patients; and then ranking such patients accordingly. Thus in one sense triage involves each individual patient, but in real life situations involving first aiders the term has relevance particularly in multi-casualty situations.

Sorting of patients may be seen from one of two perspectives.

1 From the *individual perspective,* triage aims to ensure that the patient's illness is quickly and accurately assessed, and that the subsequent care is appropriate. Thus the process must accurately identify the urgency of the patient's need for treatment. Such accuracy is limited by the difficulties inherent in making rapid clinical decisions on the basis of superficial assessments.
2 From the *group perspective,* triage aims to categorise patient populations into subgroups which, by their nature, have significance for decisions about priorities.

The concept of sorting patients according to urgency is presumably as old as the practice of emergency care itself. However, throughout medieval times, as a result of the lack of medical expertise and the barbarity of the general environment, little attention was given to medical care for the wounded in battle – let alone any establishment of priorities for that care.

In the late eighteenth century, Baron Dominique Jean Larrey, Napoleon's Surgeon-in-Chief, took military medicine to the battlefield for the first time. His *Ambulance Volante* (Flying Ambulance) was an attempt to retrieve casualties during the battle. Thus there arrived at one time at the medical units large numbers of

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Figure 2.1 *The principles of triage. An on-road scene where five surviving victims of a multiple-vehicle collision have been collected for resuscitation, stabilisation and monitoring, pending the arrival of transport. The dead have been collected in another area as part of triage principles. (Photograph courtesy of the Ambulance Service of New South Wales.)*

patients in salvageable condition. This often overwhelming workload required sorting on principles of equity and efficiency "...else much confusion would have ensued, had I not pursued the order of dressing and arrangements observed by me in all battles".

Baron Jean Larrey's answer was to provide "methodical succour" based on medical need, so that "those who are most dangerously wounded must be tended first, entirely without regard to rank or distinction".

Today, the availability and accessibility of urgent medical care in the civilian sphere is dependent on a complex interaction of geographical, social, cultural and economic factors. The relative importance of these factors has changed with time, thus varying the necessity for, and influence of, triage.

Gaining access to medical care involves a series of decision-making steps beginning with patients themselves and their immediate contacts, and ending with definitive treatment. Many variables are involved in each stage of the process.

First, the need for triage may be thrust, involuntarily but with compelling force, on the first responder to a multi-casualty scene. In such situations, triage requires courage, and informed triage requires a knowledge at least of basic first aid. Improvements in communication and transportation technology have changed the loneliness of this initial triage. In rural Australia, for example, the importance of personal triage decisions has been reduced by the availability of telephones, radios and satellites, as well as access to aerial evacuation.

Secondly, society has changed in its attitudes and values. Changes in attitudes to individual worth have resulted in a society which is prepared to spend more on the maintenance of life.

Thirdly, there have been changes over time in the attitudes of para-medical professionals. Clinical and technological improvements have had an effect on patient prognosis while improvements in professional education, standards and status have raised not only the level of performance of first aiders but also the expectations of patients.

The American College of Emergency Physicians (ACEP) has defined Emergency Medicine in functional terms: "Emergency health care exists for the benefit of the patient or family who perceive a need for emergency care". The patients, however, often have little idea what constitutes an emergency, and there have been extensive efforts to quantify the level of illness so as to improve the identification of emergencies.

Measuring patients' illnesses has indeed become one of the obsessions of recent epidemiological research, as we try to come to terms with a research environment which is difficult to control. Measurement of illness is an attempt to assign a mathematical score to a patient's illness. This enables the level of that patient's illness to be compared critically with that of other patients, or with that of the same patient at other times.

There are a number of severity scores which have

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been developed to enable comparisons of trauma patient outcome, to allow collection of basic epidemiological trauma data, and to assess pre-hospital and inter-hospital triage and the effectiveness of patient management. Various different severity scales have been developed and they can be grouped into three broad types – (a) physiological, (b) anatomical and (c) biochemical/haematological.

The physiological scales assess the response to injury by monitoring the patient's vital signs and subsequently correlating them with mortality. The Glasgow Coma Scale, for example, reliably assesses the degree of coma in patients with cranio-cerebral injuries. It can be used to assess brain function, brain damage and patient's progress, being based on three behavioural responses: eye-opening, best verbal response, and best motor response. It is excellent for pre-hospital triage of casualties and for the subsequent assessment of levels of consciousness following admission to hospital.

There are a number of different anatomical scores, and these include the Abbreviated Injury Scale (AIS), which was one of the first anatomical injury scales developed to rate and compare injuries sustained in motor vehicle accidents. The original Abbreviated Injury Scale was drawn up in 1983, and revised in 1985 to document more fully the anatomical and pathological effects of trauma injury, not only from blunt trauma but also from penetrating injuries in all body regions. Unfortunately, it did not take account of multiple injuries, and hence the Injury Severity Score (ISS) was developed. The latter is calculated by summing the square of Abbreviated Injury Scale scores for the three most significant injuries suffered in different body regions. It is a fairly reliable predictor of mortality and a valuable tool in retrospective reviews of injuries, but it is of less benefit to first aiders or emergency room staff in making triage decisions for salvageable casualties.

The great variety of trauma severity scales arises from the needs of different trauma centres, with their different styles and priorities. All paramedics and emergency physicians and surgeons should be able to use the Glasgow Coma Scale and one of the more comprehensive scales such as the AIS. In the case of multiple casualties, the use of these objective measures will enable the most effective allocation of priorities based on the needs of the patients and the available resources, and will also assist in prognosis of outcome, clinical monitoring, health care evaluation, epidemiological research and cost–benefit analysis.

Central to the measurement of illness for the purposes of triage is the concept of urgency. Urgency is the cornerstone of triage, in that the ultimate functional question in Emergency Medicine triage is how quickly should this patient be extricated, transported, seen, or treated.

The dictionary definition of "urgency" invokes such concepts as imperativeness, pressing importance, insistence and importunateness. In terms of medical triage, however, a number of other factors require consideration. First, there are a range of clinical factors that impact on urgency, including the nature of the illness or injury, its severity, its likely response to treatment, the effect of time on successful intervention and those characteristics of the patient which may alter the outcome. Secondly, there are a number of environmental factors which affect the resources available to deal with the casualties, and this too may affect decisions about urgency.

Urgency, unlike some of its components, is not an all or nothing phenomenon, nor is it constant. Rather, urgency changes within a particular patient as well as across patients with similar presentations. Further, the urgency of a patient's need for care is itself changed by medical intervention. The treated tension pneumothorax or plugged sucking chest wounds are prime examples in which the urgency of a patient can be altered dramatically by urgent intervention.

The main difficulty is in measuring the degree of urgency. This is like trying to describe the start of a race by photographing the winner. The winners are the survivors, particularly survivors with intact brains and salvaged limbs.

The key to successful triage lies in drawing a fine balance between over-triage and under-triage. Over-triage occurs when patients are assigned a level of urgency higher than that appropriate for their

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condition. Conversely, under-triage results in an urgency assessment which is less than that appropriate for the patient. In general, over-triage is an economic problem, while under-triage is a medical problem which may result in a suboptimal outcome for the patient. Initial triage assessments often have to be made in the most difficult circumstances – in the rain, or the aftermath of fires, or at multi-vehicle or aircraft crash sites. Triage is always a progressive endeavour. Initial assessments on minimal information often have a low level of accuracy.

A fundamental axiom is that triage is an endeavour that is subservient to the overall care of the patient, and is but one link in the chain which starts with site control and proceeds through rescue or extraction, assessment, triage, field management, transport, emergency medical care and definitive therapy to convalescence and cure. Therefore, triage processes should not be so complex that they become, in themselves, a source of delay for patients, with the result that the problem changes from one of waiting for medical care to one of waiting for triage. Similarly, it is important to ensure that the triage assessment does not result in an inappropriate allocation of resources, or act as a disincentive to patients to receive appropriate medical care.

Triage involves making decisions about patients which may literally be a matter of life and death. Such decisions frequently have to be made on the basis of limited information and in situations of critical urgency. It is the responsibility of those involved to ensure that the criteria used for making those decisions are in keeping with the legal parameters set by the community and respectful of the community's ethical expectations. The continuing challenge of triage is to ensure fairness and justice. The process of triage is one of rationing only if rationing is required, and only then on the basis of an allocation of waiting times.

The dilemma of triage is complicated by an erroneous belief that the required decisions are between good and bad. Rather, the decisions of triage are those between good and better.

In military and disaster triage, considerations other than medical criteria always come into play, as a result of the greater imbalance between demand and resources. Principles of ensuring the greatest good for the greatest number aim to maximise society's benefit. As well, ensuring military effectiveness and the preservation of the war effort may be ethically justifiable where the alternative of military defeat is equally unacceptable ethically or socially.

Legal aspects of triage can be considered under the broad categories of criminal and civil action. It is possible, although unlikely, that criminal action might arise from triage decisions, under charges of criminal negligence, manslaughter or assault. Such charges might be made where triage decisions have by commission or omission resulted in a less than optimal outcome for an individual patient, and where the duty of triage has been performed with gross negligence. There is no precedent for such actions to be found in the literature on the subject.

Civil action, on the other hand, may be taken against those responsible for triage decisions, either directly or through action against the organisation which has ultimate responsibility for the triage system which has been used. Such actions must fulfil the normal requirements of negligence cases whereby injury needs to be proven, a direct causal relationship needs to be demonstrated between the negligence and the injury, a duty of care must have existed, and a breach of that duty must have occurred. Action could be taken in such circumstances under Torts of Abandonment, Negligence, Assault or Breach of Contract.

While there are ample precedents in the UK and Australian judicial systems for successful actions for negligence in emergency medicine, there is no precedent of direct relevance to the process of triage. None the less, triage, involving as it does decisions of crucial importance to patients, is always potentially subject to civil action for negligence. The defence against all actions for negligence remains the same: the exercise of a degree of skill and care, reasonably concomitant with an individual's level of training and expertise, as well as being consistent with usual and normal practice in the circumstances. Triage itself is normal practice in multi-victim scenarios. Indeed, not to perform triage under such circumstances is profess-

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ionally negligent. Without triage, lives are needlessly lost, and limbs and health permanently sacrificed. The duty of care, for anyone having to act in a triage role in multi-victim situations, is for the greatest good for the greatest number. Individual care is downstream.

TRIAGE PROCESSES

The categorisation of patients may be one of two broad types, egalitarian and utilitarian.

1 *Egalitarian* approaches are those forms of selection which are based on principles of equity. Egalitarian approaches include chronological order or lottery style selections.
2 With *utilitarian* systems, individual or group criteria are used for the selection of patients. Such criteria may be economic, personal (eg age, past medical history, psychological attitudes), social or medical. The gravity and acuteness of the condition, the influence of other conditions, and the chances of recovery are relevant to such judgements.

The number of different criteria that may be relevant in making individual decisions complicates the process of triage. In any case, the triage officer is faced with a decision which is deeply significant to the patients. Triage should always be made, and if need be progressively updated, by the most experienced and senior person at the site.

Broadly speaking, the mechanisms by which individuals apply the various criteria for assessing the urgency of treatment fall into two groups – overall (subjective) and objective.

Overall (subjective) assessment

With overall assessments, triage staff use a series of often unwritten and undirected criteria, based on their own experience and knowledge, to categorise each patient individually. The aim is to assign to each patient a category based on the observer's overall view of his or her situation. Often referred to derogatorily as "subjective" assessments, these are none the less based on a complex interaction of the characteristics of the patients and their illnesses or injuries, the means by which those characteristics are communicated to the assessor, the circumstances of the assessment, and the values, experience and judgement of the assessor.

Superficially, the number of variables involved in such assessments suggests that any result will be one of almost lottery-style unpredictability. However, nowhere in the literature is there strong evidence that such overall assessments fail to achieve the administrative and clinical goals of triage. On the contrary, two major recent studies have demonstrated that overall assessments by nursing staff and ambulance officers are as accurate as any of the objective triage assessments in determining the severity of illnesses. Finally, such assessments are often more functionally meaningful, in terms of the management of the patient, than any mathematical score.

Central to the problem of assessing urgency are the attitudes of the assessor, which have been grouped into exclusionary and inclusionary. The difference between the two approaches is probably more subtle than meaningful. Those who adopt the inclusionary approach operate from the assumption that the patient should be selected unless factors may be identified which would exclude him. The exclusionary approach, on the other hand, works on the assumption that all patients should be excluded unless factors may be identified which would include them.

In terms of triage, those who use an exclusionary approach will select fewer urgent cases than those who use an inclusionary approach.

Objective assessment

Objective assessments aim to remove the subjective element, by allocation of measurable quanta to the patient's condition. Within the literature there have been few examples of successful objective categorisations. Those that have been most successful, such as the Glasgow Coma Scale and the expanded trauma score, have been of use only for aspects of the patient's condition or only for particular types of patient. The number and complexity of criteria involved in the decision-making process seriously constrain the use of

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objective assessments. Numerical scoring of individual elements of the assessment is sometimes direct (eg pulse rate), and at other times complex (eg mental state).

Triage is an evolving science. Improved methods all strive to make the decisions of sorting more reproducible and less subject to personal bias. In training, triage exercises in many parts of the world are evolving towards replacing subjective judgement with written guidelines. The following examples are some such approaches.

Recipe Approach Here injuries are identified, scores allocated to the injury components and the resultant assessment based on some mathematical interaction of the components, with the different ingredients weighted differently as in a cooking recipe.

Dictionary Approach Here lists of conditions are collected in verbal form, and the action predetermined by analysis of likely outcome, eg the Abbreviated Injury Scale mentioned earlier in this chapter.

Algorithm Approach This method uses a decision-tree approach, whereby assessment is guided by written protocol through a series of decision gates. The algorithms, designed by medical officers skilled in emergency life support, may then be applied in the field by staff such as combat medics, ambulance officers, nurses, physician assistants, or doctors.

Red Flags (Check Lists) This approach simply identifies a series of warning signs based on the history or initial assessment of the patient, which in effect "raises the alarm". Such "red flags" include historical considerations such as distance fallen, or speed of accident, or clinical considerations such as fractured first rib or shoulder tip pain.

Triage is an evolving skill, and there is clearly no ideal triage scale. Indeed assessments of objective triage scales are themselves in turn based, in many respects, on the values, attitudes and experience of the assessor.

Trauma severity indices have been extensively researched. They fall into four major groups: physiological scores, anatomical scores, mechanism-of-injury scores and mixed scores. Recent research suggests that the addition of a mechanism-of-injury factor to physiological or anatomical indices improves the sensitivity and predictive ability of field triage assessments.

There have been several attempts to characterise the features of a good scale, and from these there would appear to be general agreement on the following attributes.

Simplicity The scale should be simple to implement and to apply, requiring no additional equipment beyond that of common sense and basic emergency care knowledge and experience. It should be able to be implemented following a brief assessment and in any circumstances.

Practicality The categorisation needs to be practical within the confines of the situation. Sufficient data needs to be available to the individual performing the categorisation to enable them to make appropriate assessments.

Flexibility A good scale should have the ability to expand or contract to meet the needs of different situations. As well, it should have the flexibility to extend to disaster and military situations as well as to limited numbers of casualties. An effective scale can expand to include subsets and extra categories. The scale needs to be equally useful for single and multiply injured patients.

Meaningfulness and validity Any triage scale should imply division of patients into groups which have some significance in terms of the resources necessary to deal with those patients. As well, the descriptions should be valid in terms of correctly identifying those patients who require differing levels of resources. "Outcome validity" compares the categorisation with the patient's outcome, while "construct validity" compares the categorisation with other measures of urgency or severity.

Repeatability A triage scale should demonstrate repeatability, both by the same observer ("intra-rater

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reliability"), and between different observers ("inter-rater reliability"). Testing the repeatability and validity of a categorisation is therefore a means of determining an effective scale. Training of first aiders and ambulance officers in triage techniques, through mock-up situations, is of fundamental importance here.

Military and disaster triage

Military and disaster situations differ both functionally and conceptually from civilian patient triage. First, the patient load encountered is usually different both in terms of quantity and quality. Large numbers of patients may be encountered from a single event and may present within a short time. As well, the types of injuries, if not the urgency, may be similar (eg all burns). On the evening of 25 April, 1915 the medics and medical officers of Australia's No. 1 Casualty Clearing Station at Gallipoli were faced with an influx of 700 men all of whom had been wounded by artillery shrapnel and rifle bullets.

Secondly, the circumstances of the event may constrain the abilities of medical resources to respond in a predetermined manner. In a worst-case scenario, medical facilities themselves may be involved in the event itself. Hospitals may have been destroyed by an earthquake, resulting in a severe depletion of the ranks of skilled personnel. Or medics may themselves be affected by gastroenteritis when it is the cause of a great influx of the sick.

Finally, the social or other non-medical implications of a disaster may result in other needs taking priority over medical treatment. For example, the provision of food to starving victims of a disaster may take precedence over the provision of renal dialysis for the few with kidney failure.

As in all triage, the aim of the emergency response in every situation is to ensure the greatest good for the greatest number. With the exception of the rare overwhelming event, most major medical events should be manageable within the resources of civilian or military structures. High-quality care in such circumstances is dependent on appropriate triage, on the efficient organisation which underpins it, and on prior training.

Whilst military action places severe constraints on effective triage, at the same time military action is itself dependent on effective triage for an efficient response. The physical circumstances of war or disaster not only create personal danger for the medical responders, but also require this efficient medical response in a physically difficult environment where lighting, hygiene and ambient temperature may be poor.

At the same time, the ethical principles basic to emergency care are altered by the military circumstances. The governing principle may still be the greatest good for the greatest number, but that in turn may only be achieved by maintaining the military effort. Thus priority may have to be given, following radiation exposure for example, to those soldiers less severely exposed but potentially capable of continuing to fight.

In disasters, overwhelming numbers of patients may confront emergency resources. Unlike military medical resources, however, those resources are usually committed to less dramatic functions. This fact limits their ability either to be immediately and fully committed to the dramatic event, or to be fully committed to planning for such events. Under such circumstances, the triage response must be simple.

The most experienced senior person should be the disaster site triage officer. Ideally this person should be a medical officer, but the role may have to be assumed, at least initially, by an experienced first aider or ambulance officer. Whoever he or she may be, the triage officer must have the experience, seniority, standing and authority to co-ordinate with, and in many cases to direct, the other emergency services converging on the disaster scene. In the ideal situation, planned for in disaster training, the triage officer is a different person from the site control officer, and has different functions from those of that officer.

On site, the most critical decision may be the allocation of patients to physical areas of the site; this is referred to as "geographical triage". The marking of triage areas should be distinct, and the site well protected. It should be sited so as to provide good access to the casualties and to evacuation routes.

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Emergency teams on site should be made up of personnel who can carry out resuscitation in extremely adverse and unfamiliar conditions. Again the disaster itself is not the time to learn, and the staff should be drawn from those used to resuscitation and emergency assessment.

In the future, as in the past, there will be mass casualty disasters through fires, earthquakes, floods, aircraft and train crashes, terrorist bombs and multi-vehicle crashes. Triage will be an essential theme at all sites, if the greatest number are to be saved, and saved intact.

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CHAPTER THREE

TRANSPORT

Harry Oxer

Any transport has the potential to make sick patients sicker.

The science of patient or victim transportation is an evolving discipline. Rescue, first aid, emergency care and transport are themes which are inextricably linked. Removing an injured victim or acutely ill person to a place of shelter has always been an intrinsic part of first aid. Transporting the victims to a place of definitive treatment will demand, in the twenty-first century, an understanding of the principles not only of physiology and pathology, but also of mechanics.

HISTORICAL BACKGROUND

One of the first written records of patient transport is the biblical parable of the Good Samaritan, in which a passer-by took pity on an injured civilian, rendered first aid, organised his transport to shelter at an inn, and arranged for his further care.

A major advance in the evolution of patient transport was the belief that the victim of injury or illness should not be left to die of exposure, bleed to death, or become infected. From time immemorial (though rather later in the military context), such victims have been carried, taken, dragged or otherwise transported by friends, colleagues or retainers to someone who can shelter them and help them. In the early days often there was little to be done except good care and prayer. This often involved taking someone to an inn where they would be sheltered for a fee.

The quantum leap forward in transportation of the injured came with the innovative practices of Baron Dominique Jean Larré. His teachings in the late eighteenth century constitute one of the great milestones in the profession of emergency care.

> *I now first discovered the inconveniences to which we were subjected in moving our* ambulances *or military hospitals.* [This concept of a movable field hospital was the original meaning of the word "ambulance".] *The military regulations required that they should always be one league distant from the army. The wounded were left on the field until after engagement, and were then collected at a convenient spot, to which the ambulances, that is the military hospitals, repaired as speedily as possible; but the number of wagons interposed between them and the Army, and many other difficulties so retarded their progress, that they never arrived in less than 24 or 36 hours, so that most of the wounded died for want of assistance.*
>
> *This suggested to me the idea of constructing an ambulance in such a manner that it might afford a ready conveyance for the wounded during the battle. I was unable to carry my plan into execution until some time after. On this occasion we collected the wounded, whose number amounted to 360, in a large convent well calculated for a hospital.*
>
> *We found it impossible to bring off our wounded who fell into the power of the enemy. This misfortune induced me to propose to the General, and to the Commissioner General, who felt great solicitude for these unfortunate men, the plan of an ambulance, calculated to follow the advanced guard in the same manner as the flying artillery. My proposition was accepted, and I was authorised to construct a carriage, which I called the* flying ambulance. *I had first thought of having the wounded conveyed on horses, furnished with panniers; but experience soon convinced me of the insufficiency of this plan. I next thought of a carriage so*

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suspended, as to unite swiftness to solidity and ease.

...After having constructed the first Flying Ambulance, *I took it to Udino (in Italy), where by daily practice I taught the manoeuvres necessary to manage it. I entrusted the second to an active, zealous, and intelligent surgeon...*

These references to the need for training of the team as a whole fit well with the modern approach of combined emergency medical services systems, providing a spectrum of cover from first contact to definitive, hospital-based intervention.

The great nineteenth-century pioneer of improved standards for patient transport was the St John Ambulance organisation, based in London. The editor of the 1898 edition of the St John manual *First Aid to the Injured* noted that "the plan of carrying the patient by the arms and legs with the face downwards, commonly called the 'frog's march', must never be used, as death may ensue from this treatment".

Twenty years later, the 1918 edition of *First Aid to the Injured* was describing the improvisation of litters and the use of more sophisticated stretchers, but at this stage there is still no reference to the use of vehicles other than "wagons". In 1919, however, the manual included, under the heading "Transport", a section entitled "How to load a patient onto an Ambulance". In the 1920 edition came the next mention of an ambulance.

Many ambulances are provided with upper and lower berths. In such cases the upper berth should be loaded first, beginning on the offside.

The principles of care of the patient during transport:

a) The position assumed by the patient, or in which he has been placed, must not be disturbed unnecessarily.

b) Throughout the transport a careful watch must be kept on:

i) The general condition of the patient.

ii) Any dressings that have been applied.

c) The transport must be safe, steady and speedy.

This excellent summary, seen now with the wisdom of almost a century of hindsight, says most of what is to be said about the principles of transport of injured persons.

In the first half of the twentieth century ambulance services operated on a "swoop and scoop" principle, with one main responsibility – to get an injured patient to hospital as quickly as possible.

This strategy of "grab the patient and run" was not an entirely satisfactory one, however. The precipitate ride to the hospital itself often worsened the patient's condition, especially if – as was usually the case – the patient had not received any stabilisation before transport and was left unattended, to fend for himself, in the back of the ambulance. "Dead-on-arrival" was an all too frequent diagnosis, and in many cases death occurred – preventable death – while the patient was in transit, before he could reach medical treatment. The transportation of critically ill and injured patients to the hospital, in other words, was causing a delay in treatment that frequently resulted in further disability or death...the answer was simple yet revolutionary – bring the treatment to the patient before bringing the patient to the hospital. To make such a concept work, vehicles had to be redesigned to facilitate care *of the patient, not just transportation. Lifesaving equipment had to be added. Ambulances had to be staffed with at least two individuals and these personnel had to acquire a variety of new skills. (Nancy Caroline,* Emergency Medical Treatment, *1987.)*

Thus in the 1960s was born the concept of the ambulance as a systems platform rather than a simple litter, motor vehicle or aircraft to act as a taxi only.

By the 1970s in the United Kingdom, St John Ambulance, The British Red Cross Society, St Andrew's Ambulance Association and the coal mining industry had developed collective doctrine on the first aider's role in casualty transportation. Their published drills entitled *Handling and Transport of Injured Persons* included the following summaries.

The comfort and well being of the casualty is the first consideration. His condition must not be aggravated nor his life jeopardised by frequent handling or rough and hurried evacuation. If the casualty is seriously injured, or if he has multiple injuries, it may be better to deal with him where he lies, as much damage can be caused in removal. A first aider acting alone, or with unskilled help, may easily cause more damage trying to move the casualty than by dealing with him at the site of an accident...A sick or injured person may be removed by the following methods:

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- *Support by a single helper*
- *Hand seats and the kitchen chair carry*
- *Blanket lift*
- *Stretcher*
- *Wheeled transport – eg ambulance*
- *Air and sea transport.*

The use of more than one method may be necessary; and this will depend on:

- *The nature and severity of the injury*
- *The number of helpers and facilities available*
- *The distance to shelter*
- *The nature of the route to be travelled.*

VEHICLES

The modern road ambulance is expertly sprung, powered by a smooth, quiet, powerful motor, and provides a level of comfort and equipment which reflects space-age technology. A detailed consideration of road transport vehicles is beyond the scope of this chapter. However, in so far as first aiders may be involved in loading ambulance vehicles, acting as escorts for victims, and often being involved in multi-organisation exercises, some principles of the underlying science of road transport are covered here.

The interior of modern ambulances is fitted out with a very large range of sophisticated aids to patient care. Air conditioning, sound deadening, and provision of lighting, oxygen and other facilities are standard features which contribute to patient comfort. Monitoring and treatment facilities will be standard in all ambulances of the twenty-first century.

Helicopters have been widely promoted as ambulances. However, they still transport only a very small proportion of patients. They are often used inappropriately, just because they are available. The helicopter has a quite specific place in the movement of patients over relatively short distances, from or to otherwise inaccessible areas. However, most helicopters used for emergency medical transport are relatively small and noisy, and have limited space for equipment or medics, or facilities to care for patients.

Fixed wing medical aircraft have developed considerable sophistication, but are restricted mainly to operating from and to prepared runways. They are vital in many large countries for the longer distances transfer of patients, especially from remote areas. Other methods of transport have been used to take specialised medical teams or "paramedics" out to the scene of an accident (Figures 3.1–3.4). These have included cars, motor cycles, helicopters, hovercraft, snowmobiles, and almost any type of vehicle that can be imagined.

PATIENTS AND THEIR TRANSPORT

There are six principles of transportation of patients which apply to all forms of carriage, and in all situations. These are:

1 Positioning
2 Visibility and observation
3 Airway maintenance
4 Artificial ventilation
5 Bleeding control
6 Effects on the patient

Positioning

Appropriate positioning of patients, especially the unconscious, is both a science and an art. Unconscious patients must be in a stable side position. Travel sickness and vomiting is a high risk for victims being transported to a place of definitive care. The first aiders should endeavour to place the victim near the centre of moment of the transporting vehicle, whether this be a motor vehicle, boat or aircraft. Secondary vomiting from motion sickness is of particular risk to those who are obtunded, who have facial or neck trauma and those who have sustained eye trauma. In the latter, the raised intra-ocular pressure associated with retching can cause secondary damage to an already-damaged eye. Protecting the victim from as much noise, vibration and cold as possible is very important.

Visibility and observation

There is no substitute for the educated and involved

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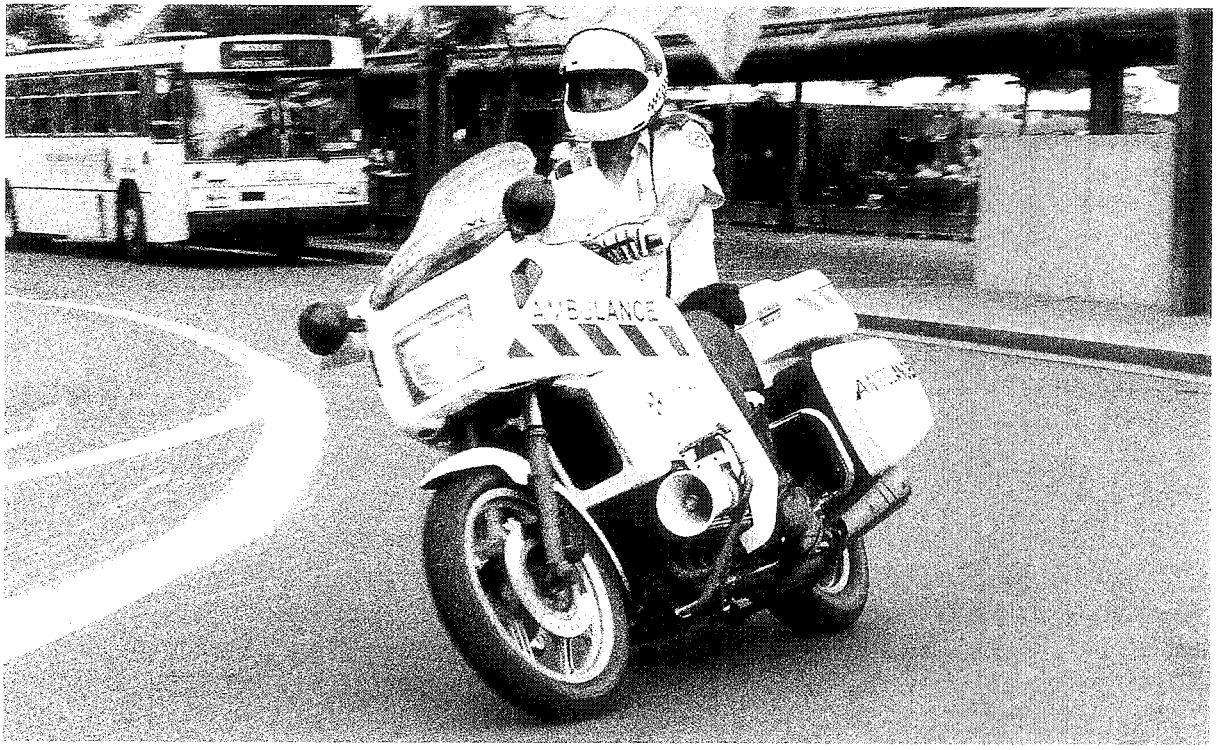


Figure 3.1 *Pre-hospital transport takes many forms. This motor-cycle paramedic of the New South Wales Ambulance Service (Sydney airport, 1994) can respond to emergencies in heavy traffic and when access to an accident site is otherwise blocked.*



Figure 3.2 *A privately operated "Citymed" ambulance, equipped with intensive care monitoring and resuscitation facilities, in Buenos Aires, Argentina.*

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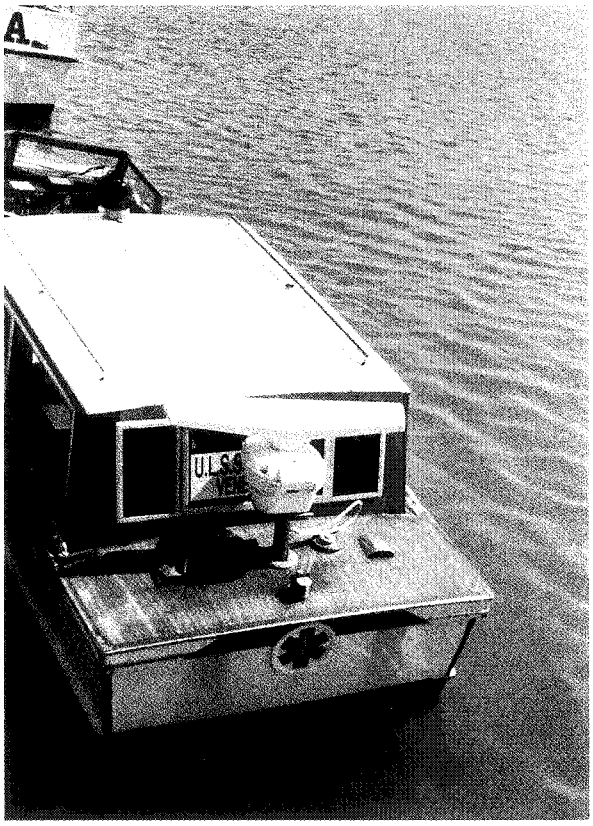


Figure 3.3 *A motor boat ambulance, fitted with radar, on the waterways of Venice, 1992.*

observer as the chief monitor. No amount of machinery and electronics can substitute for a trained and attentive attendant, though it may provide extra information for use by the attendant. Conscious casualties need the reassurance of a "hands-on" attendant beside them at all times whilst in transit.

Airway maintenance

Maintenance of a clear airway is fundamental to effective oxygenation, and indeed to survival, of a patient being transported. Various papers have extolled the virtues of one or another airway maintenance adjunct. The "gold standard" is a clear airway, enabling effective oxygenation. The means is secondary.

A number of devices have been used to assist in airway maintenance. The Guedel airway, a simple flanged oropharyngeal device, was first described as an all-rubber airway in 1933. However, Sir Frederick Hewitt in a letter to The Lancet in 1906 described a straight oropharyngeal airway of rubber, with a metal bite-block – this was shown as curved in the catalogues by 1909. Though the Guedel version became more popular, occasional Hewitt airways could be found in use until the arrival of clear plastic and disposable airways in the 1970s and 1980s. The original concept is essentially unchanged – a tube, with a reinforced area to prevent biting, which curves over into the pharynx, holding the tongue away from the posterior pharyngeal wall in its curve.

All airways need a little training to be used effectively. The endotracheal tube can offer complete protection of the airway. When such a tube with an airtight cuff is placed in the trachea, it can provide excellent, soiling-free ventilation. However, unless muscle paralysing drugs are used to facilitate intubation, it can only be placed in the dead (cardiac arrest) or the a-reflexic, that is deeply unconscious. Its use certainly makes ventilation easier and more effective.

Other airway devices have come and gone. The oesophageal obturator and its variants blocked off the oesophagus, thus allowing any inflation pressure in the pharynx to pass into the trachea. Recent devices using a similar principle include the pharyngo-tracheal airway and the Combi-Tube.

The most recent device is the laryngeal mask devised by Brain. This is a most valuable piece of equipment which has revolutionised anaesthesia in the spontaneously breathing patient. However, its air-inflatable cuff provides only a low pressure air seal and, though it has been used for ventilating non-breathing patients successfully, it does not necessarily protect the airway from reflux of gastric contents. Several studies are in progress on its use in pre-hospital care. The device is currently too expensive for routine ambulance use, but the idea is most promising.

Artificial ventilation

Ventilators for breathing the non-breathing patient are usually only used for secondary ambulance transport of the sick patient. Early devices that cycled at a

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pre-set pressure were abandoned as unsatisfactory. Flow preset, time-cycled devices such as those made by Pneupac are very popular in Europe. In the United States, Australia and New Zealand, manually triggered oxygen-powered ventilation devices have been popular. However, the rate of inflation is now recommended to be less than 40 litres per minute, to minimise inadvertent stomach inflation. (Earlier models could blow oxygen into a casualty at up to 160 litres per minute!)

Current thinking is perhaps turning back towards use of manually operated bag and valve systems, or mouth-to-mask, with suitable precautions against cross-infection.

Bleeding control

Control of external bleeding is by *pressure* and *elevation*. The tourniquet has been abandoned, except for controlling otherwise lethal bleeding when direct pressure methods have failed. The underlying principle of bleeding management is that it must be totally controlled *before* transport is commenced. Once in the air or on the water, continued or fresh bleeding is very difficult to control. In the case of land transport, if the bleeding is external, it is usually better to stop, stabilise the victim and then continue the evacuation.

Effects on the patient

The victim of illness or injury is the key player in any transport endeavour. However, this can be lost sight of in the context of high-tech equipment, a state of the art vehicle costing hundreds of thousands of dollars, the drama of sirens and the pressure of emergency. The casualty is almost invariably prone or supine, and relatively helpless. All conscious casualties being transported are frightened, and to each one the event is



Figure 3.4 *An ambulance with rear and side access, which can be used as a mobile first aid post, in Vancouver, Canada, 1993.*

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Figure 3.5 *A state of the art paramedic ambulance capable of life support monitoring and resuscitation, in Washington DC, 1988.*



Figure 3.6 *A London ambulance for non-urgent transport of sitting invalids, 1992.*

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Figure 3.7 *A ladder car as first responder in a medical emergency, Houston, Texas, 1993.*



Figure 3.8 *The problem of ambulance access and egress in unfavourable conditions. A scene on the Darwin wharves, 1994. (Photograph courtesy of NT News Services Ltd.)*

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Figure 3.9 *The interior of a Norwegian helicopter ambulance. Stavanger, Norway, 1993.*

a novel experience, with no real knowledge of what is coming; some are terrified. The act of transportation can itself cause three new complications – motion sickness, claustrophobia and the effects of vibration. The experienced first aider or paramedic anticipates the potential for these and reduces their effects. Eye contact between the first aider and casualty during loading and transport will reduce both fear and the potential for claustrophobia. Padding and the use of ear protectors will help reduce vibration effects during flight in rotary-wing aircraft, and the insistence on minimal speed (consistent with clinical urgency) will reduce the nausea and vomiting of motion sickness.

SPEED OF TRANSPORT

Speed is probably the most misunderstood of all the "needs" for patient transport. There are only small groups of patients for whom speed is necessary. These patients can be said to be "time critical" in that delay will result in a worsening of their condition.

Patients falling into this group include:

- those in whom bleeding, especially internal bleeding, cannot be controlled;
- those whose respiratory function is deteriorating in a way not able to be controlled in an ambulance; and
- those with deepening unconsciousness following head injury or intracranial problems.

Speed is a relative consideration. In practical terms, what is usually meant by "speed" is really "without undue waiting or delay".

In this latter category particularly is the question of transportation of the heart attack (coronary occlusion) victim. In this situation, recovery is optimal if thrombolytic drugs can be injected intravenously within an hour or so of the attack. However, the time-limiting factor is rarely the ambulance service. It has been found that most patients do not admit to their problem for some hours, and average times before calling an ambulance have been shown to be two to three hours. A main problem causing delay before starting thrombolytic therapy is the passing of heart attack victims from doctor to doctor before they finally arrive in the coronary care

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unit. However, in those unusual situations where the waiting for a road ambulance is prolonged, then it may be preferable to get the victim to hospital by the quickest means available, albeit in a private car. But, whatever vehicle is used, speed (as in miles or kilometres per hour) never needs to be excessive.

Professional ambulance officers have a checklist for conditions which require the so-called "swoop and scoop" approach. This philosophy has evolved following the improved in-hospital management of gross thoracic trauma, which has made possible the salvage of some of those with hitherto fatal chest lesions, provided that the victim can be transported urgently to hospital. Whilst not to be taken literally, the "swoop and scoop" concept does mean assessing the urgency of a case very early, doing only what is necessary in ambulance care to make the patient's transport safe, and then transporting them safely and urgently to the next level of care (ie a suitable hospital).

Many patients often thought by the lay public to be extremely urgent are in reality not time critical, though undue delay should be avoided. Burns may fall into this category (unless the airway has been involved), as also may spinal injuries. The spinal injury patient is better served by being properly immobilised, and then transported carefully to a spinal care centre, rather than being retrieved at speed.

Most ambulances in the developed world have a crew of two – a driver, and an attendant. These are variously skilled. In most services, both work as a team in the field, then one drives and one attends. If victims are seriously ill, especially if they require resuscitation or significant hi-tech intervention such as occurs in secondary hospital-to-hospital transfer, then a second attendant is needed.

In some countries, the ambulances involved in high-dependency transport may be staffed by physicians, as in France and Russia, or by paramedics, as in the United States and other countries. The term "paramedic" has no precise meaning. It usually denotes a trained ambulance technician able to carry out some specific interventions, such as intravenous cannulation and cannulation of the trachea. They are usually authorised to use a number of medications, from four to over sixty, administered in accordance with strict medical guidelines and protocols.

In some countries, these staff have their own specialised ambulances. Others will have one highly qualified ambulance crew member, with one less qualified assistant-driver, as a cost-effective combination. Little research has been carried out on the effect of attendant skills on victim outcome, with the exception of some work from paramedic teams in King County in the United States. This seems to show increasing survival from cardiac arrest with increasing levels of intervention sophistication.

All would agree that, except for non-intervention transport of patients, a vehicle with a crew of one is not an "ambulance".

Modern radio communications use highly sophisticated technology. However, the huge increase in equipment utilising or emitting radio frequencies has to some extent negated these advances, and radio contact is often still a problem in large cities. "Blank spots" often also occur in remote country areas, where it is not practicable to have many transmission aerials. The use of satellite technology is currently limited by expense, and by the availability of sufficient channels, but this is improving rapidly in most countries. There is a need for voice transmission to be reduced by the use, in transit, of coded buttons. In some areas computer information is transmitted and printed out in the ambulance as hard copy. On-line medical control is legally required in some countries, particularly in the United States. Theoretically advantageous, its efficacy depends greatly on the knowledge and skills of the person taking the radio calls, and they can only assess the situation through the information from the on-site ambulance staff.

Transmission of electrocardiograms by radio has been available for many years, but has had little influence on patient management or outcome. Most recently it has been used to assess a patient's suitability for pre-hospital thrombolysis, but recent work suggests that the relatively short time differentials are unlikely to affect mortality or morbidity significantly, provided that thrombolysis is commenced promptly at the receiving hospital.

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EQUIPMENT

The equipment needed for the transportation of patients, either for longer distances by hand carriage or in transportation vehicles, includes the following items.

Oxygen

The provision of oxygen is necessary for all patients who have suffered an injury or illness resulting in decreased flow of oxygen-carrying blood to any of the organs.

Splinting

Splinting is very important to immobilise fractures and serious injuries, and to prevent them from worsening due to movement and vibration during transport.

Protection

The ideal transport vehicle affords total environmental protection for the victim. Modern ambulances, ambulance snowmobiles, helicopters and fixed-wing aircraft do this. In practice, the transport of victims by hand litter, in private vehicles pressed into service, in boats and in helicopters, commonly exposes an already sick victim to cold, heat, wind and spray. The provision of extra blankets and waterproof covers requires a moment of pre-planning and extra thought.

Defibrillation

This has significantly increased brain salvage and survival in patients who can be defibrillated within 7–8 minutes, and who have either ventricular fibrillation or a non-perfusing tachycardia. By the twenty-first century, all transport vehicles and indeed many volunteer first aiders will have access to such equipment.

Medical anti-shock trousers (MAST)

The MAST (sometimes called the pneumatic anti shock garment (PASG)) is the subject of some controversy. However, it seems that there is sound evidence that the equipment has a place in controlling external bleeding below the waist, and splinting fractures of the lower extremities. It probably tamponades (controls by pressure) bleeding inside the abdomen. It is likely that it will be disadvantageous in cases of bleeding *above* the diaphragm, including head and intrathoracic injuries. However, if all else fails, the MAST suit may still be an option to attempt to preserve life until the patient reaches hospital.

Intravenous infusions

In some parts of the world these have become a legal requirement and are standard equipment in ambulance transport vehicles. However, research has not shown significant advantages from the infusing of fluids in many pre-hospital situations, and their place in the overall process is under constant review. The intravenous infusion may be used as a vehicle for administering pre-hospital medications, should they be necessary.

Medications

Up to sixty or more different medications are carried by some pre-hospital paramedical groups. Careful examination shows that few of these are used with any frequency. Recent research has failed to confirm significant benefits from the administration of most medications in the pre-hospital situation. It is likely that nebulised bronchodilators (such as salbutamol for the asthmatic patient), some form of analgesia, and a coronary vasodilator drug such as nitroglycerine provide significant pre-hospital benefits. Analgesics may be inhaled (nitrous oxide/oxygen 50–50 or methoxyflurane) or in some systems narcotic analgesics are injected intravenously.

Intubation

It is undoubted that intubation, especially of the victim of cardiac arrest who has not responded to defibrillation, will increase the ease of handling such patients during transport. It will also diminish the amount of respiratory contamination by vomitus. It is less clear whether intubation results in a significant increase in the number of survivors of cardiac arrest. There are few other groups in whom intubation can or should be performed in the field. There have been no published studies carried out to show the outcome benefits of intubation of the trachea when carried out in the field.

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MONITORING

The main advances in pre-hospital patient monitoring are the greater availability of monitor – defibrillators, including shock advisory systems, and the availability of oxygen saturation monitors. The latter can be of value in assessing the effectiveness of oxygen delivery to tissues, and will undoubtedly help in management. However, it has been pointed out that if all patients receive 100 per cent oxygen (which few currently do, in most ambulances) then only those with compromised circulatory perfusion will be identified by decreased oxygen saturation.

Automatic pulse read-outs are effective in counting the pulse. However, this data can be obtained equally well by the watch and finger, and bring the first aider closer to the patient.

Devices for regular automatic blood pressure assessment usually work beautifully in well perfused patients in a stationary vehicle. Movement artefact, or poor tissue perfusion, as in shock, make them much less reliable, just when they are needed most. As all anaesthetists and expert emergency physicians will tell you, if you doubt the number on a machine, a manual systolic blood pressure assessment with the finger on the pulse, is still the final arbiter of accuracy.

It is important to remember that only a small proportion of patients who require ambulance transport will require sophisticated monitoring, and even this should be related to its ability to provide information that will change significantly the care given, and decrease morbidity and mortality.

CONCLUSION

Transport is but one part of the continuum of care from rescue and first aid to definitive institutional care. Transport providers in this day and age are trained and well skilled in the appropriate movement of their clients. First aiders will serve the needs of their patient best by providing skilled caring first aid, by arranging for the transport system to be accessed swiftly, and by providing the transport team with as much relevant information as they possess when the team arrives. The person of first response, giving first aid in the field, may be a doctor, nurse or trained first aider. Irrespective of primary calling, the first aider on the spot forms a link in the evacuation chain. Transport from the field is the next link in patient care, and this interface requires training and the development of experience. For this reason, first aiders can best prepare to take their place in this chain of management by interface training with their colleagues who provide the ambulance transportation service.

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CHAPTER FOUR

FIRST AID IN THE AIR

Eric Donaldson

Approximately 1.5 billion passengers flew on the world's commercial airlines in 1995, and of these at least 4 million suffered sudden illness or were injured and required first aid. It is common for a jumbo-sized passenger aircraft on longer hauls to have a medical incident of some type. Skilled and experienced first aiders, as fellow passengers, are thus a vital resource to the air-travelling public.

An analysis of the medical emergencies in one year on a medium-sized international airline carrying 4 million passengers per year shows syncopal incidents are by far the most common. These are often associated with abdominal discomfort and nausea and normally respond to simple first aid measures (Table 4.1).

Table 4.1 Medical conditions involved in medical emergencies arising during air travel. Data from QANTAS international operations over one year with 4 million passengers carried. Relates to cases where a medical practitioner or a trained nurse passenger responded to a call for assistance.

Medical condition	Percentage of cases
Syncope	35
Gastro-intestinal symptom	13
Abdominal pain	4
Angina	10
Cardiac emergencies	6
Respiratory tract infection	6
Asthma	3
Allergic response	1
Anxiety	5
Trauma	4
Seizure	3
Migraine	1
Miscellaneous	9

Breathing difficulties and chest pains are not infrequent and because of their significance for underlying serious pathology are a cause of major concern. Simple measures may not be adequate and the assistance of a medical practitioner may be necessary. World-wide, the incidence of death in flight is 0.38 per million passengers carried and the majority of these deaths are due to sudden cardiac causes. On long hauls the incidence of death in flight is higher, and varies from 1 to 2.5 deaths per million passengers carried.

In spite of constant warnings to passengers and good flight attendant care, injuries occur, often as a result of falling luggage from the overhead bins and scalds from hot beverages.

FACTORS WHICH CAUSE MEDICAL PROBLEMS IN THE AIR

Prolonged relative immobility, gastro-intestinal irritability, stress associated with preparation for a flight, airline terminal bustle, barotrauma, alcohol abuse, hypoxia and dry cold air are triggers that may precipitate a medical problem during flight. Almost all can be managed optimally by trained and skilled first aiders.

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Hypoxia (lack of oxygen)

As the altitude increases and the barometric pressure falls, the partial pressure of oxygen in the air falls. Haemoglobin saturation with oxygen depends among other factors on the partial pressure of oxygen in the alveoli. The sigmoid shape of the haemoglobin dissociation curve ensures that the drop in saturation is slight to an altitude of 10 000 feet. Below this height, the effects of hypoxia on healthy people are not considered significant. Large civilian passenger aircraft are pressurised so that they maintain a pressure environment the equivalent of 6000 feet to 8000 feet above sea-level. However, although a minor fall in haemoglobin saturation may be insignificant to a healthy adult, to a patient with a compromised myocardial or cerebral circulation even a small degree of hypoxia may be catastrophic; the shocked patient who has lost much of his haemoglobin may not tolerate a further decrease in his available oxygen.

The problem may be overcome by increasing the proportion of oxygen in the inspired gas and thus the partial pressure of oxygen in the alveoli. Oxygen/air mixture may be delivered by mask.

Barotrauma

Gas expands as the pressure decreases with altitude. The human body has gas trapped in the gut and in the semi-enclosed cavities of the middle ear and paranasal sinuses. At 8000 feet a gas occupies approximately 1⅓ as much volume as it did at ground-level.

Gas in the gut is usually passed freely and only in rapid decompression episodes does it sometimes cause pain and syncope.

In the middle ear and paranasal sinuses, gas also tends to escape freely on ascent, and can be felt escaping as the "ears pop". During descent, active movements are necessary to equalise the pressures inside and outside the tympanic membrane (ie swallowing, moving the jaw, and increasing the pressure in the nasopharynx by pinching off the nose and forcibly attempting to exhale). This technique may be used to overcome unequal pressure in the sinuses.

These methods fail most often when there is inflammation of the respiratory tract due to infection or allergy. Discomfort is then followed by severe pain as the tympanic membrane is pushed in or the mucous membrane of the sinuses is stretched. As the pressure differential increases blood vessels rupture and bleed, the tympanic membrane may rupture and the sinus or middle ear may fill with blood.

Otic barotrauma is the commonest presenting symptom of aircrew due to their occupation. It also frequently occurs in passengers (1 or 2 per cent), even in pressurised aircraft, as they have a pressure differential of 200 millimetres of mercury (mm Hg) to cope with on descent. Patients with recent facial trauma should be prudent about air travel. In the case of planned acromedical evacuation, particularly those with gut obstructions, pneumothorax or penetrating eye injuries the risks of gas expansion may be high.

Some gas (usually nitrogen) remains in the abdomen for several days after operations. Asthmatic patients in acute attack have air trapped in their lungs. Travellers with tooth decay may have small pockets of air in dental cavities. The air trapped in all these cases will expand, potentially causing pressure, during flight.

Decompression sickness

Fortunately, this condition is rare in altitudes of less than 24 000 feet. It is the pilot's equivalent of the diver's "bends" and is due to bubbles of inert gas, principally nitrogen, coming out of solution in the body fluids and forming bubbles in the tissues when the partial pressure of the gas is reduced at altitude. Patients who already have decompression sickness due to flying or diving should not be transported by air unless pressure conditions can be maintained at approximately ground-level or below. Occasionally, air travellers who have been scuba-diving are unaware of this risk, and develop symptoms during flight. Reducing the cabin altitude and the administration of oxygen may lessen the symptoms.

Cold

Aircraft are potentially cold, as the external temperature decreases with altitude. The air-conditioned air at altitude is very dry. This latter feature leads to

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Figure 4.1 *First aid in the air. Access to treat a collapsed victim is a necessary first step in all first aid work. Note that in a real life incident involving the use of a defibrillator the kneeling attendants would be well clear of the casualty. (Posed photograph to illustrate this point, courtesy of QANTAS Airways Limited.)*

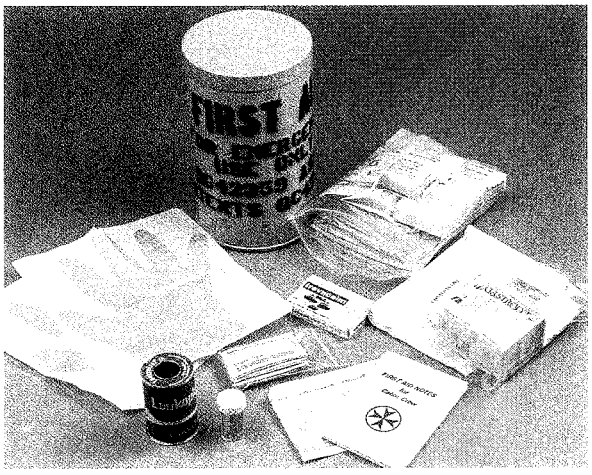


Figure 4.2 *Basic first aid kit. This kit is carried by all airlines for the general use of flight attendants or passengers who happen to be first aiders. This illustration shows the Yellow First Aid Tin with its contents displayed. 747 ("Jumbo") aircraft carry four of these Yellow Tins; 767 aircraft carry three. (Photograph courtesy of QANTAS Airways Limited.)*

evaporation which in turn causes cooling. Usually this is not a problem for air travellers, but it may trigger an asthma attack or sudden illness in the elderly, the sick or the very young. Maintenance of body temperature is a significant consideration, particularly for neonates. Many light aircraft have poor cabin heating systems and the pilot may not be able to sacrifice the power required for heating during the flight.

Additional patient considerations

As well as the risks due to altitude, there are those due to the vehicle itself. These include airsickness, the psychological stress of flight, noise, vibration, the confined area and turbulent conditions. For the experienced air traveller these are usually trivial; for others they can become overwhelming, and hysteria, psychological collapse, overbreathing with sudden tetany, and over-indulgence in alcohol may be provoked. Each of these conditions is an individual challenge to the first aider.

THE FIRST AIDER IN FLIGHT

A major problem in providing any first aid in the air is that of access to the patient.

In a large passenger aircraft, if all the seats are not occupied the flight attendants may be able to arrange for the passenger to lie across several seats, if the arm rests are movable. Recent legislation in the United States making movable arm rests mandatory in 50 per cent of seats in new large passenger aircraft could facilitate this procedure.

If the passenger is collapsed in his or her seat, an airline blanket may be used as a sling and with the help of many hands it is comparatively easy to transfer patients to the floor where they may be able to be dragged to an open area for attention (Figure 4.1).

Diversion of an aircraft from its planned route and an unscheduled landing is undesirable from the airline viewpoint. Often fuel must be dumped before landing, passengers miss connecting flights, the airline must

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Figure 4.3 *The opened emergency medical kit (physician's) carried on international airlines. Bag A contains the needles, syringes and other hardware for parenteral administration. Bag B contains instruments. Three extra hard plastic containers hold injectable and other drugs. (Photograph courtesy of QANTAS Airways Limited.)*



Figure 4.4 *On-board medical equipment. The overwing exit locker of an international aircraft, opened, showing the medical equipment therein. At left is a semi-automatic defibrillator carried on Virgin Airlines and QANTAS international flights. At centre is the standard clear plastic bag containing stethoscope and sphygmomanometer. At right is the orange-coloured, hard plastic physician's kit. (Photograph courtesy of QANTAS Airways Limited.)*

meet extra accommodation costs and extra expenditure of $100 000 is not unusual. If the patients can be adequately managed on board, it is preferable for the flight to continue. In few areas can first aid skills be more valuable or more appreciated.

The first aider or medical attendant should ask for the aircraft's first aid kit or, in the case of doctors or nurses, the emergency medical kit (Figures 4.2, 4.3 and 4.4).

The quality and sophistication of the first aid kit and emergency equipment supplied by an airline varies considerably and depends on national legislation, company attitude, litigation experience and security, among other factors. There are often different kits designed to permit different levels of response.

The basic kit may contain items for use by those with first aid training. Many airlines require their cabin attendants to hold a current St John Ambulance First Aid Certificate or equivalent, while others provide first aid training in their induction and refresher courses. A basic kit (see Figure 4.2 and contents listed in Table 4.2) contains an inhalant for barotrauma, an anti-nauseant, an electrolyte replacement, burns dressings, bandages, scissors, etc. This is typical of that carried on most passenger aircraft. Often, the more frequently used items – mild analgesics and inhalant sachets – are stored separately where they are more easily accessible. Oxygen is freely available and is very frequently used.

A kit (Figure 4.3) requiring a higher level of medical expertise is released only to a qualified practitioner or with the permission of the captain of the aircraft. It contains equipment such as a laryngoscope, syringes, drugs in tablet and injectable form (including morphine and pentazocine) and resuscitators (Tables 4.3 and 4.4). The most frequently used drugs are analgesics, bronchodilators, tranquillisers and glyceryl trinitrate. Some international airlines also carry semi-automatic defibrillators. The commonest items used from such a physician's kit are the stethoscope and sphygmomanometer.

Those airlines (such as QANTAS and Virgin Atlantic Airlines) which also carry semi-automatic defibrillators

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Table 4.2 Contents of basic first aid kit carried on all passenger airlines. Usually given to first aiders for use on board; it meets international standards developed by the Equipment Sub-committee of the International Air Transport Association, IATA

	1 Aircraft First Aid Book	
	1 contents list	
	2 questionnaires *re* gastroenteritis	To be filled in by the FSD if there is an outbreak of diarrhoea and/or vomiting on the aircraft.
Dressings	2 packets multipack Bandaids containing 20 Bandaids	For minor incidents
	2 medium or large wound dressings	For pressure dressing to control bleeding from cuts or wounds
	10 butterfly closures in plastic container	For holding together cut which may require stitching
	1 gauze bandage	Support for sprains and strains
	Melolite (non-stick dressing)	For burns
	1 card safety pins	
	Cetrimide prepacked swabs	Antiseptic swab for cuts and abrasions
Tablets	1 adult, 1 junior packet Andrumin tablets	For motion sickness (airsickness)
	1 packet Gastrolyte (10 x 4.9 g sachets)	For diarrhoea
	1 packet of inhalant sachets	For pain in the ears on descent or blocked nose
	8 plastic hand gloves	To be worn when there is contact with body fluids

Note: Tablets should not be given unless requested by the passenger.

(Figure 4.4) have flight attendants trained in their use.

In no other situation than air travel does one encounter a better illustration that first aid is a profession in its own right. The special skills required of the competent first aider are so often needed during in-flight emergencies, yet are so often found wanting in those called upon to help. In general, doctors who respond to in-flight emergencies do not practise optimal first aid techniques. A recent (1995) survey of defibrillator use during in-flight cardiac emergencies has revealed that not one of 50 doctors who offered help in these cardiac crises was currently familiar with the operation of the particular model of semi-automatic defibrillator.

The special features that characterise in-flight first aid are that almost every situation is different, and that the responding first aiders do so altruistically but in unfamiliar circumstances and with some uncertainty as to their own skills and their status with respect to the other passengers. Ethically, no trained first aider can withhold help when this is required during flight. On the other hand, response to a call for help in such circumstances requires courage and resolution. Such situations are more easily coped with if there has been prior training and rehearsal in handling the special exigencies of the casualty's needs during flight.

IN-FLIGHT FIRST AID IN THE FUTURE

Skilled first aid will become increasingly more important for all air travellers in the twenty-first century. The aircraft are likely to be larger and carry more passengers. The world's air passenger numbers are increasing by 7.4 per cent per annum and will have doubled by the year 2005.

Having in mind also that so many more of the older, younger and less fit will be flying more, there will clearly be many more medical incidents in the air. The ideal of "every air passenger a first aider", if implemented, would bring skilled help to passengers who need it, reduce suffering and save life and limb in the future.

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Table 4.3 Contents of the physicians kit (medical emergency kit) carried on international aircraft

Contents list
Forms to be completed by attending doctor

1 Laerdal resuscitator
1 adult/1 child mask for resuscitation
1 pocket mask plus valves
1 bag (500 ml) Hartmann's Solution
1 suction unit
1 laryngoscope
4 endotracheal tubes
1 introducer
4 Guedel airways
Cloth tape – 1 metre
I.V. infusion stand
Oxygen – resuscitated tubing

1 sphygmomanometer
1 stethoscope

8 alcohol swabs
1 cannulae 18G
1 cannulae 21G
1 cannulae stopper/plug
8 gloves in clear plastic bag
1 intravenous giving set
4 needles 21G
4 needles 23G
2 scalp vein sets
4 syringe 2 ml
2 syringe 10 ml
1 sharps disposal container
1 tourniquet

1 artery forceps
1 adhesive tape
2 batteries AA and C
1 bulb for laryngoscope
2 cord clamps
1 energiser torch
1 bottle glucose testing strips for blood (Glucostix)
1 pair scissors
1 thermometer (Clinitemp)

Acetyl salicylic acid 300 mg (Aspirin)
Chlorpromazine (Largactil) 50 mg
Diazepam (Valium) 5 mg
Glyceryl trinitrate (Anginine) 600 micrograms
Mylanta
Paracetamol 500 mg/Codeine 8 mg (Panadeine)

Table 4.4 List of drugs carried in the physician's kit on international aircraft

Quantity	Generic name
10 tablets	acetyl salicylic acid (Aspirin)
3 amps	adrenaline
2 amps	aminophylline
5 amps	atropine sulphate
3 amps	benztropine (Cogentin)
1 vial	calcium chloride
10 tablets	chlorpromazine (Largactil)
3 amps	dexamethasone phosphate (Decadron)
6 amps	diazepam (Valium)
12 tablets	diazepam (Valium)
3 amps	frusemide (Lasix)
1 syringe	Glucagon
1 minijet	glucose injection BP
1 bottle	glyceryl trinitrate (Anginine)
9 amps	haloperidol (Serenace)
4 amps	hyoscine-n-butyl bromide (Buscopan)
2 amps	lignocaine 100 (Xylocard)
3 amps	metoclopramide (Maxolon)
10 tablets	Mylanta
1 minijet	naxolone (Narcan)
10 tablets	paracetamol/codeine phosphate (Panadeine)
3 amps	pentazocine (Fortral)
3 amps	promethazine (Phenergan)
1 inhaler	salbutamol (Ventolin)
1 bottle	sodium bicarbonate
5 amps	syntocinon
3 amps	water for injection

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CHAPTER FIVE

AEROMEDICAL EVACUATION

Howard Roby

The essentials of the art of aeromedical evacuation are planning and communication. If all is planned well, then the air evacuation will stand a greater chance of being trouble free. Teamwork involves doctors, nurses, paramedics and first aiders. Success depends on this teamwork and on the anticipation and resolution of potential problems before the aircraft leaves the ground.

This chapter discusses the escort's role in planned transportation of the sick and injured in aircraft. When organising any aeromedical evacuation it is vital to have as much information as possible about the patient prior to departure. The skills and training required of the escorts, and the equipment they will need to carry, will depend largely on the organiser's understanding of the nature and severity of the patient's condition. A misunderstanding of this may doom the mission to almost certain failure before it has even begun. In order to gain information, one must speak directly with the person treating the patient and not through a third party. One can never have too much information on the patient's condition, prior to the flight. The initial contact is also a good time for the receiving hospital team, the escort team and the referring hospital staff to discuss changes in treatment that may better stabilise the patient's condition prior to boarding the aircraft.

There are six phases, six crucial steps in an escort's role in any aeromedical evacuation. These are listed in the aeromedical checklist shown in Table 5.1.

EQUIPMENT

There are basically two different types of aeromedical evacuation. First, there is transport of a patient on a dedicated air ambulance and, secondly, there is transport on a commercial aircraft. In the former, many but not all of the problems relating to equipment will already be solved before the escort, whether paramedic, doctor or nurse, arrives. In the latter, however, the medical or paramedic escort will need to start from the beginning.

Anticipation, equipment checks and pre-planning are the best recipe to prevent disaster.

All equipment that could conceivably be required for the evacuation must be taken by the escort. Once airborne, the only equipment that you have to treat the patient with is what you have brought with you. There is no one else from whom you can borrow more!

Equipment can be conveniently divided into hardware and disposables. Hardware is equipment such as ventilators or ECG machines that will be used either to treat the patient (therapeutic equipment) or to monitor physiological variables such as temperature or blood pressure (diagnostic equipment). The term "disposables" includes gloves, airways, dressings and intravenous giving sets or syringes that will be used only during the evacuation and will be discarded after use.

A backpack of disposables should be standard equipment for each patient. This backpack should be divided into pockets so that equipment is easy to find in an emergency. The Thomas Pack is a commercially available disposables backpack produced in the United States. It contains dividers for different types of

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Table 5.1 An aeromedical checklist as an *aide memoire* for the escort on a planned flight to transport a sick or injured patient

1 Patient's condition

2 Equipment
- Patient-compatible
- Transport-compatible

3 Escort requirements
- Training level and skills
- Number of escorts needed

4 Flight details
- Will the escort(s) be on the ground long enough to recover from jet lag prior to transporting the patient?
- Has adequate time been allowed for flight changeovers?
- Seating requirement, ie stretcher etc.
- Altitude requirements
- Oxygen requirement + one-third

5 Patient preparation
- Gas filled spaces
- Potential for hypoxia
- Securing all lines

6 Evacuation planning
- Hospital to airport
- Tarmac to aircraft
- In flight
- Aircraft to tarmac
- Tarmac to hospital
- Handover

disposables and groups them in such a way that equipment for use with disorders of particular body systems are stored in the same area. The dividers are colour coded to make it easier to find the appropriate equipment in a hurry. The backpack configuration for disposables has been found in practice to be far more convenient to use in the con-fined spaces of an aircraft, and also more capacious than the toolbox configuration. It is also easier to carry during transport from the hospital to the aircraft and vice versa. The Thomas Pack contents are shown in Table 5.2.

Some dedicated air ambulances have opted to store their disposable equipment in pockets on the wall of the cabin. This is fine for the actual flight but causes problems during the critical phases of transport to and from the aircraft.

Regardless of the brand or type of container and regardless of whatever other equipment is carried, the "disposables" kit must contain equipment and drugs for the management of airways, breathing and circulation. Intravenous fluids should always be carried as plastic bags not glass bottles. Even if the drip bag is attached to the ceiling of the aircraft, the height above the patient will not be sufficient to allow rapid free flow of fluid. Therefore, some type of pressure infusion device should also be added to the pack, if the escort is to supervise the giving of intravenous fluids.

As there is never any guarantee that the patient will actually be suffering from the condition that they were thought to be suffering from when the evacuation was initiated, the disposables pack should contain enough equipment to treat as wide a range of conditions as possible.

All equipment taken on board the aircraft will need to be securely stowed on the aircraft so as not to become a missile during times of turbulence. Methods of securing it should be considered when deciding what equipment is to be taken. This is usually not a problem on dedicated air ambulances.

Electrically powered equipment

Most therapeutic and diagnostic medical equipment is powered by both batteries and mains power. Mains power is obviously not available on aircraft, though most do have wall outlets for their own generated electricity. This is usually 28-volt DC power, although larger commercial aircraft have 110-volt AC power. Unless prior arrangements have been made with the airline, this power is not usually available for use. When the aircraft's own electricity supply is to be used, the airline engineering department will need to ensure that the medical equipment will not short circuit the power needed to drive the flight deck instruments! This is usually a time-consuming process and cannot be organised for individual evacuations unless done well in advance. Battery power is acceptable provided the battery does not contain liquid acid. Car batteries are forbidden on aircraft as a spill would weaken the aluminium or titanium alloy fuselage. Internal batteries

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Table 5.2 Thomas Pack contents. This equipment is carried on all aeromedical evacuations along with other equipment taken specifically for the individual situation (With acknowledgements to NRMA CareFlight, Sydney)

FRONT POCKETS	Large	garbage bags disposable gloves contents list and paperwork
	Small	stethoscope aneroid B.P. cuff
SIDE POCKETS	Left	chest drains Heimlich valves urine drainage bags 12G I.V. cannula
	Right	procedural disposable pack dressing pack scalpel sutures Alco-wipes povidine iodine chlorhexidine surgical gloves
TOP POCKET		nasal oxygen prongs oxygen tubing and connector adult Hudson mask nebuliser attachment Salbutamol neb. solution and inhaler Yankauer sucker Y suction catheters – 2 each of 6, 10, 14 Oxymethazole nasal drops suction tubing
PLASTIC POUCHES	Top	crepe bandages large Telfa op-site airstrips large eye pads steri-strips
	Lower	tape: Elastoplast & Transpore marking pen scissors torch thermometer nasogastric tubes
HIDDEN POCKET		procedure kit (for CVC's ICC's etc.) Drumcath long line multilumen central line 3-in-1 junction (crows foot) cricothyroidotomy kit
YELLOW FLAP		syringes and needles

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Table 5.2 Thomas Pack contents (Continued)

YELLOW DRUG BOX

Drug	Form	Dose	Number
Cardiac section			
Adrenaline	minijet	1/1000	1
Adrenaline	minijet	1/10 000	2
Adrenaline	ampoule	1/10 000	2
Atropine	minijet	0.5 mg/0.5 ml	2
Calcium chloride	minijet	1 g/10 ml	2
Lignocaine	minijet	100 mg/5 ml	2
Bretylium	ampoule	500 mg/10 ml	1
Propanolol	ampoule	1 mg/1 ml	2
Sotalol	ampoule	80 mg/8 ml	3
Hydrallazine	ampoule	20 mg/1 ml	2
Potassium chloride	ampoule	10 mmol/10 ml	2
Magnesium sulphate	ampoule	10 mmol/5 ml	1
Verapamil	ampoule	5 mg/2 ml	2
GTN	ampoule	50 mg/10 ml	1
Frusemide	ampoule	250 mg/25 ml	1
Frusemide	ampoule	50 mg/5 ml	1
Dopamine	ampoule	200 mg/5 ml	2
Isoprenaline	ampoule	1 mg/5 ml	1
Metaraminol	ampoule	10 mg/1 ml	2
Digoxin	ampoule	0.5 mg/2 ml	2
General section			
Dextrose 50%	minijet	50 ml	1
Sodium bicarbonate 8.4%	minijet	50 ml	2
Naloxone	minijet	2 mg/5 ml	1
Thiopentone	ampoule	0.5 g	2
Dexamethisone	ampoule	5 mg/1 ml	4
Bupivicaine/Ad	ampoule	0.5% 10 ml	1
Bupivicaine/Pl	ampoule	0.5% 10 ml	1
Aminophylline	ampoule	250 mg/10 ml	2
Salbutamol	ampoule	500 mic/1 ml	2
Promethazine	ampoule	50 mg/2 ml	2
Metaclopramide	ampoule	10 mg/2 ml	2
Neostigmine	ampoule	2.5 mg/1 ml	2
Atropine	ampoule	1.2 mg/1 ml	2
D.D. pouch			
Ketamine	10 ml vial	100 mg/1 ml	1
Midazolam	ampoule	5 mg/1 ml	5
Diazepam	ampoule	10 mg/2 ml	3
Pancuronium	ampoule	4 mg/2 ml	3

Extra morphine and relaxants taken depending on the case.

Dividing panel

Cardiac side	*General side*
Nifedipine bulbs 10 mg by 4	Lignocaine amps 1% 5 ml by 10
assorted labels	water for inj. 10 ml by 10
syringe pump protocol table	N/saline 10 ml by 10
GTN patches assorted	2, 5, 10 ml syringes assorted
	needles assorted

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Table 5.2 Thomas Pack contents (Continued)

RED SECTION	
3 infusion bundles each of which contains:	tourniquet on handle pre-cut adhesive tapes cannulae 14G, 16G, 18G, 20G, 22G, each by 2 alcohol swabs Bandaids, Steristrips, Op-sites short extension set by 1 3-way tap by 1 Luer lock caps by 2
Bundles 1 and 2 also contain:	500 ml Haemaccel by 1 I.V. pump giving set by 1
Bundle 3 also contains:	500 ml 5% Dextrose by 1 microdrop giving set by 1
BLUE AIRWAY SECTION	
Yellow-striped compartment usually empty. Used for carrying items specific for the individual case.	
Green-striped compartment:	oxygen tubing Guedel airways, sizes 3 and 4 nasopharyngeal airways complete adult Laerdal bag kit expiratory air diverter and PEEP valve
Intubation roll containing:	large white bougie endotracheal tubes 9, 8, 7 introducer by 1 laryngoscope handles by 2 laryngoscope blades sizes 3 and 4 gooseneck connector with HME and swivel Magill forceps 10 ml syringe pre-cut tapes lubricant sachets Succinylcholine amps by 2 Pancuronium amps by 3
Between bundles uncut size 7 and 8 ETTs	

on most medical equipment will last only for 4–6 hours before requiring recharging. As many medical evacuations from hospital to hospital take longer than this, one is left with a dilemma. The alternatives include

- taking multiple pieces of the same equipment,
- only using the equipment intermittently (possible for monitoring equipment but not for therapeutic equipment), and
- using alternative power sources. It may be possible to modify equipment to run off nickel–cadmium (NICAD) batteries (similar to Lifepak batteries), in which case the escort can then either carry enough replacement batteries to last the estimated duration of the evacuation or apply for approval to run a NICAD battery charger off the aircraft's own power and exchange batteries as needed.

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Many commercial airlines have lists of the specific items of electrical equipment that may be used on board their aircraft. Each of the types of equipment on the list will have been checked out by the airline's engineers. If a piece of equipment does not appear on the list, it must not be brought on board. A particular brand not appearing on the list may be safe but has not yet been checked and, therefore, cannot be used. For example, at the time of writing, one airline will allow Lifepak 5 monitor/defibrillators to be used but not Lifepak 10.

Oxygen

If it is determined that oxygen will be required for the transport, then one should calculate the volume required by estimating the duration of the flight, adding to that a reasonable time for transport from the hospital to the airport at one end and from airport to hospital at the other (each of these rarely takes less than 2 hours) and adding one-third to the total time to account for unforeseen delays. This total time should be multiplied by the greatest estimated minute volume of oxygen required during the transport to calculate the total volume required. Extra oxygen will be needed if it will also be used to drive a venturi suction apparatus (eg a twin-o-vac) or a ventilator.

It is important to ascertain not only the volume of gas required but also what type of cylinders will be used. Different countries have different fittings for equipment mating with oxygen cylinders and there are at least 20 different mutually incompatible systems used throughout the world.

In Australasia, cylinders with volumes up to 440 litres (C cylinders) have valves with a pin index while larger cylinders have a screw thread to fit a bull nose adaptor. The fitting necessary to mate with one of these types of cylinder is useless for the other. Furthermore, many commercial airlines have a third type of cylinder valve that is incompatible with both of these two.

Therefore, time spent ensuring that the equipment is compatible with the cylinders is time well spent. This may require the equipment to be attached to the airline cylinder in the engineering department some time before the aeromedical transport occurs, just to ensure that everything fits together. This vital point is often forgotten and it is usually assumed (wrongly) that everything will match. Time spent pre-planning will prevent great embarrassment, or worse, at the time of the transport. When ordering oxygen, all that will be delivered is the oxygen cylinder. In order to have it available to the patient, other apparatus will be necessary. If the patient is going to breathe it through a mask, then an adaptor to fit the cylinder valve and a flowmeter will be needed as well as oxygen tubing and the mask.

Suction

Any patient with a potentially compromised airway who cannot swallow secretions or who has certain types of surgical drains will require suction. This can be produced either by using high pressure cylinder gas and a venturi system (eg twin-o-vac) or by battery powered suction systems. The venturi system is probably easiest to use as it only involves screwing the device onto the oxygen cylinder. However, it uses approximately 22 litres per minute of oxygen to drive it and this must be included in the calculations when estimating oxygen need. The battery suction system suffers from the problems that all battery powered systems have in evacuations: limited life before recharging is necessary.

Ventilator

One popular evacuation ventilator in use is the Oxylog, manufactured by the Dräger Company. In different parts of the world, however, there are a number of different transport ventilators available. The vast majority are oxygen driven so, if such a device is to be used, one must ensure that the gas volume required to drive the ventilator is added to the total oxygen ordered for the transport. One must also ensure that the fitting from the ventilator oxygen tubing to the cylinder is compatible with the cylinder valve. If a piston-driven ventilator, such as the Dräger EV800, is used, one must bear in mind that the volume of gas driven will decrease with an increase in cabin altitude (see below). Ventilators should never be used in aircraft, or anywhere else, without a disconnect alarm attached.

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Fluid administering equipment

If rapid administration of fluid is envisaged, then a pressurised rapid infusion device will be required. If a constant infusion is required (eg for drug administration), then syringe pumps are very convenient. However, they suffer from the same problems, in the air, as other electrically powered equipment.

Monitoring equipment

There are some general points that must be considered in relation to the monitoring of equipment in aircraft.

- Aircraft cabins are noisy and suffer from vibration. Therefore, items of equipment with only audible alarms may not be as suitable in the air as they would be on the ground. All important alarms should have both visual and audible outputs. The vibration that is inherent in any airborne craft may interfere with the patient–equipment interface and create artefacts. ECG tracings, automated blood pressure readouts and pulse oximeter readings may suffer from this problem. The only way to ensure that these problems will not occur with a particular piece of equipment is to try it in advance in the particular aircraft that is going to be used for the retrieval or transport.

- The decrease in cabin pressure at altitude compared with sea-level will also interfere with the accuracy of some equipment. Some brands of non-invasive blood pressure monitor compare blood pressure with ambient pressure. As the ambient pressure will have dropped at altitude, there will be an inaccuracy in the machine reading of the patient's blood pressure. There is some suggestion also that end tidal CO_2 monitors ($ETCO_2$) become inaccurate at altitude for similar reasons.

- Pulse oximetry is probably the most useful method of monitoring both cardiac and respiratory function, and should be used on all evacuations where there is any concern about the patient's general condition. Perhaps surprisingly, it can be quite difficult to estimate blood pressure in flight. The cabin is often too noisy to use a regular sphygmomanometer and stethoscope technique. Even if this is not the case, one must choose the sphygmomanometer carefully. Mercury sphygmomanometers are banned on aircraft because of the potentially destructive effect of leaked mercury on the aluminium content of the metal fuselage. The alternative is to use aneroid sphygmomanometers but, as mentioned already, these compare the patient's blood pressure with atmospheric pressure at sea-level and, therefore, will be inaccurate. Some gauges have a zeroing screw which will allow them to be re-zeroed at altitude, and these will be more accurate. Some non-invasive blood pressure monitors (NIBP) will automatically recalibrate for altitude and these may also be appropriate for use. In patients whose blood pressure is very unstable, it is safer to use an invasive technique, but without a dome on the pressure transducer. The newer disposable transducers will need to be re-zeroed at cruising altitude to remain accurate. There is some suggestion that at the low ambient pressures found at high altitudes, in patients with high blood pressures the degree of stretch on the disposable blood pressure transducer may add an inaccuracy to the system.

  Because of cabin vibration, some ECG monitors will give inaccurate heart rate calculations. The better monitors have two modes of function: the first is a diagnostic mode which is used for examining fine changes in the patient's ECG, and the second mode is used for rhythm monitoring and incorporates a filter to stabilise the trace. This mode is more appropriate for aircraft use as it will hopefully filter out movement-generated artefact.

In addition to the problems caused for medical equipment by the airborne environment, it should not be forgotten that some equipment may actually interfere with the aircraft's navigation equipment and cause the pilots to lose the function of some of their instruments. Some defibrillators emit Radio Frequency (RF) radiation when they are used, and this can interfere with aircraft navigation equipment. One should liaise with the airline as to what equipment is and is not allowed to be used on board.

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PREPARATION OF THE PATIENT

Any procedure that needs to be performed on a patient is easier to perform in their hospital bed than on the aircraft. If the patient may need an intravenous cannula at some stage during the evacuation, then this should be inserted prior to leaving the hospital. If it is essential that they have one cannula, then a second one should be also inserted and bandaged up prior to leaving the hospital. It will then be available when required.

Every line or drain attached to the patient will stand a reasonable chance of falling out or being pulled out at some stage during the transport. Past experience would suggest that the more important the line is to the patient's wellbeing, the more likely it is to fall out! All cannulae and drains must be secured to the patient prior to leaving the hospital. This may mean suturing them to the patient's skin as well as securely taping them in.

The incidence of deep venous thrombosis (DVT) is much higher among passengers in aircraft than in the general community. This may be because they sit or lie in cramped spaces for long periods of time but it may also be related to the cabin environment. Any patient with cardiac or respiratory disease or any chronic illness is already at increased risk of developing DVTs and these patients should be given subcutaneous heparin for the duration of the evacuation to lessen this risk.

Any transport of a patient has the potential to make sick patients sicker. This is true whether the patient is being transferred from one side of the hospital to the other or from one side of the world to the other. Aeromedical transports expose patients to added dangers in addition to those occasioned by transport *per se*. These dangers may be classified as:

1 Hypoxia due to altitude
2 Decreased atmospheric pressure
3 Variable temperature and humidity of the cabin environment
4 Confined spaces in which the escort must work

Commercial aircraft at cruising altitude usually have a cabin altitude of about 8000 feet. At this altitude, arterial PO_2 is about 57 millimetres of mercury compared with about 100 millimetres of mercury at sea-level. Therefore, any patient suffering from hypoxia may become acutely unstable, physically or mentally, during flight. Conditions that cause concern include:

Pneumonia
Acute asthma
Angina
Arrhythmias
Anaemia
Lung cancer
Congestive cardiac failure
Adult respiratory distress syndrome
Cystic fibrosis
Pulmonary fibrosis
Myocardial infarction
Congenital heart disease
Chronic airways disease
Sickle cell anaemia
Acute myocardial infarction

All of these patients should have supplemental oxygen during flight. The symptoms and signs of hypoxia are so subtle that unless one has a pulse oximeter to help with the diagnosis, all patients with these conditions should be considered to be hypoxic and should have added oxygen from at least takeoff until landing. Because hypoxia is so difficult for the patient or escort to diagnose, there is little indication for ever using intermittent oxygen during flight.

At a cabin altitude of 8000 feet, any gas-filled cavity will expand to 1.4 times its volume at sea-level. Therefore, patients with trapped gas anywhere in their body may experience difficulties ranging from discomfort to life-threatening conditions. Conditions that cause concern include:

Pneumothorax
Recent pneumoencephalography
Bowel obstruction
Sinusitis
Middle ear infections
Severe upper respiratory tract infection

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Recent ear surgery
Recent eye surgery
Recent brain surgery
Endotracheal tube cuffs
Recent bowel surgery

In such cases, if the pressure change associated with altitude is likely to cause serious adverse effects on the patient's condition, then one should consider two possibilities. Either the evacuation should be delayed until the volume of trapped gas has decreased to an acceptable level, or the evacuation should be carried out in an aircraft that can be pressurised to sea-level, thus averting the problem, but increasing the cost of transport.

Endotracheal tube cuffs are usually filled with air to prevent leaks. The increased volume occupied by the air at altitude will result in increased pressure which may cause tracheal mucosal ischaemia and sloughing, with catastrophic results. Therefore, either a close watch must be kept on the cuff pressure with air being withdrawn during ascent and replaced during descent, or the air should be replaced by water prior to takeoff. Saline tends to crystallise out in the cuff, and may therefore, be difficult to aspirate, so it should not be used.

The low humidity of the cabin environment will cause two types of problem in patients.

- First, it will speed up the rate at which patients become dehydrated. If they are capable of drinking, they should be given increased fluids to compensate for this. If they are incapable of drinking, they should be given fluid either by nasogastric tube or intravenously, and a constant watch kept on them in flight to alert the escort to impending dehydration. Urine output is the best feature to observe and, if the patient is catheterised they should be producing at least half a millilitre per kilogram per hour. If conscious and not catheterised, they should want to pass urine at least every second hour.
- Secondly, the low humidity will cause drying of the eyes and mouth in unconscious patients. If left untreated, this will rapidly cause corneal and mouth ulceration and measures must be taken to prevent these problems from occurring. Drops of castor oil may be used to keep the corneas moist, but any artificial tears will work if applied often enough.

THE EVACUATION

Once all the planning has been carried out, and the escorts and medical equipment selected, the actual evacuation comprises five phases, each with its own particular problems.

1 Hospital to airport

In the case of relatively stable patients, organisation of this phase may simply involve booking an ambulance. However, in the case of unstable critically ill patients this may be the most worrying step, and the transport to the aircraft may need to be rehearsed several times prior to the actual evacuation in order to find and solve potential problems. For patients who are so ill that they cannot tolerate their therapeutic or monitoring equipment being removed even for a short time, one must plan in advance how to transport the equipment plus its power source, with the patient from their hospital bed to the ambulance, and thence to the airport, taking into account possible delays on the way. The internal battery life in many pieces of equipment is too short for even this journey, much less that of the actual flight.

It helps to have a person at the airport with whom to liaise, so that departure from the hospital can be delayed until such times as there is minimal delay on the ground after loading before takeoff. For international flights, it will also be necessary to liaise with the authorities to cover problems with Customs and airport security. Airline ground staff are usually very helpful in organising this phase of the transport. These formalities are usually carried out in the ambulance on the tarmac immediately prior to loading. I make no apologies for repeating that for very unstable patients the time taken to rehearse this step will be well spent. You get only one chance "in real life", and often a problem will be discovered during rehearsal that was never even

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considered during planning – for example, the oxygen cylinder to drive the ventilator may be too large to allow room for the stretcher to fit into the ambulance.

2 Tarmac to aircraft

This phase may be carried out with stable patients by simply having them walk or be wheeled on board the aircraft in a wheelchair. However, unless the aircraft is an international commercial jet leaving from a large airport, there may not be a skybridge available and the only access may be by a staircase which is difficult for an incapacitated patient to negotiate. For commercial aircraft, a Hilift may be available which will elevate incapacitated patients to a cargo entrance, whence it is a relatively easy matter to carry them on board (Figure 5.1). For stretcher-ridden patients on chartered aircraft, this stage may cause almost insurmountable problems, and again a rehearsal is recommended prior to the actual evacuation. Many aircraft have doors that are too narrow to allow a patient on a stretcher to be carried on board with the stretcher remaining horizontal. If you have never previously put a stretcher on board the type of aircraft you intend to use, you must first try it without a patient to see if everything fits.

During the process of moving the patient onto the aircraft, the escort should be constantly by the patient's side (Figure 5.2). The helpers often have no medical training and will be unaware of the significance of snagging the endotracheal tube or intravenous line during the movement.

3 In flight

If the pre-planning phase has been effectively carried out, the in-flight phase should be the least troublesome section of the transport. Once on board, the litter and all equipment must be stowed securely (Figures 5.3–5.5). The equipment must also be positioned in such a way that the dials can be seen and so that it can be used without the possibility of accidentally bumping against it and changing the settings. Commercial jets cruise with the nose of the aircraft slightly higher than the tail; it is usually preferable, therefore, to position the patient with their head towards the front of the aircraft.

During the flight, the patient should receive, at the very least, the same level of care that they would in their hospital bed. It is not enough just to keep them alive. Special care must be taken with pressure areas, and it is good practice for at least all stretcher patients and probably all disabled passengers to be nursed on sheepskins or other surfaces that will limit the production of skin pressure sores. Many aeromedical retrieval organisations nurse their unconscious or debilitated patients on vacuum mattresses. The author has found these to cause, in most cases, more trouble than benefit, but they do have a special role to play in the case of patients, for example those with spinal injuries, who need to be immobilised for the trip. If a vacuum mattress is to be used on a commercial aircraft then it is important to ensure that the mattress is no wider than the stretcher, otherwise problems may be encountered in securing it to the stretcher base or in using the stretcher "seat belts". Warm blankets should be made available, as the cabin may become very cold.

For long trips, the escort staff will become very fatigued. Rather than have back-up staff also board the aircraft at the commencement of the evacuation, it is better to have them board at some stopover *en route* if this is feasible. This technique is used in the transport of unstable patients on the long hauls from Europe to Australasia, with a second escort team boarding the aircraft at the Asian refuelling stop.

If patients are going to experience difficulty during the flight, it will usually occur during ascent and descent; it is very important to be vigilant and have patients fully monitored during these phases. Full observations should be taken during the flight, and they should be recorded in such a way that a copy can be handed over to the receiving hospital at the end of the trip. If the flight is trans-meridian, then for the purpose of timing observations and the dispensing of medication a decision should be made prior to takeoff as to whether the team will work on the time zone of departure or that of arrival; the use of this time zone should be maintained for the entire length of the trip. Once this decision is made, it may be helpful to draw up a 24-hour chart and to mark on the chart at what times

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Figure 5.1 *Stretcher cases with their attendant equipment and support personnel may be transferred from a motor vehicle ambulance to a medical Hilift van and thereby directly onto the aircraft.*

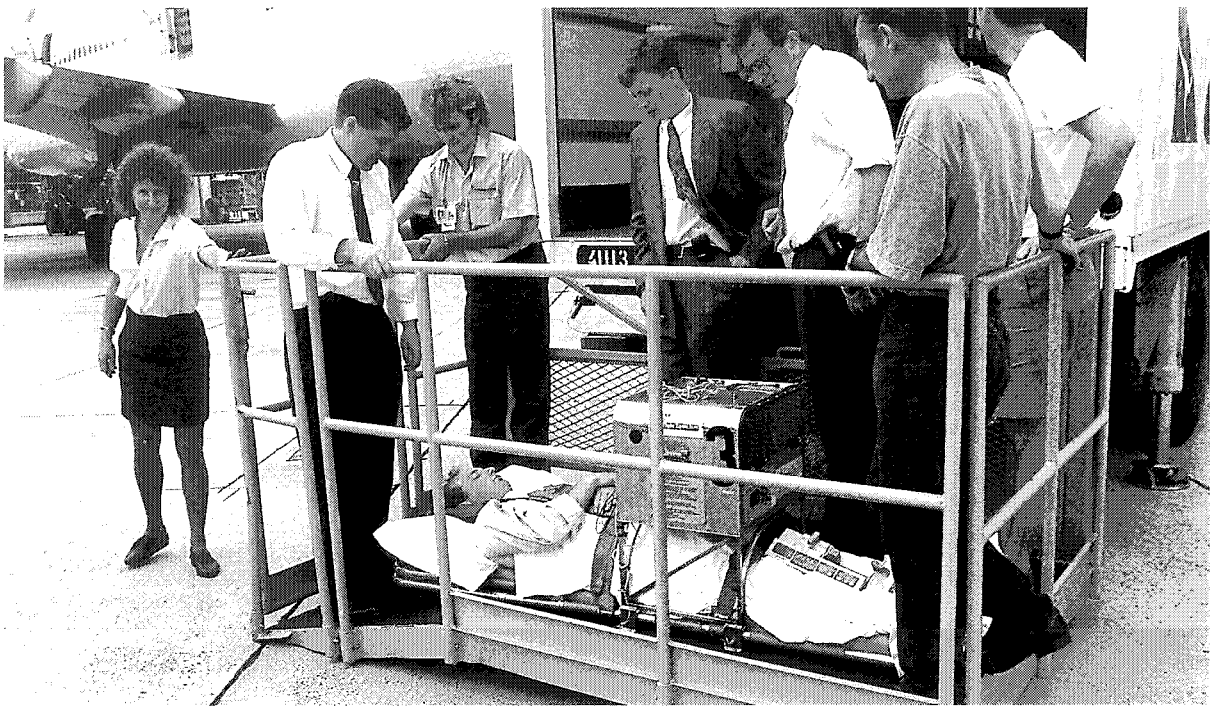


Figure 5.2 *The "tarmac to aircraft" phase is an important link in the evacuation chain. Escorts must be constantly at the patient's side. Equipment, especially intravenous infusion lines and electrical cables, is especially vulnerable to dislodgement during this phase.*

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Figure 5.3 *Securing the litter in the aircraft requires many hands, and takes time. A patient-free rehearsal using all the intended equipment is often necessary, especially on aircraft not routinely configured for such transfers.*

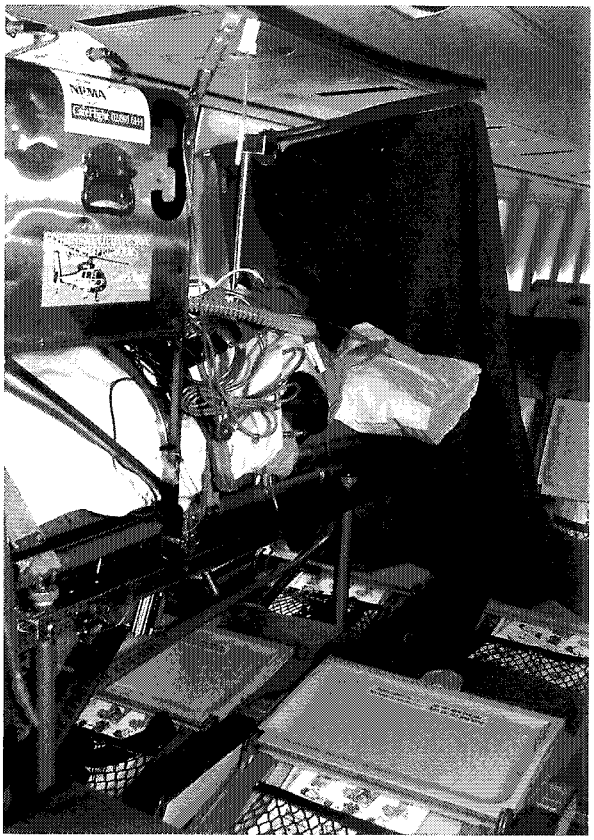


Figure 5.4 *On larger aircraft, most casualties are transported in a modified bay at the rear of the cabin. This photograph shows a patient stabilised and in place, prior to embarkation of the other passengers. Fore and aft curtains have been rigged for the privacy of the patient.*

observations are to be made and medications administered. This will minimise confusion regarding time zones during flight.

4 Aircraft to tarmac

This is phase 2 in reverse, except that the escort is, by now, always fatigued and lacking his or her pre-flight vigilance. This phase is also dangerous as lines are particularly liable to be pulled out or disconnected. Help will be required from the ground, and this should have been organised prior to commencement of the flight. On commercial aircraft, the patient should not be moved until all the other passengers have disembarked. A check should be made for all loose equipment, which by this time may well be scattered from one end of the cabin to the other. It has been found in practice that phases 4 and 5 can be run more smoothly if the escort has access to a mobile phone.

5 Tarmac to hospital

As with phase 4, the success of this phase will depend on prior planning. It is tempting to leave the organisation of this step to the receiving hospital, but, whilst they should definitely be involved, it is the overall responsibility of the escort to ensure that acceptable arrangements have been made. If an ambulance is the appropriate means of transport, the ambulance service should be given the flight details in advance so that they can check with the airline on an estimated time of arrival (ETA). For chartered aircraft, the airport control tower will usually be able to supply an ETA. Prior to the commencement of the evacuation, the ambulance service should be given information on the patient's condition and any special equipment that may be required for this phase of the operation. Again, steps should be taken in advance to ensure that oxygen and electrical fittings in the ambulance will be compatible with those accompanying the patient.

Finally, the patient's wellbeing is the responsibility of the escort until the patient is handed over to some person willing to take responsibility. This may be a hospital accident or emergency department, a hospital

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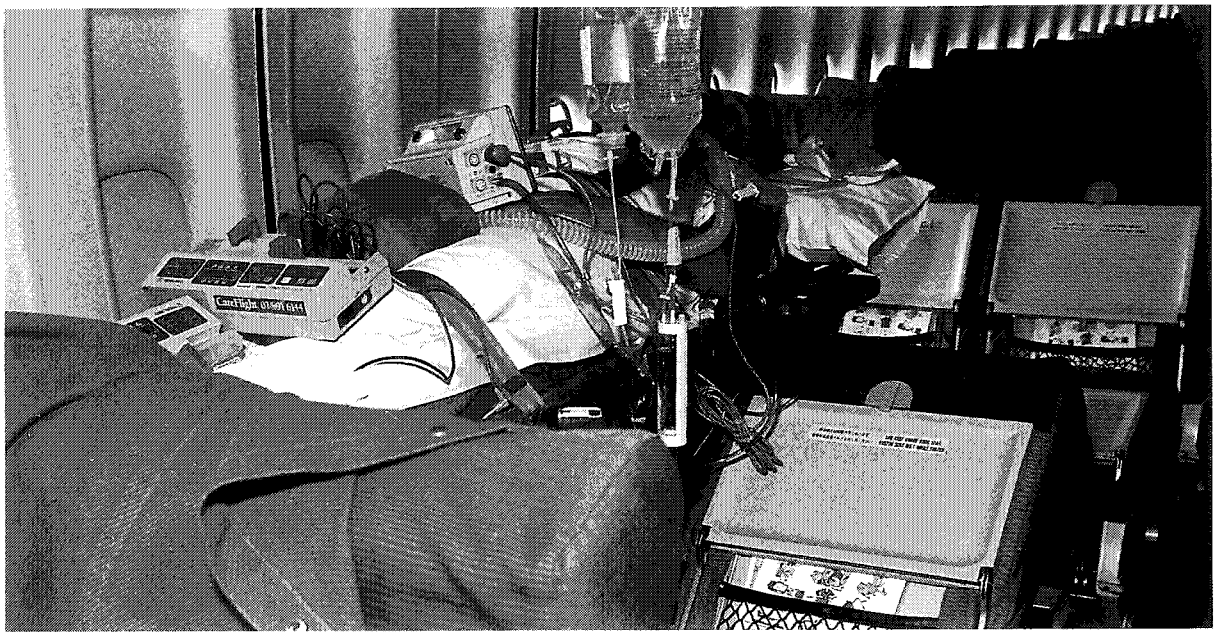


Figure 5.5 *It takes considerable time to configure the litter bay to transport casualties by both commercial and military aircraft. In this case, nine seats have had to be utilised for space. Again, the need for a patient-free rehearsal is evident.*

ward, or, if the patient is to be taken to his home, his local doctor. If a decision is made prior to transport that the patient will go to a hospital, it is helpful for the escort to know *exactly* where in that hospital the patient is to be received and the name of the accepting doctor. The escort should hand over a letter to the new treating doctor or nurse, containing a copy of the medication chart and observations during the evacuation.

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PART

II

CARDIOPULMONARY RESUSCITATION

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CHAPTER SIX

THE AIRWAY

John Williamson and John Pearn

Clearing and maintaining an airway is one of the most basic and important of all first aid skills. In the patient who is unconscious because of intoxication, or following such catastrophes as a stroke, heart attack or near-drowning, maintaining the airway may be straightforward. However, in those who are obese, in those with maxillofacial or cervical spine trauma and in those with impacted foreign bodies in the pharynx, establishing and protecting an airway may call for some of the most important skills in the whole range of first aid.

Under the St John Ambulance DRABC action plan, establishing and maintaining a clear airway is always the first thing to do – the "A" of the "ABC" – once the accident scene has been assessed ("D" for Danger) and once the patient has been diagnosed as being unconscious following the "gentle shake and shout" test ("R" for Response).

A number of experiments have measured airflow using different methods of emergency airway control – such as the chin-lift-head-tilt method (Figure 6.1), the jaw-thrust method (Figure 6.2), and a neck-lift-head-tilt method. In most unconscious patients breathing spontaneously, the simple chin-lift-head-tilt method is the most efficient. If air entry is not adequate, the jaw-thrust technique can then be tried; such patients are usually on their backs (supine). The neck-lift-head-tilt method should never be used.

Tidal volume is the amount of air breathed in (and, therefore, also normally breathed out) in an average breath at rest. It varies with size and age, and is approximately 10 millilitres per kilogram of body weight. Whenever an airway is partially or completely blocked in an unconscious or paralysed victim, this tidal volume is reduced. Clinically, the first aider can detect this in a patient who is attempting to breathe by

a seeing that the chest does not rise or fall normally, or

b hearing noisy breathing – a snore, stridor or a wheeze means that the airway is at least partially blocked.

In the clinical laboratory or in the operating theatre, obstruction of the airway can be detected by measuring the tidal volume. Much of the research on airway control and expired air resuscitation is now undertaken using such studies of tidal volume and tidal flow.

Why do unconscious but untraumatised patients have potential trouble maintaining the patency of their upper airway? The commonest cause is obstruction by the tongue.

> *The tongue is often the cause of upper airway obstruction. In the comatose or anaesthetized person, support muscles of the mandible are relaxed. Thus, the tongue, which is attached to the mandible, can drop against the back of the pharynx. This may occur more often in the unconscious person who is making respiratory efforts. Even though the head is tilted backward and the neck extended the mandible frequently needs support to lift the tongue adequately off the posterior pharyngeal wall to provide an open airway. When the chin is lifted the mandible moves forward, pulling the tongue with it. Both chin-lift and jaw-thrust techniques lift the mandible. If the victim has loose (but undisplaced) dentures they will sometimes be held in position with the chin-lift method, and may facilitate the mouth-to-mouth*

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seal if resuscitation is needed. When using manikins to teach cardiopulmonary resuscitation, it should be kept in mind that they (ie manikins) are not as heavy as a human and that the manikin's mandible does not relax as an unconscious human's does. (Charles Guildner in "Opening the Airway" in Advances in Cardio-Pulmonary Resuscitation, *1976.)*

With the standard chin-lift-head-tilt method, almost all untraumatised, spontaneously breathing adult victims in need of airway assistance can achieve a tidal volume in excess of 100 ml, and two-thirds of subjects can achieve a tidal volume of over 400 ml per breath.

Special problems of airway control arise in the case of victims with facial or neck trauma (Figure 6.4).

The multiple-injured patient may have injuries of the face and neck that are life threatening or sufficiently serious to require specialist advice and management. In the initial assessment such patients may present with airway obstruction. The resuscitation team must be aware of these problems, know which specialists to call and when to call them and be able to initiate manoeuvres to prevent the demise of the patient before the arrival of the specialist.

The first priority of the resuscitation team is to secure and maintain an airway. This may involve finding a path through the site of maxillofacial trauma, which may be littered with broken teeth and dentures, bits of fractured bone, and macerated soft tissue. (Dr Iain Hutchison in "Major Maxillofacial Injuries", British Medical Journal, *1990.)*

In these circumstances, the patient's life depends on clearing the airway.

1 Turn the patient into the lateral position, with appropriate precautions. Clear the oral cavity by using a (preferably gloved) finger inserted laterally (just inside the cheek) to the back of the mouth, then hook the finger medially and forward to pull debris out of the mouth. If the finger is pushed centrally foreign bodies may be pushed further down the airway. Ensure that the patient cannot bite, before inserting your finger!
2 Repeat this manoeuvre from the opposite side of the mouth.
3 If available use a large bore sucker and good illumination to aspirate the oral cavity. Ignore potential pain from fractures of the jaws – the airway takes precedence over all else.

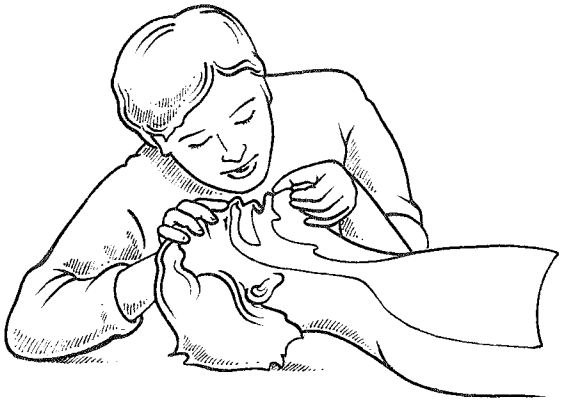


Figure 6.1 *Chin-lift-head-tilt method for emergency airway control.*

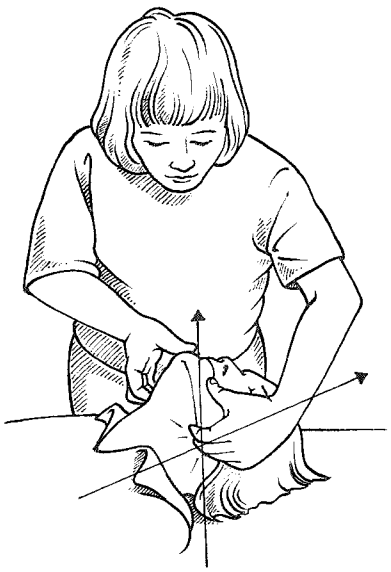


Figure 6.2 *Jaw-thrust method for emergency airway control.*

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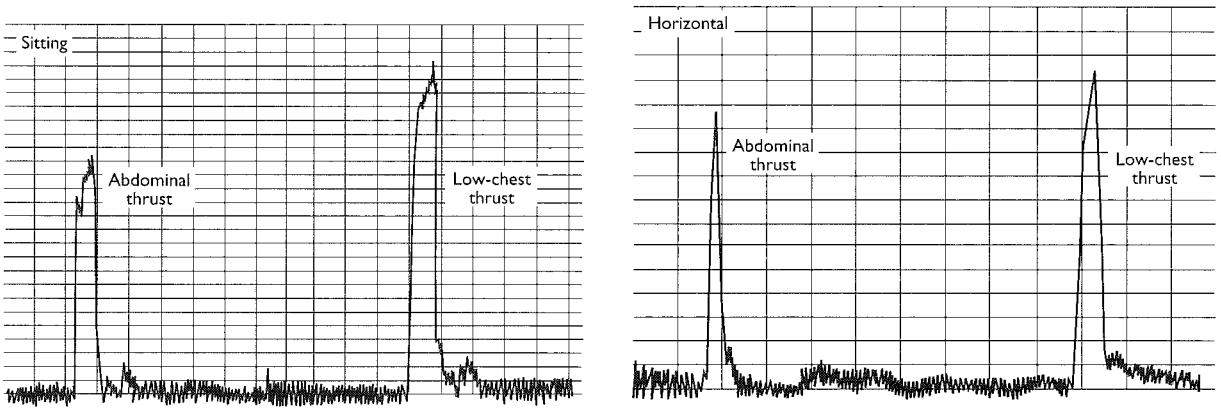


Figure 6.3 *Typical airway pressure tracings in an anaesthetised apnoeic volunteer, with tracheal tube occluded. Abdominal thrust (left) and low-chest thrust (right). Subject sitting (left) and horizontal (right). (Courtesy of Charles Guildner and* Journal of the American College of Emergency Physicians, *1976.)*

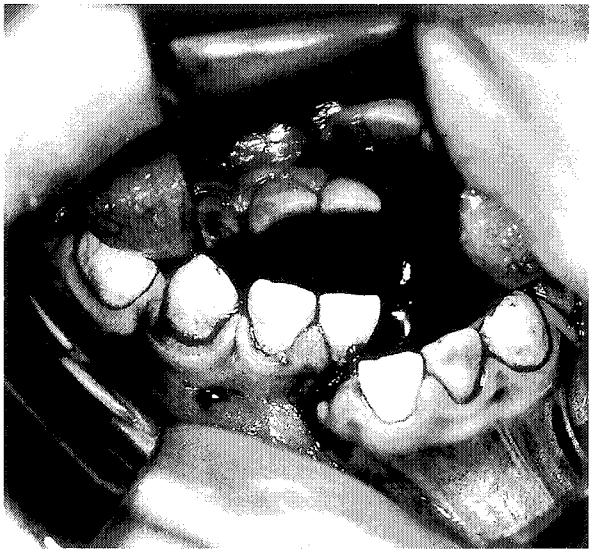


Figure 6.4 *Fractured lower jaw of a four-year-old child, caused by a horse kick to the chin. Pre-hospital care includes both manual stabilisation of the jaw and maintenance of a clear airway. Blood clot from the mouth has to be removed manually. (Photograph courtesy of Dr Caroline Acton, 1994.)*

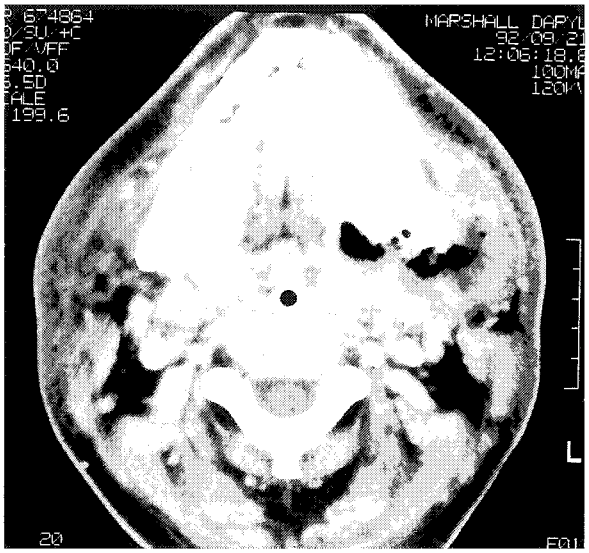


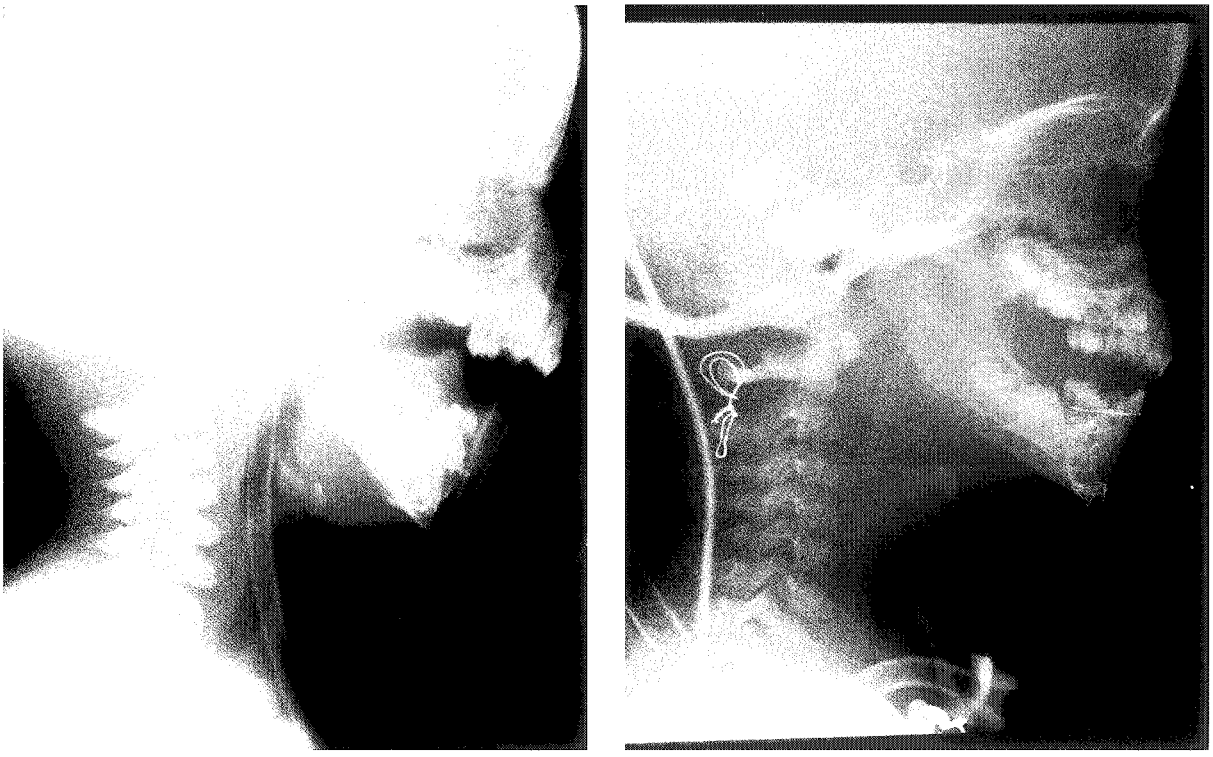
Figure 6.5 *A life-saving airway. This CAT scan is a horizontal section through the lower jaw of a victim who almost suffocated from obstruction to the airway by gross infection and swelling in the soft tissues of the mouth, face and neck. The airway has been maintained by an endotrachial tube which maintains a clear pipeline to the lungs, in spite of the swollen tissues pressing upon it. (Photograph courtesy of Professor Alastair Goss, of the Oral and Maxillofacial Surgery Unit, University of Adelaide.)*

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Maxillofacial trauma may occasionally be associated with trauma to the larynx and trachea, which may cause obstruction to the airway by swelling or displacement of structures such as the epiglottis, arytenoid cartilages and vocal cords (Figure 6.5). The skilled first aider must anticipate increasing problems with the airway under these circumstances.

Maintain a high index of suspicion concerning developing airway obstruction if the mechanism of injury suggests trauma to the larynx and trachea – for example, in cases of blunt trauma of the neck caused by impact with a steering wheel. Note any neck swelling, dyspnoea, coughing, voice alteration, central cyanosis or frothy haemorrhage.

Palpate the neck gently for surgical emphysema (crackling), tenderness, and, before swelling progresses, laryngeal or tracheal crepitus at the site of the fracture. See below for comments concerning spinal injury.



A B

Figures 6.6 A and B *A first aider's nightmare – an unstable fracture of the neck with a compromised airway. Figure 6.6A (left) is a lateral radiograph of an 18-month-old child involved in a motor vehicle accident. There is a displaced fracture of the odontoid peg forward of the body of the second cervical vertebra. There is also a "stove-in" fracture of the front half of the mandible with backward displacement of the fragments which are seen overlying the pre-molars on the radiograph. This means that the anterior fixation of the tongue has no anchor, and that the tissues are now mobile and the tongue can fall back and occlude the oropharynx very easily. Fortunately for the child, this did not occur, and the radiograph shows a clear airway still present shortly after the accident.*

Figure 6.6B was taken soon after the operation and shows, most importantly, a tracheostomy tube which has been inserted in order to maintain the airway, and wiring of the neural arches of cervical vertebrae 1 and 2 so that the fractured neck has become stabilised. The teeth have also been wired to reconstruct the mandible in its proper alignment. This shows how far backwards the fragment of mandible had been displaced at the time of the first radiograph. The airway is now completely occluded because of swelling in the oropharynx and root of tongue, possibly because of operative measures. A naso-oesophago-gastric tube is shown, by which route nutrition was maintained. The child made a complete recovery without any paralysis and without any brain damage due to anoxia. (Photographs courtesy of Dr J. Ratcliffe of the Royal Children's Hospital, Brisbane.)

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Figure 6.7 *A prickly warning for the first aider. This infant choked on something with which he was playing while his mother was sewing. He was wheezy and refused to eat or drink so his mother brought him to the hospital where this radiograph revealed the nature of the obstructing object.*

A second specific problem related to airway clearing and maintenance is one which the first aider encounters in the post-trauma victim with a fractured cervical spine. The correct management of such patients by first aiders has generated extensive debate and some confusion.

Certain principles require re-stating here.

1 Suspect cervical spine injury in any of the following situations:
   - any deceleration injury
   - any significant fall
   - surf board or surf wave injury
   - any diving-into-water injury
   - any injury with neck pain and/or numbness or weakness.
2 Airway maintenance takes absolute priority. Do not sacrifice an airway for the sake of concern for the spine (Figure 6.9).
3 Avoid lateral flexion or rotation of the head on the neck. Support in a neutral position.
4 Obtain airway clearance using gentle sagittal (midline forwards and backwards) head and neck movements only.
5 Maintain firm but gentle head-on-neck support with midline alignment at all times.
6 Any turning of the patient should be by "log rolling".

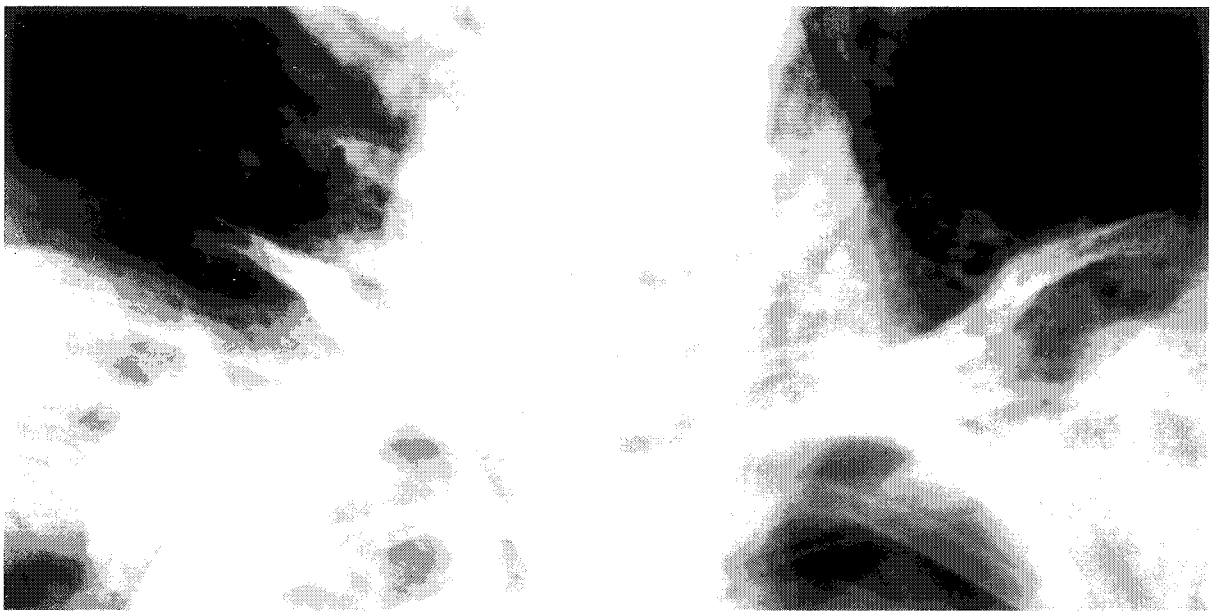
The optimal position for a clear airway is usually compatible with the appropriate care for an unstable neck injury. Avoid all extreme, sudden or forceful movements of head on neck. Maximal extension of head on neck should be avoided, and is often not necessary. In infants and babies, the optimal airway position may normally be achieved with something less than full head-on-neck extension.

The third specific problem with the airway is when it is suddenly blocked in a conscious patient, usually by food. Data from the National Safety Council of the United States of America claims that foreign-body obstruction of the airway accounts for more than 3000 deaths annually in that country. (If these figures are reliable, it is of interest that the incidence of this event seems to be much higher in the United States than elsewhere.)

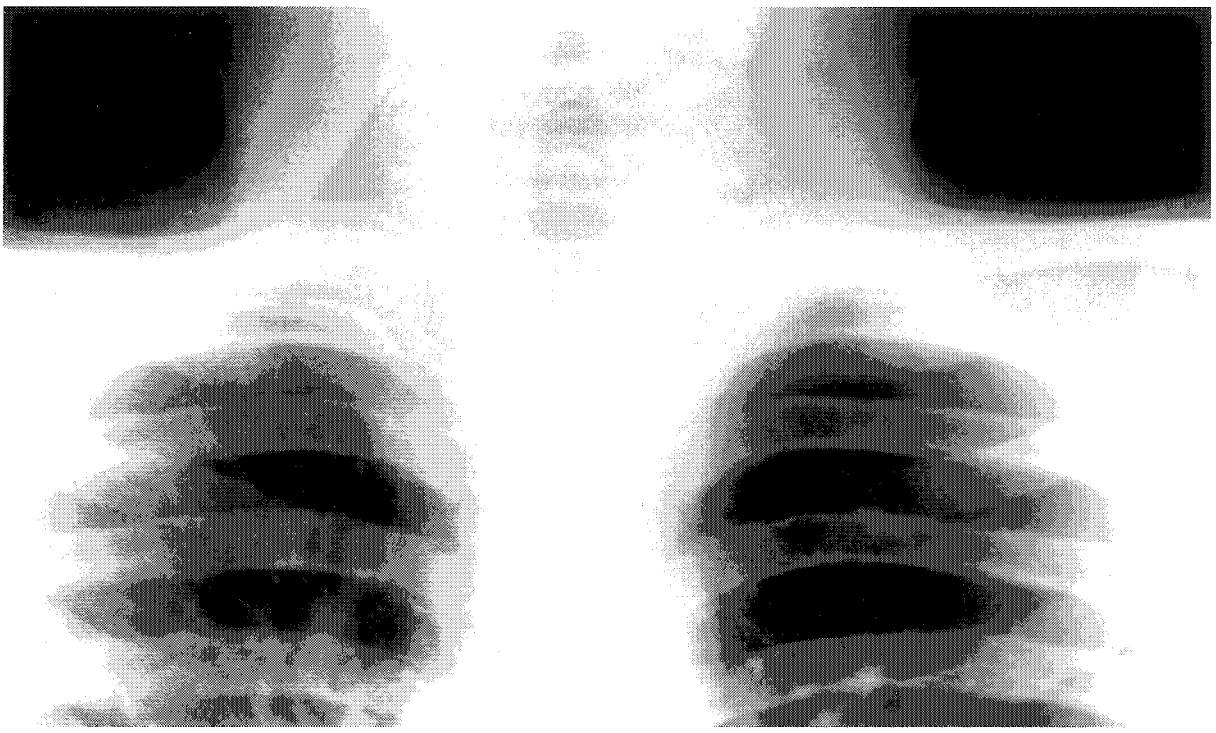
Choking from any cause is one of the worst experiences anyone can have. In such episodes, for both patient and first aider the tendency to panic may be present. A person who is eating may suddenly grasp their throat (Figure 6.10), and stand or thrash about, unable to speak, cough or breathe. If the person is conscious, the standard first aid drills involve a four-step sequence, repeated cyclically if necessary.

Step 1 First, try to see the foreign object and remove it with fingers or equipment. Beware of being bitten. At a famous American Red Cross meeting (27 October, 1975) at which the subject of choking was dealt with in depth, it was demonstrated (from autopsies) that a proportion of complete, untreated, fatal airway obstructions by chunks of meat were due to lodgement high in the pharynx and probably could have been removed by a first aider's finger or with forceps. As a first step:

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A



B

Figures 6.8 A and B *Two different airway problems. Figure 6.8A is a radiograph of the neck and upper chest of an infant with a ruptured airway, and shows dramatic swelling of the soft tissue. The child also has a left small pneumothorax, and had previously had one on the right which had been drained by the prompt insertion of an under water, sealed, intercostal drain. Figure 6.8B shows a normal neck, with uniform density of soft tissue on either side of the spine. What can be seen of the lungs is not normal. There is air trapping in the left lung, with reduced vascular shadows, because of an inhaled peanut in the left main bronchus. (Photographs courtesy of Dr J. Ratcliffe.)*

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Figure 6.9 *Although there may be a high index of suspicion that the spine may be injured, nothing takes precedence over checking the airway and ensuring that it is clear. Photograph in a Sydney street, 1995.*



Figure 6.10 *Universal distress signal for foreign-body airway obstructions.*

> *Paramedics, emergency department nurses, and physicians...should certainly not waste time with any indirect manoeuvres [such as chest or abdominal thrusts] but should first try to extract the foreign body under direct visualisation. (W. Haugen, autopsy pathologist, 1975.)*

Whilst it is important not to push any partly visible object further down the pharynx or airway, this much-feared contingency, in adults at least, is rarely a problem in practice.

Step 2 If the chunk of food or other foreign material cannot be seen or quickly extracted, do not waste further time. The next step is to apply four back blows to the patient, whatever his posture, in rapid succession.

Step 3 If the object is not dislodged apply low chest thrusts. If this does not dislodge the object and if the patient lapses into unconsciousness, use one's finger again to try to clear the airway in accordance with the standard ABC drill.

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Step 4 If this does not work, commence EAR.

Repeat these sequences, trying (once) the back blows with the victim held upside down or "head down". Repeat the sequence until the airway is unobstructed or can be cleared by a bronchoscope or until the victim is pronounced dead.

American studies have shown that 75 per cent of patients in that country who suffer "cafe coronaries" (choking which occurs during a long meal with alcohol) had high blood alcohol levels. Under these circumstances, swallowing, coughing and other protective reflexes are impaired. Vomiting or regurgitation will also usually occur.

The 1985 (United States) National Conference on Cardiopulmonary Resuscitation and Emergency Cardiac Care recommended the Heimlich manoeuvre – subdiaphragmatic abdominal thrusts – to relieve foreign-body airway obstruction. In this procedure (which has not been adopted by the first aid organisations of many other countries), each individual thrust is administered with the intent of relieving the obstruction. It is claimed that it may be necessary to repeat the thrust six to ten times to clear the airway. During application of the Heimlich manoeuvre it is possible to cause damage to internal organs, such as rupture or laceration of abdominal viscera. Also regurgitation may occur as a result of abdominal thrusts, with the attendant risk of aspiration of stomach contents into the airway, especially with the victim in a head-up position.

Much international controversy has surrounded the best methods of applying thrusts to expel an impacted bolus of food in the upper airways. Whilst the Heimlich manoeuvre recommends that abdominal thrusts be used, studies of pressure tracings show that higher propulsive pressure can be generated, more safely, by applying low chest thrusts (see Figure 6.3), with the victim lying in the lateral position, and the rescuer lying behind.

If conscious victims have a bolus of food, broken teeth, or laryngeal oedema blocking the airway, they reflexly try to cough. Instinctively the victim tries to inhale prior to coughing, but this is not possible with total obstruction. Thus, during asphyxiation, the lung volume tends to shrink. Also when foreign material covers or irritates the larynx or trachea, reflex spasms of the vocal cords (laryngeal spasm) and bronchus occur, and more general reflexes may also come into play. Even a fragment of inhaled nut or celery fibre, or a drop of vinegar, can produce paroxysms of coughing, hypoxia, cyanosis and reflex apnoea. If this progresses to unconsciousness, first aid drills remain the same. Expired air ventilations may become effective as soon as this reflex laryngospasm relaxes. Such reflex spasm nearly always does relax before the heart stops from hypoxia. Thus there is an imperative demand to continue with expired air resuscitation even in an apparently non-responding case.

AIRWAY MONITORING

An airway that is initially clear may not remain so, eg there may be progressive swelling, bleeding, vomiting or regurgitation. Thus it is imperative that the first aider does not leave an unconscious victim alone for even a few minutes unless it is unavoidable. The lateral position offers the best airway protection in the unconscious, spontaneously breathing victim.

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CHAPTER SEVEN

EXPIRED AIR RESUSCITATION

John Williamson

Expired air resuscitation (EAR) is the act of inflation of an inadequately breathing, or non-breathing, casualty's lungs with the exhaled air from the lungs and mouth of a rescuer who is breathing air (Figure 7.1).

Many international bodies including the Resuscitation Council of the United Kingdom and the Australian Resuscitation Council have adopted the term "expired air resuscitation" (EAR), as the best description of this life-saving technique.

Other terms used in the international medical literature for EAR include:

- oral resuscitation
- exhaled air ventilation
- emergency artificial ventilation
- life-supporting first aid ventilation
- expired air ventilation

The use of such mixed terminology can lead to confusion, as can the various popular terms such as "mouth-to-mouth", "the kiss of life", "the breath of life" and "life breathing". The universal use of "expired air resuscitation" and its acronym "EAR" is therefore recommended.

EAR may be performed directly:

- mouth-to-mouth
- mouth-to-nose
- mouth-to-both mouth and nose
  (eg on infants and small children)
- mouth-to-adjunct
  (eg mouth-to-mask, mouth-to-stoma tube)

EAR may be performed while supplementing the rescuer's expired air with oxygen, thus raising the oxygen concentration or partial pressure of oxygen in the expired air being blown into the patient's lungs. This is called "oxygen-supplemented EAR" (Figure 7.2).

THE HISTORY OF EAR

> *The good things in history are usually of very short duration, but afterwards have a decisive influence over what happens over long periods of time. (Hannah Arendt, 1971)*

The modern techniques of EAR were pioneered in the 1950s. Prior to this decade, few immediate, effective methods existed for first-aid respiratory rescue. Isolated forerunners of the technique are documented, beginning with possible clues in the Bible:

> *...and his* [the woman's son] *illness was so severe that there was no breath left in him...Then he* [Elijah] *stretched himself upon the child three times,...and the soul of the child came into him again, and he revived.*

In 1771, Tossach resuscitated a near-asphyxiated coal miner with respiratory arrest using mouth-to-mouth ventilation, and at about the same time the "Practical version of the rules of the Humane Society for recovering drowned persons" included a description which is startlingly similar to present-day EAR.

It is likely that down the centuries other such successes and insights occurred in many countries, well ahead of their time, and that these were either never documented or recorded in inaccessible languages or in obscure or destroyed sources.

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Figure 7.1 *Correct technique for expired air resuscitation (EAR). Note that the rescuer is not inhaling any of the patient's expired gases while she is watching the fall of the casualty's chest.*

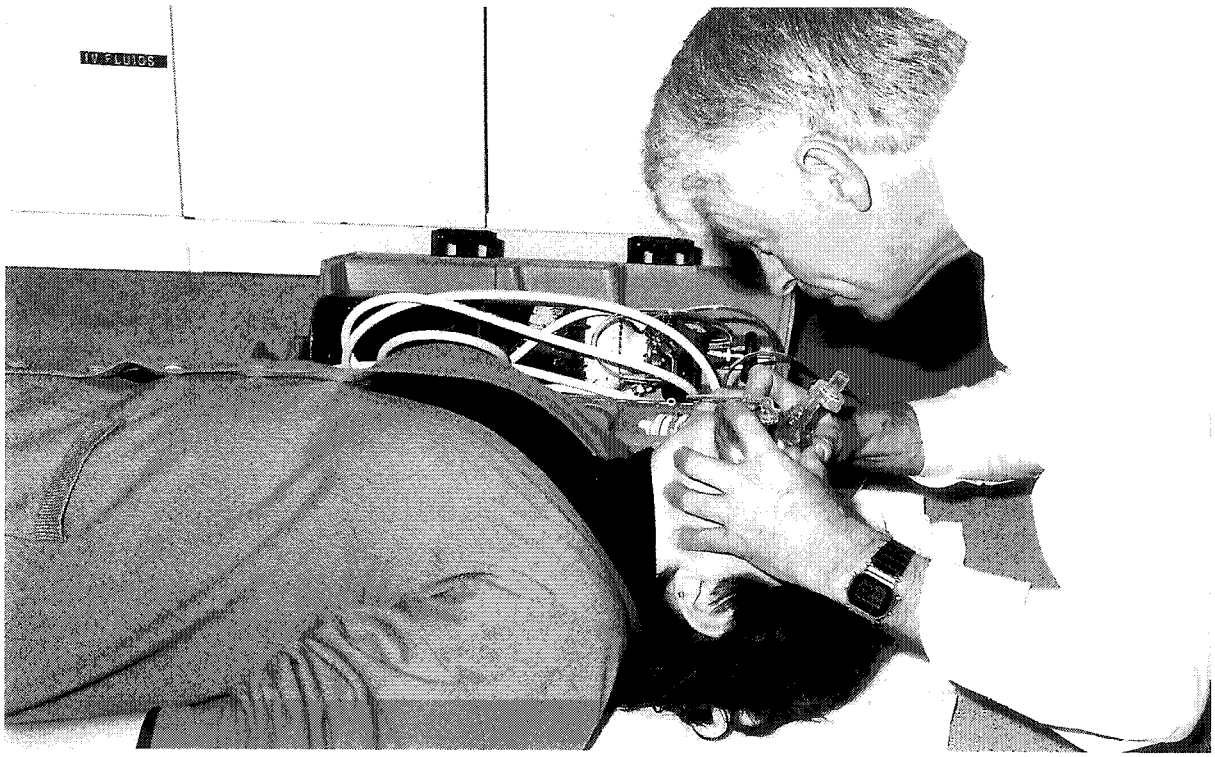


Figure 7.2 *Oxygen supplementation mouth-to-mask EAR. Note the C-size oxygen cylinder delivering continuous flow oxygen via a catheter passing between the rescuer's index fingers, and attached to the oxygen nipple on the mask. The rescuer's expired air is being enriched under the mask with the added oxygen.*

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PHYSIOLOGICAL EFFECTS OF EXPIRED AIR

Normal inspired air contains 20.93 per cent by volume of oxygen (O_2). Medically this is described as the fraction of the inspired mixture (in this case air) which is O_2, and is represented by the symbol "FIO_2". Thus the FIO_2 of air is 0.2093, or 0.21 (or even 0.20 for convenience). Most of the rest of air (about 80 per cent) is nitrogen (N_2). It should be noted, despite widely held misconceptions to the contrary, that there is almost no carbon dioxide (CO_2) in normal air. Thus 21 per cent of air being oxygen results in a partial pressure of oxygen in air equal to 21 per cent of 1 atmosphere of pressure (760 millimetres of mercury (mm Hg), or 102 kilopascals (kPa)), which is 159 mm Hg (21.3 kPa), but only at, or a little above, sea-level. With increasing altitude the atmospheric pressure decreases, so that the partial pressure of O_2 will fall, although the percentage of O_2 in the air (FIO_2) will remain the same. It is the partial pressure of the oxygen which drives the oxygen from the alveoli of the patient's lungs, across his or her alveolar-capillary membrane, and into the blood flowing through the lungs.

Expired air contains 16–18 per cent oxygen (FIO_2 = 0.16–0.18). This has been shown to be an adequate amount of oxygen for effective resuscitation and preservation of a patient's brain, provided that

1. the patient's airway is not obstructed (see Chapter 6);
2. the patient's lungs are normal;
3. the rescuer uses a tidal volume (single breath volume) of ideally about twice normal for the patient (normal adult tidal volume is about 400–500 millilitres; for children tidal volume is about 10 millilitres per kilogram of body weight). In practice, this means observing a definite chest movement in the patient, during EAR ("blow, look, listen, and feel") (see Figure 7.1);
4. EAR is not being performed at extreme altitude, where O_2 supplementation of the rescuer's expired air would become necessary; and
5. the patient's blood is circulating adequately.

Effective EAR may achieve a partial pressure of oxygen in arterial blood (PaO_2) of greater than 70 mm Hg (9.4 kPa) (normal 90–100 mm Hg or 12.0–13.4 kPa), which corresponds to a haemoglobin saturation of over 90 per cent (normally 97–99 per cent) (Figure 7.3).

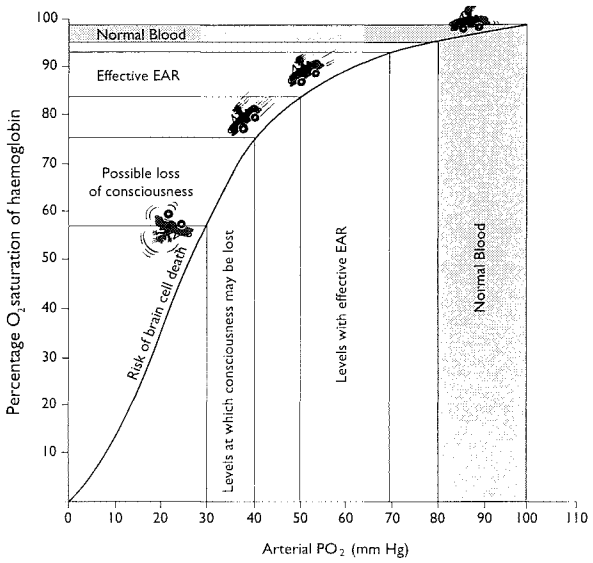


Figure 7.3 *The human oxygen haemoglobin (Hb) dissociation curve (pH 7.40 and temperature 38°C) in arterial blood (the blood oxygenating the brain). Note that expired air from a rescuer (at least 16 per cent O_2) provides a partial pressure of oxygen in the patient's arterial blood in the range 50–70 mm Hg (6.7–9.4 kPa). This is compatible with preservation of normal brain function, and corresponds to an Hb saturation with O_2 of around 90 per cent (normal range in health about 97–99 per cent). It is well above oxygen levels (partial pressures) which do not support consciousness, when brain cell death may occur. Note also that at oxygen levels below those achieved by EAR, any further fall in PaO_2 is accompanied by a precipitous decline in HbO_2 levels (the "runaway car" on the steep portion of the dissociation curve!).*

Effective EAR will also usually result in CO_2 partial pressures in the arterial blood ($PaCO_2$) of the patient of about 30–40 mm Hg (4.0–5.3 kPa) (normal about 35–44 mm Hg or 4.2–5.7 kPa), and in the rescuer of about 20–30 mm Hg due to the hyperventilation that may be associated with effective EAR.

Such partial pressures in the patient's arterial blood are achievable when EAR alone is being performed (ie on a non-breathing patient who has a palpable pulse). During cardiopulmonary resuscitation (CPR) there is

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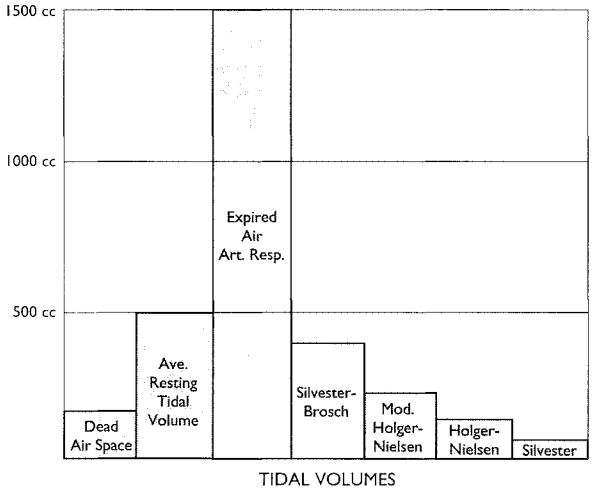


Figure 7.4 *A quantitative comparison between the different methods of respiratory resuscitation, of tidal volumes delivered into a normal adult airway. Note that the old "arm-lift, chest-compression" techniques fail to deliver even one tidal volume. Note also the clear superiority of expired air resuscitation.*

no breathing and no palpable pulse; in this case the blood gas partial pressures will be less favourable, especially during prolonged CPR.

EAR ON PATIENTS WITH A TRACHEOSTOMY OR A TRACHEAL STOMA

The fact that a victim may actually have a tracheostome may not always be immediately obvious to rescuers.

Attempts at EAR at the mouth or nose will simply produce a massive air leak in the neck, which should be uncovered to reveal the tracheostome. The presence of a tracheostomy tube may permit the rescuer to place his or her mouth over the tube protruding from the patient's neck, to seal with the lips, and to blow into the patient's trachea and lungs in the normal way. If the tracheostomy tube has no endotracheal cuff, or this is not inflated, it will be necessary for the rescuer to seal the patient's nose and mouth during inflations. It may also be necessary to seal around the tracheostomy tube where it penetrates the patient's neck. However, in some cases with an inner and outer tube the inner tracheostome tube may have to be removed first, to overcome the gas leak back around a loose inner tube. It is important not to remove the outer tracheostomy tube, as this may result in loss of access to the patient's trachea, and re-insertion may be impossible.

A simple tracheal stoma without tubes in the patient's neck may be treated as a "mouth" in the neck. Once again, the patient's nose and mouth may require to be sealed with the rescuer's hand during inflations.

IMPORTANCE OF MINIMAL DELAY IN COMMENCING EAR

There is now longstanding and copiously documented evidence that brain preservation and a successful outcome from EAR are directly related to the brevity of the time during which the patient's brain was hypoxic. It is essential to appreciate that while several *in vivo* physiological mechanisms exist for coping with short-term excesses of acid and CO_2 that may occur in a patient requiring and receiving resuscitation, the primary target organ of all resuscitation, the brain, carries no reserves of oxygen. Under worst possible conditions of global brain ischaemia (severely inadequate or absent cerebral perfusion) coma occurs in 5–10 seconds, and brain oxygen stores are exhausted, with the electroencephalogram becoming isoelectric (flat) within 15–20 seconds. However, this does not mean that brain function is not recoverable. Fortunately the healthy human myocardium, although progressively affected by hypoxia, is more resistant to hypoxia than the brain.

In the patient who has ceased to breathe adequately, but who still has a readily palpable carotid pulse (eg as may occur in near-drowning, or drug overdosage), the march of hypoxia has already begun. But provided oxygen from a rescuer's expired air ($FIO_2 = 0.16$) can be promptly delivered to the patient's pulmonary capillary blood by effective EAR, worsening brain hypoxia can be halted and even partially corrected. Simultaneously, the

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heart muscle will also be rescued from the hypoxic threat, and will keep beating, thus effectively propelling the now oxygenated blood to the brain.

Time is of the essence. Direct EAR requires no equipment, is always available to a rescuer, is always superior to richer oxygen mixtures delivered minutes later, and should never be delayed by the search for, or the use of, unfamiliar equipment.

PERCEIVED OBJECTIONS TO PERFORMING EAR

1 Aesthetic objections

Acceptance of the responsibilities of resuscitation invariably presents the rescuer with unpleasant and stressful situations. The destitute, the dirty, the near-drowned, the facially traumatised, and the vomiting patient all present objectionable aspects to the performance of EAR. Without EAR, such apnoeic patients (many of whom are young) will surely deteriorate and usually die, or suffer permanent brain damage. Fortunately most first aid resuscitators in the past, the world over, have not hesitated to "put the patient first". Simple and rapid measures, such as wiping the patient's mouth and nose clear, performing EAR through a clean handkerchief, employing mouth-to-nose or mouth-to-mask EAR, and the use of EAR "face-pieces" all assist in this regard. The risk of disease transmission from patient to rescuer (and vice versa) is not high, but introduces new and real difficulties which may compel the introduction of routine mouth-to-adjunct ventilation techniques.

2 Rescuer/patient cross-infection risks

Despite a heavy emphasis on the risk, there have been few documented cases of actual disease transmission to date, and none of HIV infection, as a result of the performance of EAR. Circumstantial evidence would support the advice that normal routine personal hygiene precautions, such as wiping the face of a patient clear prior to commencement of EAR, and washing hands after performing EAR, offer significant protection. It would appear on theoretical grounds at least that ulceration of the rescuer's skin around the lips, or, in the case of an infected rescuer, trauma or ulceration of the patient's oro-nasal skin, carries a small risk of cold sores and of hepatitis B and hepatitis C transmission.

Hepatitis transmission

While media and political over-reaction has emphasised the risk of Acquired Immune Deficiency Syndrome (AIDS) transmission, the risk of viral hepatitis (especially hepatitis B) is far greater for EAR exponents. There are also other possible diseases which are relevant.

AIDS

The AIDS virus (HIV) is inactivated in 10 minutes by alcohol or hypochlorite solution, and AIDS is not transmitted by casual personal contact through intact skin, by touching inanimate surfaces, or via normal air. At the time of writing there has not been a single documented case of AIDS transmission during EAR, either to or from a rescuer. With their impaired immune resistance, it is well to remember that the AIDS patient may be more at risk from than to the rescuer! However, a theoretical risk would appear to exist when the skin is breached, so mouth-to-adjunct EAR is logical and available.

3 Complications of EAR

These are remarkably few and, when balanced against the clear benefits, pale into insignificance. There are some risks in maximum backward head tilt if the spine is damaged (see Chapter 6). Stomach inflation may occur, especially in the presence of partial airway obstruction, when a pause for passive exhalation of the patient's lungs is not employed during EAR, or in infant patients. This will predispose them to regurgitation of stomach contents into the pharynx, with the attendant risk of aspiration into the trachea and lungs. Thus maintenance of an absolutely unobstructed airway at all times, and the avoidance of epigastric compression, are necessary during EAR. As pointed out above, concern about the amount of CO_2 in the rescuer's expired air (about 4–5%) is groundless.

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4 Dangers from absorbable drugs and poisons

Inhalational poisons (eg carbon monoxide, cyanide, methylene chloride, hydrogen sulphide) and trans-cutaneous poisons (eg organic phosphate compounds and cyanide liquids) cause rescuers to be fearful of performing EAR on persons affected by these poisons. Employing common-sense precautions (see above) and correct EAR technique (see Figure 7.1) to avoid direct poison contact or inhalation, there is to date no evidence of harm befalling a rescuer performing EAR. However, mouth-to-mask EAR (see Figure 7.2) is recommended.

5 Low pulmonary compliance ("stiff lungs") situations

Severe acute asthma, respiratory anaphylaxis, acute pulmonary oedema, and near-drowning all produce "stiff lungs". Such patients in need of EAR will usually succumb to hypoxia if EAR is not promptly administered. It should not be withheld, despite the attendant inflation difficulties. It is of particular importance to persist with EAR in near-drowned hypothermic children. Near-complete brain recovery after 60 minutes of total immersion in ice-cold water has been documented!

IN-WATER EAR FOR THE NEAR-DROWNED WITH A PULSE PRESENT

This is an advocated technique in some first aid training programmes, and has been used with success by expert rescuers. It can be performed with or without a mask. However, except when the rescuer is highly practised, or can stand in shallow water, it is very difficult to perform in practice. Even for the expert, pre-requisites for performing deep in-water EAR are: considerable in-water skill, positive buoyancy of the patient, reasonably calm weather, assistance with patient towing, or an anticipated significant delay before leaving the water is possible. The disadvantages of deep in-water EAR are difficulties in clearing and protecting the airway, and of diagnosing circulatory arrest. Treating the latter in-water is impossible, and in-water EAR attempts will delay its instigation. There is also a need for early administration of 100 per cent oxygen in near-drowning, and for all injured scuba divers; irrespective of the nature of the aspirated fluid in the near-drowned, the dominating threat is acute brain hypoxia.

SUPERIORITY OF EAR OVER CHEST-PRESSURE ARM-LIFT RESUSCITATION METHODS

The former chest-pressure arm-lift methods of artificial first-aid ventilation must never be substituted for EAR. There is virtually never an indication to do so, and none of these former methods sustain adequate tidal volumes for brain survival (see Figure 7.4).

PERFORMANCE OF EAR ON ADULTS BY CHILDREN

Children can be trained to perform effective EAR. The limitations for their rescuing adult patients with EAR are the smaller tidal volume they can deliver, and physical fatigue. Nevertheless, a child rescuer for an adult with respiratory arrest is better than none, may save a life, and has indeed done so.

OXYGEN-SUPPLEMENTED EAR

During EAR, the rescuer may increase the FIO_2 of his or her expired air above 16–18 per cent by taking a breath of an oxygen-enriched mixture from a suitable apparatus, prior to each patient inflation. A demand-flow oxygen valve is ideal for this purpose. Alternatively, the rescuer may allow a continuous-flow oxygen catheter, delivering oxygen at about 2–4 litres per minute, to lie between his/her lips, or a mask and the patient's face (see Figure 7.2), during inflation of the patient. Such mouth-to-mask EAR in practised hands would be expected to increase the FIO_2 for the casualty to approximately 50 per cent.

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CONCLUSION

Expired air resuscitation is a manual skill. Full competence can only be achieved by repeated practice on properly designed manikins. Full precautions for manikin hygiene, attention to aesthetics and prevention of cross-infection of students, are essential. EAR should not be practised on live, conscious, breathing persons. Effective EAR must be preceded by, and is absolutely dependent upon, the establishment and maintenance of an unobstructed patient airway (see Chapter 6). EAR is tiring for the out-of-practice rescuer, and when combined with external cardiac compression is exhausting. When two person CPR is in progress, the more experienced rescuer should perform the EAR role. The maintenance of optimal EAR skills requires at the very least annual manikin practice, but one lesson, or even watching a video, may lead to the saving of a life. EAR is a dramatically successful resuscitation technique.

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CHAPTER EIGHT

EXTERNAL CARDIAC COMPRESSION

Nicholas G. Bircher

Cardiopulmonary resuscitation (CPR) ought to comprise an important part of the skills of every citizen. However, many of the key aspects of CPR are performed poorly by those with limited training. The first aider or paramedic delivers these essential skills in the pre-hospital setting. Without these skills being swiftly applied, any patient experiencing cardiac arrest will surely die.

Optimal patient care requires a complete knowledge of CPR techniques as well as the evidence concerning their use. Cardiopulmonary resuscitation is divided into three phases, each of which has three steps (Table 8.1)

This chapter includes a consideration of both the scientific and the clinical principles of artificial circulation by chest compressions. This chapter is not designed to present basic skills; these are presented, as drills, in all reputable books on first aid.

Table 8.1 Cardiopulmonary resuscitation (CPR) has three phases each with three steps. A successful resuscitation means that a victim has passed successfully through these nine steps

Phase	Step
1 Basic Life Support	**A** Airway control
	B Breathing support
	C Circulation support
2 Advanced Life Support	**D** Drugs and fluids
	E Electrocardiography
	F Fibrillation treatment
3 Prolonged Life Support	**G** Gauging
	H Human mentation
	I Intensive care

INTERACTION OF VENTILATION AND CHEST COMPRESSIONS

Airway control and artificial ventilation are necessary adjuncts to chest compressions in cardiac arrest since chest compressions alone do not provide ventilation in humans. Initiation of chest compressions without ventilation results in severe arterial hypoxaemia within minutes. Endotracheal intubation should be performed as soon as feasible in all patients for whom cardiopulmonary resuscitation (CPR) is indicated. In the pre-hospital setting, this must not delay the institution of ventilation by any *immediately* available means.

Hypoxaemia is a common occurrence during CPR, and a number of experiments have been conducted to determine how extra oxygen can be transferred to the blood of the victim whose life is being maintained by CPR. One method (unsuccessful) has been to increase the positive end expiratory pressure (PEEP) of the ventilated air or oxygen during chest compressions. Unfortunately PEEP has been shown to depress cardiac output. The mechanism of the latter effect is likely to be the result of reduction of venous return to the chest. Even a brief application of PEEP can severely compromise haemodynamics during CPR. High inflation pressures can inflate the stomach. For these reasons, PEEP should not be used during external

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cardiac compression (ECC). Rather, 100 per cent oxygen should always be administered.

Ventilations alone produce some circulation, but rarely sufficient to restart the heart once it has arrested. Simultaneous ventilation and compression, either manually, mechanically, or with a vest, have given variable results. Independent or asynchronous ventilations (delivered randomly with respect to chest compressions) provide oxygenation and ventilation comparable to interposed or alternating ventilations (delivered in between compressions). The ratio of interposed ventilations to chest compressions has little impact on oxygenation or ventilation until the interval between ventilations exceeds 15 seconds. High frequency jet ventilation provides adequate ventilation and oxygenation during CPR. The transtracheal route offers protection against aspiration. The necessary techniques and equipment can be mastered by skilled and experienced first aiders such as emergency medical technicians, thus obviating the need to interpose ventilations between compressions.

EMERGENCY CIRCULATION

No variety of artificial circulation offers tissue preservation on an indefinite basis. Spontaneous circulation, therefore, must be rapidly restored or the patient will die. This is facilitated by the use of algorithms or protocols.

Recognition of cardiac arrest

The prompt and accurate diagnosis of cardiac arrest is essential, as the arrest interval is among the principal determinants of outcome. Cardiac arrest is defined as the absence of a palpable pulse in a large artery, that is in the carotid, femoral or brachial artery. If the patient is truly in cardiac arrest, unconsciousness will follow rapidly. The profoundly hypotensive patient, however, may be able to sustain consciousness. A pulse pressure of less than 5 millimetres of mercury (mm Hg) constitutes cardiac arrest as measured by an arterial line. The diagnosis of pulseless electrical activity (electro-mechanical dissociation) is imprecise because the absence of a clinically palpable pulse does not necessarily mean cessation of mechanical activity of the heart.

Fundamental physiology

Fluctuations of intrathoracic pressure (ITP) cause blood flow during cardiac arrest in a variety of circumstances. A longstanding debate continues about the extent to which cardiac compression contributes to flow. A diffuse rise in intrathoracic pressure cannot by itself account for blood flow, since it cannot produce a pressure gradient for flow inside the chest. The peripheral vasculature plays a critical role in the generation of pressure gradients for forward flow during CPR. The valves of the jugular venous system attenuate pressure transmission to the peripheral veins of the head, and the inferior vena cava functions variably as a Starling resistor. Because arteries transmit an unattenuated pressure, there is a peripheral arteriovenous perfusion gradient and consequently blood flows.

Relatively little haemodynamic data is available concerning human external CPR. Aortic pressures and cardiac output are inferior to direct cardiac massage. Measurements of arterial and venous pressures tend to support the "thoracic pump" mechanism of blood flow. Duration of compression is a major determinant of flow in adult man. Differences in rates of compression between 40 and 80 compressions per minute do not influence carotid blood flow in adult humans, nor does increasing the compression rate from 60 to 140 alter end-tidal CO_2 during CPR. Experimentally, depth of compression shows a threshold of effectiveness, so depth of compression should be evaluated clinically to ensure that it is sufficient to produce a femoral or carotid pulse. Intermittent abdominal compression (IAC) and active compression decompression (ACD) CPR appear promising means of improving flow but require demonstration of consistently improved outcome.

The brain during CPR

High central venous pressures during chest compressions are partially transmitted to the sagittal sinus. In most circumstances, intracranial compliance is not

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Figure 8.1 *Cardiac arrest.*



Figure 8.2 *The first aider establishes that the airway is clear.*



Figure 8.3 *Breathing support.*



Figure 8.4 *Circulation support.*

Figures 8.1–8.4 *Basic life support (see Table 8.1). (Photographs courtesy of St John Ambulance Australia (NSW), 1995).*

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altered by cardiac arrest. Intracranial pressure (ICP) is only modestly elevated by chest compressions, through both direct transmission via CSF and non-valved vertebral veins.

Cerebral blood flow (CBF) during CPR is a critical concern, since the goal in resuscitation is to return the patient home with minimal neurologic sequelae. Cerebral perfusion pressure (CPP) during CPR is the difference between arterial pressure and either ICP or cerebral venous pressure; the greater of the latter two is the subtrahend. The maintenance of CPP is essential since autoregulation cannot maintain cerebral blood flow if CPP is too low. Hypoxaemia worsens the situation. Maximal CBF generated by chest compressions falls very rapidly with increasing arrest interval; thus CPR must be instituted rapidly and skilfully.

Cerebral blood flow during CPR may maintain cerebral venous oxygen tension at levels which indicate that irreversible damage has not occurred. Although the difference in outcome with complete ischaemia versus very low flow has been debated, minimal blood flow clearly is better than no flow at all. While cerebral blood flow values as high as 90 per cent of control (spontaneous circulation) have been reported during standard CPR, the majority of reported flows are in the range of 0–33 per cent of controls. Cerebral perfusion can be improved during standard CPR by epinephrine (adrenaline), is improved or unchanged by other alpha adrenergic agonists, and is made worse by isoproterenol, and by volume loading. Alternative means of performing CPR variably improve or reduce CBF but none have been demonstrated to improve outcome. In particular, despite very promising findings in the laboratory, simultaneous ventilation–compression CPR has failed to improve outcome in randomised prospective human clinical trial.

The heart during CPR

Coronary blood flow is usually exceedingly poor during standard CPR. The increase in right atrial pressure produced by chest compression in humans is nearly equal to the increase in aortic pressure, that is the perfusion pressure across the heart is nearly zero. During the relaxation phase (CPR diastole) perfusion pressures are higher and are predictive of outcome. Coronary perfusion can be improved by epinephrine (adrenaline) and this effect can be enhanced by sodium bicarbonate after prolonged arrest.

The lung during CPR

End-tidal CO_2 is known to be markedly decreased due to the profound low flow state, but CO_2 production is unknown. Dead space is estimated to be quite high (Vd/Vt to be 0.59). With initially normal lungs, PaO_2 can be maintained around 80 mm Hg with room air, but because arterial hypoxaemia is a common finding in clinical resuscitation, 100 per cent oxygen should always be administered when available.

One rescuer CPR

Current recommendations in the United States, Australia and Europe call for a rate of 80–100 compressions per minute, a ratio of two slow (1–2 seconds to deliver, to minimise peak inspiratory pressure) ventilations to every 15 compressions, a minimum tidal volume of 800 ml, and a 50 per cent duty cycle and 4.0–5.0 cm (1.5–2 inches) depth for compressions. This set of recommendations is for education of the lay public, who are unlikely to encounter a second trained rescuer just when they need one most. Health care professionals should learn both one and two rescuer techniques because two rescuers are frequently available in both pre-hospital and in-hospital settings.

Recommendations for one rescuer CPR in infants (the first year of life) and children (age 1–8 years) are similar to those for an adult, except that, for infants, research has shown that the rate of chest compression must be greater than 100, the ratio of compressions to ventilations is 5:1, tidal volume is the minimum required to make the chest rise, and compressions are delivered with the index and middle finger in the middle of the sternum with a depth of 1.5–3 cm (0.5–1 inch). In older children, compressions are performed with the heel of just one hand and to a depth of 3.0–4.0 cm (1–1.5 inches).

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Two rescuer CPR

Current recommendations call for the rate of compression for adult CPR to be 80–100 compressions per minute, a brief pause after every fifth compression to deliver a slow ventilation as above, and a ratio of five compressions to every ventilation. Since both chest compressions and ventilations are physically taxing, rescuers should train themselves in co-ordinated means of switching roles in order to avoid interruption of artificial perfusion. The energy cost to the rescuer of performing CPR has never been measured but it is a rapidly fatiguing activity. Once the trachea has been intubated, interposition of ventilations between compressions yields better oxygenation. High pressure ventilations in an intubated patient can overcome this problem but have variable effects on cardiac output and coronary and cerebral perfusion.

MONITORING THE EFFECTIVENESS OF CPR

Essential and desirable monitoring equipment

No devices are required for the evaluation of the effectiveness of artificial ventilation, oxygenation and circulation. Careful attention must be paid to inspection and palpation. Inspection determines whether or not the chest rises and falls with ventilation, the depth of chest compression is adequate, and the rescuer's hands are properly positioned. Palpation is essential to establish the clinical diagnosis of cardiac arrest, to locate landmarks, and to evaluate the quality of the pulse generated by chest compressions. These simple means of assessing the quality of CPR are valuable in the pre-hospital setting. They are not reliable, however, as it is possible to meet the criterion of clinical pulselessness and still have a feebly beating heart. For this reason technological assessment should be part of the in-hospital assessment of quality control of real life resuscitations.

Ventilations

The chest must rise and fall with every ventilatory effort. Capnography confirms endotracheal intubation and provides a useful assessment of cardiac output as well.

Circulation

Palpation of the pulse only serves to ensure that the chest compressions delivered are of sufficient depth or force to generate a palpable arterial pressure wave. Because the intrathoracic perfusion pressures are small and the extrathoracic perfusion pressures difficult to predict, the presence of a pulse may or may not be indicative of flow. This sad but true fact is startling not only to many first aiders and paramedics but to doctors as well.

Flow is typically too low to allow auscultation of arterial pressure using a cuff, though flow may be detectable using a Doppler device. An arterial line, if present, is a useful indicator of the adequacy of chest compressions, allows immediate recognition of the return of a pulse pressure, and allows repetitive sampling of arterial blood. Pulse oximetry may give some indication of the quality of circulation but end-tidal CO_2 is a much more sensitive indicator. Direct measurement of cardiac output during CPR is feasible but technically difficult due to the profoundly low flow state.

Neurological function

Neurological assessment during CPR is a very poor predictor of outcome, and decisions to continue or stop resuscitative efforts should not be made on this basis. Absence of specific neurological signs during basic life support does not preclude their prompt return following the hoped-for restoration of spontaneous circulation.

Mechanical CPR

Mechanical devices may offer some advantages during clinical CPR. Early versions had a wide variety of problems, but currently available devices offer consistent compressions and higher arterial pressures in humans. They have never been shown to influence outcome in patients, however.

Complications of chest compressions

The complications of chest compressions are legion, but

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unavoidable complications are of course acceptable compared to an otherwise certain death. On the other hand, many potentially fatal complications are avoidable. Hepatic trauma was common early in the modern era of CPR and can lead to rapid exsanguination. The recently reported incidence is less. Gastroesophageal damage was also a remarkably frequent complication, which carries the risk of fatal mediastinitis or haemorrhage in spite of restoration of circulation and consciousness. Reduced gastric damage may have resulted from attention to landmark location, limiting gastric insufflation by cricoid pressure, earlier intubation or gastric decompression.

Mild cardiac contusions following ECC are routine in the laboratory. Clinically they are small haemopericardia or pericardial effusions. Laceration or rupture of the heart or the great vessels is rare, as are papillary muscle rupture, atheromatous emboli, and subclavian thrombosis. Direct mechanical trauma to the lung is ordinarily limited to the hilum but barotrauma tends to be parenchymal. Aspiration of gastric contents or blood, pneumonia and adult respiratory distress syndrome are also common in survivors.

Bony trauma is very common and typically involves ribs and sternum and rarely vertebrae. However, not all patients receiving chest compressions have rib fractures. Haemothoraces secondary to rib fractures can be substantial and may contribute to the relative or absolute hypovolemia of the patient. Bone marrow emboli are common sequelae of CPR but can also occur from other causes.

PHARMACOLOGIC SUPPORT OF ARTIFICIAL CIRCULATION

Virtually any secure peripheral intravenous route will allow access for the administration of drugs used in resuscitation.

Central venous access is not necessary but offers rapid drug delivery and higher peak levels, though these are not entirely reliable. Delay in restarting the heart should not be caused by lack of intravenous access. Alternatives include intratracheal, intraosseous, intra-arterial, and intracardiac administration of drugs.

Aside from oxygen, epinephrine (adrenaline) is the single most useful drug currently available for the treatment of cardiac arrest. There is now an enormous body of evidence supporting the use of epinephrine (adrenaline) during CPR. Withholding this drug if CPR and defibrillation fail to restore spontaneous circulation immediately can rarely be justified. Optimal use of epinephrine requires that all other components of an ongoing resuscitation be conducted efficiently. Both metabolic and respiratory acidosis limit its effectiveness, as does hypoxia. Although epinephrine does not alter the rate of successful defibrillation after brief periods of cardiac arrest, it does increase the rate of return of spontaneous circulation. This effect appears to be a combination of the increased myocardial perfusion provided during CPR and the increase in myocardial contractility which accrues immediately after restarting the heart. Although there is a dramatic increase in coronary blood flow with epinephrine, there is a down side. The beta adrenergic stimulus can limit the degree of improvement of the ratio of either subendocardial to epicardial blood flow or myocardial oxygen supply to demand. Some patients may benefit from higher doses of epinephrine than those presently used clinically, but the optimal dosing regimen remains unknown. In pigs, raising the dose from 0.02–0.2 milligrams per kilogram improves myocardial blood flow, but four randomised clinical trials in adults have failed to demonstrate improved outcome. Epinephrine is the drug most commonly administered via the endotracheal tube during CPR, but this route of administration is not entirely reliable.

While it is erroneous to automatically use buffer therapy, especially for arrests of brief duration, or in an attempt to correct acidosis of respiratory origin, the correction of severe metabolic acidosis after prolonged periods of complete circulatory arrest enhances the effect of epinephrine and improves outcome in animal models. Unwitnessed cardiac arrest associated with severe acidosis is a frequent phenomenon in hospital wards and in the pre-hospital setting. Similarly,

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prolonged resuscitation efforts require bicarbonate to maintain physiological blood gases in spite of early CPR and aggressive hyperventilation. Clinical administration of sodium bicarbonate must be done judiciously, titrated to the treatment of severe metabolic acidosis documented by arterial blood gases if possible, but with the aim of enhancing the effect of epinephrine on the circulation during chest compressions.

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CHAPTER NINE

PAEDIATRIC CARDIOPULMONARY RESUSCITATION

Linda Quan

To resuscitate a child is to give perhaps seventy years of life. The factors which cause infants and children to stop breathing are generally quite different from those which strike adult victims. The discipline of paediatric CPR is an evolving one with much still to be learned.

The special features which characterise the resuscitation of children were first recognised in the first aid world in 1980 when the American Heart Association wrote the first national guidelines specifically for paediatric cardiopulmonary resuscitation. However, much of these guidelines was predicated on existing adult CPR guidelines and experience with adult resuscitation. Since paediatric resuscitations are rare encounters in the pre-hospital as well as the hospital setting, the scientific data on paediatric cardiopulmonary resuscitation has just begun to emerge. Unfortunately, the data to support a different approach from adult CPR lags behind an increasing interest in this crucial area of paediatric care.

In 1992, the American Heart Association revised its Pediatric Basic and Advanced Life Support Guidelines. The changes were based on three themes.

1 Changes would be dictated by scientific evidence. Yet even within that realm the strength of supporting data varies; a rating scale was used to reflect the strength of the recommendation.
2 Drills for paediatric CPR would be similar to those taught as the doctrine of Basic Life Support and Adult Life Support wherever the data permitted.
3 Every effort would be made to simplify the guidelines. Feedback from professional providers and lay persons had made it clear that the complexity of the CPR sequences and the emphasis on performance were counter productive. It had been found that would-be CPR providers were deterred from attempting resuscitation in real life situations; or, having "had a go" developed feelings of failure or wrongdoing, especially if the victim did not respond. Thus, the 1992 Guidelines represent a practical as well as scientific approach to treating cardiac arrest and an evolutionary step from an existing set of guidelines.

This chapter looks at the underlying scientific base which leads to current CPR drills to resuscitate children.

PAEDIATRIC BASIC LIFE SUPPORT (PBLS)

Assess the victim for possible trauma

When assessing a victim of cardiac arrest, the provider must be alert to the possibility of trauma, especially in the child found in cardiac arrest out of hospital. Data on paediatric pre-hospital cardiac arrests shows that an injury is the most likely underlying etiology of cardiac arrest (Figure 9.1), when victims of Sudden Infant Death are excluded. Usually, the injury, such as drowning, closed head injury, asphyxiation or suffocation results in hypoxia/anoxia which causes the cardiac arrest. However, any injury such as those involving motor vehicles or bicycles, falls, diving, or electrocution should alert the first aider to protect the patient's cervical spine during resuscitation.

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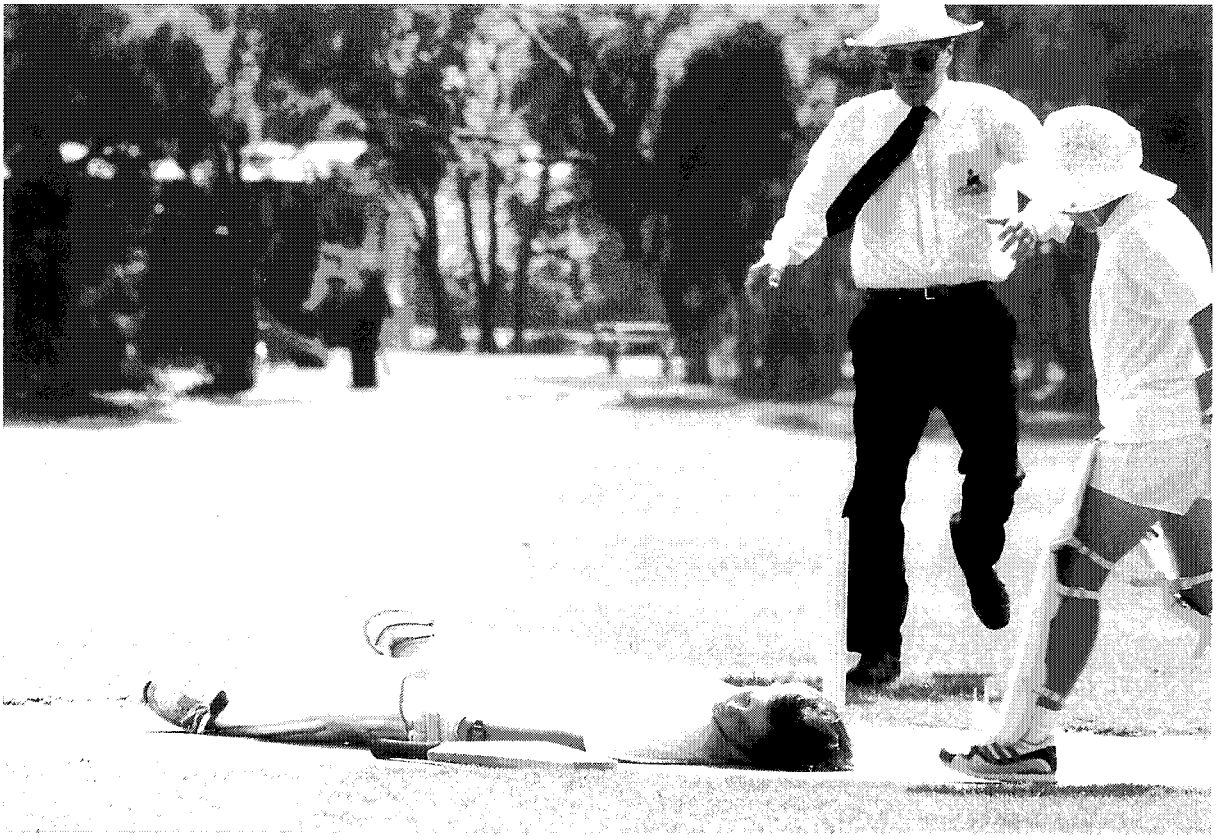


Figure 9.1 *Trauma, in this case a blow to the head by a cricket ball, may lead to the necessity for child resuscitation. (Photograph by Stephen White, courtesy of St John Ambulance Australia (WA).)*

Protection of the cervical spine necessitates careful handling of the child to avoid aggravating an existing spinal cord injury. Therefore, immobilisation of the cervical spine and the jaw-thrust are used to achieve airway alignment. Any concern for the cervical spine should not take precedence over the assessment and institution of breathing and circulation. Fortunately, cervical spine injury is relatively rare in young patients while airway compromise and failure to maintain the airway and circulation are not uncommon problems in paediatric trauma. Ideally, cervical spine protection and attention to the ABCs (airway, breathing, circulation) can be accomplished simultaneously.

Determine unresponsiveness and breathing; provide Basic Life Support (BLS) for one minute

Once the rescuer determines that the accident scenario is safe and that the victim is unresponsive, the lone rescuer should be prepared to provide Basic Life Support (BLS) to the child for one minute. This recommendation reflects the data from most studies of paediatric cardiac arrest that the cause of the arrest is likely to be respiratory. This holds true for paediatric cardiac arrest victims in hospital as well as pre-hospital. The rapid provision of BLS may restore oxygenation and effective ventilation. In addition, the effectiveness of this approach has been well described in child drowning victims who are blue and

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A



B

Figures 9.2 A and B *Near-drowning. A – The worst sight a parent can see in their backyard pool. B – The first step towards a happy outcome – clearing the airway. (Photographs courtesy of St John Ambulance Australia (NSW).)*

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apnoeic when retrieved from the water; rapidly provided CPR or even just expired air resuscitation often immediately revitalises the victim (Figures 9.2A and B).

Align the airway, check breathing

The rescuer aligns the airway using the head tilt-chin manoeuver, or the jaw-thrust if a neck injury is suspected. Airway alignment remains a critical technique in paediatric life support. Alignment in the infant is achieved by placing the head in the neutral position. This achieves alignment because the infant's head is relatively so large that it lifts the neck into slight extension. Indeed, if the infant's head is tilted up, the neck becomes overextended. The infant's trachea, because of its pliability, can actually obstruct itself in hyperextension. Only as the child approaches adult head:body proportions does the adult technique of achieving airway alignment with hyperextension become appropriate. What is clear is that no one position will reliably achieve airway alignment for all paediatric age groups. Therefore, the rescuer must watch carefully for chest rise as the indicator of adequate ventilation. If ventilations meet resistance or do not produce chest rise, the rescuer must reposition the child's head in an effort to find empirically the position that provides the best airway alignment.

If the victim is not breathing, provide two slow rescue breaths

The goal is to provide ventilation and oxygenation without incurring the risks of overventilation. For this reason each of these breaths and each breath delivered during CPR should be delivered over 1–1.5 seconds.

A recent important change in the Basic Life Support procedures is the manner in which respirations are delivered. Data from animal and human studies show that slow delivery of breaths increases the volume of air ventilating the lung while generating lower pressures. The lower pressures generated minimise the amount of air entering the oesophagus and, therefore, minimise the amount of gastric ventilation. Therefore, this slow-delivery method decreases the risk of vomiting with the subsequent aspiration of stomach contents.

The unfounded fear of disease transmission from adult victims has led to the development of several barriers such as face shields. While several alternative devices exist for mouth-to-mouth ventilations, a present lack of data precludes the recommendation of any specific device.

If the victim is not breathing, also provide chest compressions

While the next rescue step is to assess circulation, the Pediatric Basic Life Support (PBLS) Guidelines de-emphasise determining the heart rate. It must be remembered that PBLS is directed to the lay person, in contrast to neonatal and paediatric Advanced Life Support (ALS) which is for medical and paramedical health-care providers. Most paediatric cardiac arrests occur in the home or when children are under the supervision of their parents or another caretaker. Two studies show that lay persons have difficulty locating and determining the paediatric pulse. An older study showed that the brachial artery pulse was most reliably detected by parents. This same study found that a precordial pulse is not reliably palpated despite good cardiac function and should not be recommended to lay persons. A subsequent larger study found that the brachial pulse was not reliably located and counted by adults. Such confusion wasted precious minutes, delaying the initiation of CPR. Therefore, since the non-breathing victim inevitably becomes pulseless, compressions should be started by the rescuer as soon as apnoea is determined. The professional health care rescuer can, of course, assess the pulse. However, the ability to determine the pulse has not been evaluated among the many health-care provider types.

The concern raised is: are there risks to cardiac compressions? Radiologic and pathologic studies have shown that cardiac compressions do not produce the complications in the paediatric victim that are seen in the adult; rib fractures do not occur, probably because the paediatric rib cage is so pliable. In recent studies, no rib fractures were found in 50 children or 73 infants following chest compressions. The benefits of providing cardiac compressions, therefore, seem to outweigh the low risk of complications.

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In the 1980s much cardiac arrest research was focused on determining which mechanisms are responsible for blood flow during CPR and how best to provide chest compressions. Early in the history of CPR, the effectiveness of closed chest compressions was believed to be due to direct compression of the heart between the sternum and vertebral bodies. In this conceptual model chest compressions compress the heart during CPR; the heart thus functions as a pump, forcing blood forward into the systemic circulation. In the 1980s, a new concept, that of the thoracic pump, developed when Criley noted that coughing during cardiac arrest with ventricular fibrillation could maintain forward blood flow. In this model, chest compressions produce changes in intrathoracic pressures which cause passive flow of blood in and out of the thorax; the heart is purely a conduit in this theory. Echocardiographic studies of the heart support this theory as do the findings that pressures in all four cardiac chambers, major vessels, and the intrathoracic space during CPR are the same. This led to the development of vest CPR, simultaneous chest/abdominal compression CPR, and simultaneous chest compression and ventilation CPR. These alternative CPR methods increase intrathoracic pressure during CPR and might be expected to improve cardiac output during CPR if the thoracic model is the correct underlying mechanism. However, when studied, these newer techniques do not offer an advantage over conventional CPR. There are some data, including echocardiographic studies, which support the cardiac pump theory. A still viable heart's mitral valves close during chest compressions, the heart's area decreases, and forward flow occurs. Clinical experience with paediatric patients supports the cardiac pump model as the best explanation for resuscitation success.

Currently, research in adults or adult animals, supports the concept that both the cardiac pump and the thoracic conduit mechanisms have a role in maintaining cardiac output during CPR.

The ongoing support for the cardiac pump model in paediatric CPR supports the practice of applying maximal pressure over the heart. Studies of paediatric cadavers show that the paediatric heart is situated high in the chest. However, subsequent studies in living infants show that the paediatric heart is situated behind the lower third of the sternum. Thus, the recommended changes in finger/hand placement represent an effort to achieve correct placement for maximal CPR results.

Proper finger or hand placement for chest compressions is considerably simplified in the 1992 Guidelines. The goal is to apply pressure on the lower sternum below the intermammary line but above the xiphoid so that xiphoid fracture does not occur. This may be accomplished in the infant by placing fingers one fingerbreadth below the intermammary line, and in the child by placing the heel of the hand one fingerbreadth above the xiphoid notch.

While the 1992 Guidelines recommend slower respirations, they recommend faster chest compressions. The recommended rate of 100 or more cardiac compressions per minute for all age groups has two bases. One is based on the higher normal heart rate of the young paediatric age group. The other is based on adult animal and human studies of CPR demonstrating greater effectiveness with chest compression rates greater than 100. However, compression rates greater than 100 per minute were not recommended because two studies using "paediatric" animals were unable to show improved outcomes with chest compression rates greater than 100 per minute. These different findings raise important issues about research in "paediatric" animal studies. First, what is effective for the adult may not be effective for the paediatric patient; paediatric physiology differs from adult physiology. Secondly, the ideal study animal approximates the human physiology; which young animal type does this best is still to be determined. Happily, the chest compression rate recommendation of 100 per minute for all age groups after the newborn period simplifies paediatric Basic Life Support.

The delivery of chest compressions should be smooth and non-jerky. The compression phase should approximate no greater than half of the cycle. These recommendations are based on studies showing that the decompression or lift phase is the period in which

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cardiac filling occurs. With a longer decompression or uplift phase, the greater the cardiac filling and subsequent cardiac output in the paediatric model.

At the end of every fifth compression, a pause should occur during which a ventilation is delivered

Interposing breaths and chest compressions results in better cardiac output from compressions; it also delivers better ventilations. Alternative CPR methods have been the subject of many recent studies. At this time the efficacy of interposed abdominal compression CPR, simultaneous chest and abdominal compression CPR, and vest CPR in the adults and animals studied remains confusing. These alternatives to standard CPR have not been well studied in the paediatric patient or paediatric model. The only alternative evaluated, that of simultaneous compressions and ventilations, showed no advantage over standard CPR in a paediatric animal study.

Activate emergency medical services (EMS)

After one minute of basic life support pre-hospital emergency medical services should be activated by the lone rescuer. This differs from the adult BLS sequence and reflects the essential difference between paediatric and adult resuscitation. The unresponsive, non-breathing paediatric victim most likely has suffered a respiratory event resulting in hypoxia that may well respond to good ventilatory interventions. In contrast, the adult victim almost always has had a cardiac arrest due to a primary cardiac event, usually resulting in ventricular fibrillation (VF). While 80–90 per cent of adults in cardiac arrest are in VF, only 10–19 per cent of paediatric victims outside of a hospital are in VF. With these different etiologies for cardiac arrest and different likelihood of ventricular fibrillation, the initial first aid approach must also differ. The paediatric arrest victim more than likely needs ventilation, while the adult in ventricular fibrillation needs voltage, that is, early defibrillation. Therefore, for the paediatric victim, the lone rescuer delays activating EMS until he or she has provided CPR for one full minute as soon as it is determined that the victim is unresponsive. In contrast, for the adult, the rescuer should activate EMS as soon as he determines the victim is unresponsive so that defibrillators can be brought as soon as possible. The "one minute" rule, for paediatric CPR, is based on consensus and common sense and not on research studies. It may change in the future.

When the victim's breathing and pulse return, roll the victim into the recovery position. A new step has been added to protect the airway of the post-cardiac arrest victim. Once stabilised, the victim is rolled to his side with legs and arms positioned to support this position.

Routinely used post-anaesthesia, this "stable side position" or "recovery position" allows for airway alignment and positioning that will facilitate keeping the airway protected should the patient vomit. Vomit is thus more likely to pool in the mouth and run out rather than be aspirated.

NEONATAL RESUSCITATION

The newborn should be dried, warmed, positioned, suctioned, and respirations checked. If not breathing, the neonate should receive tactile stimulation. If respirations are absent or gasping *or* if the heart rate is less than 100 beats per minute, positive pressure ventilations with 100 per cent oxygen should be initiated.

If the newborn's heart rate is less than 60 beats per minute or 60–80 beats per minute and not rapidly increasing after 30 seconds of ventilation with 100 per cent oxygen, then provide chest compressions at 120 per minute with ventilations interposed in a 3:1 ratio of compressions to ventilations.

As the data on adult simultaneous ventilation and compressions (SVC) accumulated to show it had no advantages to conventional CPR, data from infant pigs showed similar results. In addition, SVC may have real disadvantages in the neonate. When SVC is provided, very high inflation pressures are needed in order to achieve ventilation. These high inflation pressures may hinder the development of the transpulmonary gradient. The transpulmonary gradient in the newborn

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is needed to clear pulmonary fluid and to provide an adequate functional residual capacity in the lung. These data led to the recommended use of conventional CPR with interposed ventilations and compressions in the neonate.

Both the two finger method and the encircling chest (thumbs) method of providing chest compressions are recommended. Two studies comparing the two methods in human infants show that the thumbs method generates greater mean arterial pressures than the two finger method. However, it may be difficult to use the thumbs method in a resuscitation in the neonate whose umbilical cord is being accessed because the rescuer's hands may interfere with access to the umbilical cord. In this setting the two finger method is logistically more feasible. If following ventilation with 100 per cent oxygen and chest compressions, the heart rate remains less than 80 beats per minute, medications should be administered.

Other than naloxone, epinephrine (adrenaline) is the sole medication used for neonatal resuscitation. While there is abundant data on the efficacy of epinephrine (adrenaline) in adult cardiac arrest, there is no human neonatal data. The effect on cerebral blood flow is of concern in the neonate because of the neonate's predisposition to have an intracranial haemorrhage following severe stress or asphyxia. Therefore, since intravenous high dose epinephrine (adrenaline) could potentiate an intracranial bleed, only intravenous standard dose epinephrine (0.01 milligrams per kilogram (mg/kg) of 1:10 000) is recommended for neonatal cardiac arrest.

FOREIGN BODY AIRWAY OBSTRUCTION

Unfortunately, there is no new data to resolve several questions regarding the chest thrust versus the Heimlich manoeuver, such as which is most effective at foreign body removal, or in which sequence, or at what age. Therefore, there are no significant changes in this area. The only change in the 1992 Guidelines was an increase to five Heimlich manoeuvers or chest thrusts delivered if needed in each sequence. This increase to five was purely to simplify for the rescuer all CPR procedures to multiples of five when possible.

ADVANCED CARDIAC LIFE SUPPORT

Airway management

In the past few years several adjuncts have facilitated the use of invasive procedures in ALS. These adjuncts are especially welcome for the emergently ill paediatric patient who is so likely to have had, or to be at risk of, respiratory difficulty. Furthermore, these adjuncts assist in the recognition of respiratory decompensation, a condition that is often difficult to recognise in the young paediatric patient. Fortunately, these adjuncts have been well studied in both the in-hospital and out-of-hospital paediatric patient.

- *Pulse oximetry* measures oxygen saturation and, therefore, provides early recognition of hypoxia. Its use improves monitoring of the pre- and post- arrest victim. Because its measurement assay is dependent upon good capillary perfusion, its use is limited to the well-perfused patient.
- *End-tidal carbon dioxide monitoring* of the intubated patient has been well studied in paediatric arrest and transport situations. By measuring carbon dioxide levels in exhaled air, high carbon dioxide readings confirm correct endotracheal placement – even in those patients in cardiac arrest. However, care must be taken regarding interpretation of low carbon dioxide levels in the patient in cardiac arrest. A low or absent reading is not useful in the cardiac arrest setting as low readings occur with oesophageal placement as well as with proper endotracheal placement in a victim with poor pulmonary blood flow from CPR.

For the victim in whom endotracheal or bag–valve–mask ventilation is impossible or contraindicated, percutaneous cricothyrotomy is recommended in lieu of surgical cricothyrotomy. The greater than 50 per cent mortality and morbidity caused by surgical cricothyrotomy precludes its continued recommendation.

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Figure 9.3 *In-hospital paediatric resuscitation. (Courtesy of Dr Jeremy Raftos, Director of the Paediatric Emergency Department, Women's and Children's Hospital, Adelaide.)*

While percutaneous cricothyrotomy will not provide adequate ventilation, unless jet insufflation is used, it will allow some measure of oxygenation. This may keep a victim alive for a short period of time.

Vascular access

Of all recent changes, the most gratefully received is the use of intraosseous access for the delivery of ALS medications. A profusion of studies in the past five years demonstrates the efficacy and safety of this technique in providing adequate drug levels. Risk of infection, specifically osteomyelitis, was less than 1 per cent following intraosseous placement in large older studies and was associated with long-term use of intraosseous access. Recent studies demonstrate that intraosseous access and delivery of medications do not damage the tibial growth plate when appropriate landmarks are used, nor do they damage the marrow, regardless of the medication infused. In addition, fluid resuscitation can be accomplished with the use of pressure bags or syringe pushes; in the trauma situation, a second, vascular access line is always indicated.

Medications

The most discussed and controversial paediatric ALS recommendation has been epinephrine (adrenaline) dosing for the treatment of asystole. By increasing aortic systolic and diastolic pressures, epinephrine (adrenaline) increased coronary artery perfusion. Coronary artery perfusion has been the single most important predictor of outcome following CPR. Epinephrine's effect on coronary artery perfusion should improve cardiac responsiveness, an effect that was shown in studies conducted in the 1980s in animals treated with high doses (0.045–0.2 mg/kg) of

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Figure 9.4 *An optical illusion? A basketball falling from a roof towards the head of an unsuspecting toddler, or a basketball floating on the surface of a swimming pool above a submerged toddler? In either event, resuscitation may be needed, though the "toddler" in this case is of course only a manikin. (Photograph courtesy of St John Ambulance Australia (NSW).)*

epinephrine. These studies led to debates over if and when higher doses of epinephrine should be used. While multiple studies showed higher doses of epinephrine improved resuscitability in adult animals, several studies showed that epinephrine effectively increased myocardial and cerebral blood flow in infant animals. In 1990, one study of 20 children in cardiac arrest showed that standard epinephrine doses followed by a higher epinephrine dose improved outcome. Based on this study, the 1992 AHA Guidelines recommended use of high-dose epinephrine (adrenaline) (0.1 mg/kg) in the paediatric patient but limited use of the high dose to the second epinephrine treatment given for asystole. This limitation was placed because of concerns regarding the small size of the study and possible unrecognised deleterious effects of high-dose epinephrine. However, recent large studies of epinephrine dosing in adults show no advantage of high-dose epinephrine over standard-dose epinephrine. The final answer for the paediatric patient is not in yet. Studies have shown that epinephrine is unable to open up coronary vessels distal to coronary lesions. High-dose epinephrine may yet prove useful in the paediatric cardiac arrest victim who has normal responsive coronary vasculature like the studied animals, which showed improved responsiveness, and unlike the adult whose cardiac arrest is due to coronary artery disease. However, these results in adults emphasise the need for restraining optimism.

The ongoing debate demonstrates most clearly the dynamic nature of this field and the need for more studies of paediatric cardiac arrest. The fervour over this issue also reflects the desperate desire to improve the 0–7 per cent survival rates reported for most pre-hospital and the up to 24 per cent survival rates of in-hospital paediatric

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cardiac arrest victims. It also demonstrates the risks of assessing interventions in terms of purely physiologic parameters, such as cardiac output, cardiac perfusion, and return of spontaneous circulation. Outcome studies in cardiac arrest must use quality survival as the critical outcome measure. In addition, a study should be of adequate size to claim scientific validity. Given the low frequency of cardiac arrest in the paediatric population, this factor is especially difficult and challenging for the study of life-threatening paediatric problems.

All endotracheal epinephrine (adrenaline) doses are increased ten-fold to 0.1 mg/kg in order to achieve serum levels obtained with standard-dose epinephrine administered intravascularly. Data from several animal and one human adult study shows that standard epinephrine doses administered endotracheally result in inadequate serum levels. Optimal dosing for the paediatric patient has not been determined.

Adenosine 0.1 mg/kg replaces verapamil as the first-line drug therapy for supraventricular tachycardia. It is highly effective if delivered in a system that does not allow the drug to absorb to intravenous tubing. In addition, it is well-tolerated at all ages. Except for the transient asystole it produces, adenosine appears to have no side-effects or complications. Therefore, its use precludes the possibility of hypotension and cardiac death reported with verapamil use in the young infant.

The use of glucose and glucose-containing solutions should be limited in a resuscitation. Several studies in adults and paediatric patients undergoing resuscitation following head injury, drowning, and shock have demonstrated an association between very elevated serum glucose levels and bad outcome. While the cause and effect of hyperglycaemia in these studies are not clear, an adult animal study showed glucose administration during resuscitation was harmful to the brain. These studies suggest that hyperglycaemia should be avoided. Therefore, glucose use during a resuscitation should be used when certain medications, such as epinephrine or dopamine drips, need to be delivered via glucose-containing solutions, or when hypoglycaemia is documented during a cardiac arrest. The latter is not a rare situation in a young paediatric patient.

SUMMARY

Paediatric CPR is still in its nascency. Many basic anatomy, physiology, and pathophysiology questions need answering in a scientific manner. Fortunately, increasing interest in this area of medicine will eventually unveil these answers. Hopefully, these answers will come rapidly, but will inevitably result in further change to the drills and rules of first aid. This will increase the save rate following paediatric cardiac arrest. Meanwhile, concerted efforts must continue concurrently in the area of prevention, the real life-saving effort. As paediatric CPR comes of age, its effectiveness will the more depend upon the informed and technically-skilled resuscitation.

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Chapter Ten

DEFIBRILLATION

William Griggs

Early defibrillation is the single intervention most likely to result in recovery from cardiac arrest. While early and efficient cardiopulmonary resuscitation (CPR) is essential to maintain tissue oxygenation, it is defibrillation which has the best chance of reversing the arrest situation. Early defibrillation is the third element in the Chain of Survival which includes early access, early CPR, early defibrillation and early Advanced Cardiac Life Support (ACLS).

Not all cardiac arrests are potentially reversible. Every person on the planet will experience a non-reversible cardiac arrest once in their life. There are two main groups of cardiac arrest victims (Table 10.1). The first has a primary cardiac event as the cause of cardiac arrest. These victims are often conscious until shortly after the cardiac arrest occurs. An example might include a dysrhythmia associated with a myocardial infarction. This is the subgroup which is most likely to be salvageable. The second group includes patients whose cardiac arrest is secondary to non-cardiac causes, for example, blood loss from trauma or asphyxia from strangulation. This subgroup's cardiac arrest can usually be effectively treated only if the underlying cause is addressed. With the exception of child victims, the chance of survival in this group is much lower. In the asphyxia group, by the time cardiac arrest occurs, significant cerebral hypoxia may have already occurred. However, it is important not to deny resuscitation on the assumption of cerebral hypoxia in either presumed cardiac or presumed non-cardiac caused cardiac arrests. A patient who has been collapsed for some time may have had a low, but brain-preserving, cardiac output state until just prior to CPR commencing. Certainly patients have made good recoveries after being "arrested" for half an hour or more. When in doubt CPR should always be commenced.

Table 10.1 Cardiac and non-cardiac causes of cardiac arrest

Cardiac	Non-cardiac
*Primary dysrhythmia	Asphyxia/hypoxia
*Dysrhythmia due to acute ischaemia/infarction	Poisoning
	Hypovolaemia
Pump failure due to wall/septal rupture	Flow obstruction, eg pulmonary embolism
Pump failure due to massive infarction	

*Most likely to respond to defibrillation.

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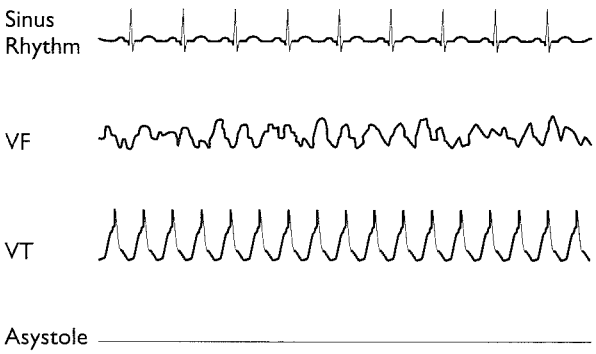


Figure 10.1 *Sinus rhythm, ventricular fibrillation (VF), ventricular tachycardia (VT), asystole.*

Defibrillation and cardioversion

The terms "defibrillation" and "cardioversion" are sometimes used interchangeably. This is not correct. To be strictly accurate, defibrillation is the application of an electrical current with the intention of reversing ventricular fibrillation (VF) (Figure 10.1). Cardioversion is usually reserved for an attempt to revert any other cardiac rhythm such as ventricular tachycardia (VT) (Figure 10.1), atrial fibrillation (AF) or supraventricular tachycardia (SVT).

Defibrillators

A defibrillator is a machine which can provide a direct current discharge across the heart. It usually contains a variable transformer to allow the operator to select varying energy levels. It includes a capacitor to store the charge, a charge switch to activate the charge and a discharge switch (usually two) to complete the circuit from the capacitor to the electrodes. Historically it has been a manually operated device relying on operator interpretation of rhythm. More recently automatic and semi-automatic external defibrillators have become increasingly used (Figure 10.2). These rely on an internal computer-based digital rhythm recognition and a shock advisory system.

Background

Defibrillation is not a new concept. In 1775, Abildgaard, a Danish veterinarian, revived chickens from cardiac arrest using electric shocks. In 1948, the first human was successfully defibrillated. Since that time there have been a number of advances and many studies looking at the effect of defibrillation on survival after cardiac arrest, particularly in the pre-hospital phase. Although it is difficult to compare cardiac arrest survival data, owing to variations in definition, the literature suggests strong support for early defibrillation. An early study in 1980 showed a four-fold increase in cardiac arrest survival with manual defibrillation when compared with CPR alone, followed by transport to hospital. A review of data from 29 United States cities in 1990 again was strongly supportive of early defibrillation. A recent Australian study in 1992 showed statistically significant improved long-term survival following introduction of a pre-hospital defibrillation programme. Another recent United States study of pre-hospital cardiac arrests, published in 1993, showed a significant improvement of survival to discharge, from 2.2 per cent prior to the introduction of defibrillation to 16.4 per cent immediately following its introduction.

ELECTROPHYSIOLOGY

Each cell in the heart has a resting electrical potential across its cell wall. This is called the resting membrane potential and is usually about minus 90 mV. These cells all have the capacity to undergo electrical depolarisation (ie sudden change of polarity back towards and beyond zero into the positive) and subsequent repolarisation (ie change of polarity back to minus 90 mV). This will occur spontaneously when a threshold potential is reached (usually around minus 40 mV). This is called an action potential (Figure 10.3).

The threshold potential can be reached due to depolarisation in adjacent cells. Alternatively, some cardiac cells will have a slow leak of their resting membrane potential until eventually threshold potential is reached and depolarisation is triggered. This is called

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Figure 10.2 *Semi-automatic defibrillators are now standard on-site equipment for use by appropriately trained first aiders in many parts of the world. This photograph (courtesy of Dr Eric Donaldson) shows an example of a semi-automatic defibrillator carried by several of the world's international airlines.*

automaticity and is the ability of a specialised cardiac cell spontaneously to initiate depolarisation. The first area to depolarise then triggers depolarisation in adjacent areas; thus the sino-atrial (S-A) node, which usually has the fastest rate of automaticity, is normally the pacemaker of the heart.

The normal cardiac cycle involves an electrical depolarisation beginning at the S-A node and progressing through the atria to the atrio-ventricular (A-V) node, where a slight delay occurs. Next a wave of depolarisation is conducted through the bundle of His, along the Purkinje fibres and finally to the rest of the ventricles (Figure 10.4). This electrical activity will normally stimulate mechanical activity (ie muscle contraction) and thus cause a heart beat to occur. Repeated activity of this sort is read as sinus rhythm on an electrocardiograph (ECG) (see Figure 10.1).

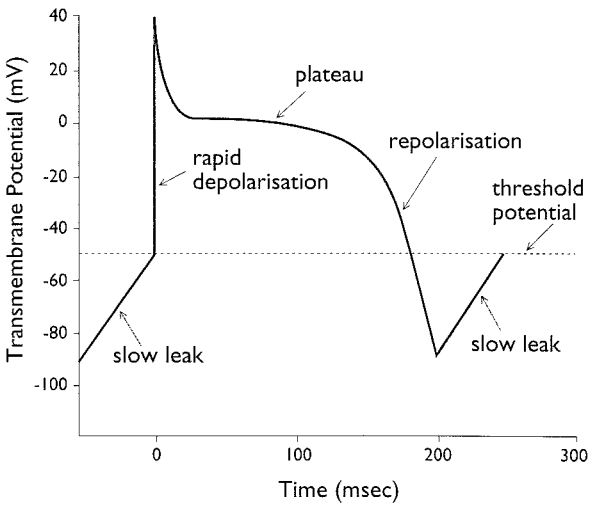


Figure 10.3 *Myocardial action potential.*

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The ECG shows the sum of all the individual cellular depolarisations and repolarisations. The depolarisation of all the atrial cells is shown on the ECG as the P wave. The P–R interval indicates the delay at the A-V node. The QRS complex is caused by ventricular depolarisation and the T wave indicates ventricular repolarisation (Figure 10.5). Repolarisation of the atria is virtually never seen on an ECG trace.

Disorders can occur at any point in this cycle. A normal looking ECG in the absence of palpable pulses indicates uncoupling of the electrical from the mechanical activity of the heart and is called electromechanical dissociation. Unless a remediable cause is found and reversed, the outcome is poor. If the ECG is "normal" but there are no signs of life, it is essential to commence and continue CPR until an appropriate medical decision can be taken to abandon resuscitation.

Other disorders relate to variations in the wave of depolarisation and its travel through the heart. Although the S-A node is usually the pacemaker of the heart, in some cases other parts of the heart can take over this role. This can be due to disease or drugs, or can even occur in some "normal" patients such as victims of simple vaso-vagal syncope or faint. In particular, ischaemic cells can become disordered in their behaviour and can spontaneously depolarise and thus trigger an unusual cycle.

Once a cell has been depolarised it cannot be depolarised again until it has repolarised. This period of time is called the refractory period. Thus if an abnormal depolarisation occurs when some of the heart has repolarised and some is still refractory (ie during the T wave), the current may be blocked from travelling in one direction by an area of myocardium that is refractory. However, it may be possible for the current to circle the heart from the original location, arriving when the area is no longer refractory. Thus the current can return to its starting point and begin the process again. This means the electrical activity can continue in this circle indefinitely. This is known as a "circus" movement (Figure 10.6). A circus can pass the same point at variable speeds up to three or four hundred times a

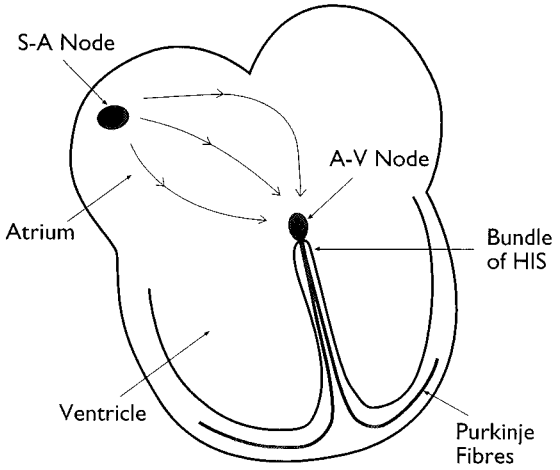


Figure 10.4 *Normal conducting pathways.*

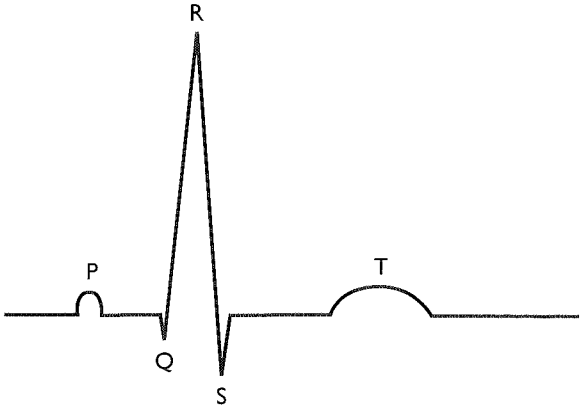


Figure 10.5 *Single sinus beat showing P, Q, R, S and T waves.*

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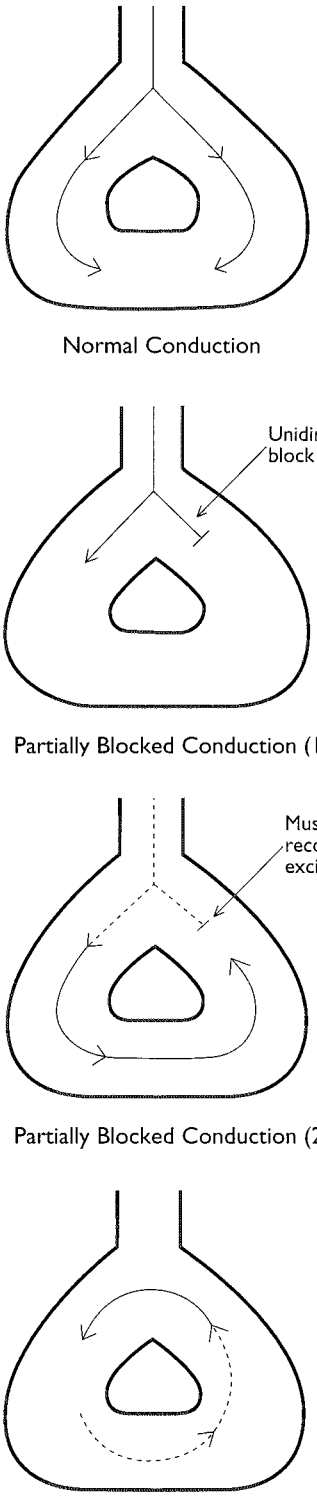


Figure 10.6 *Diagram group showing unidirectional block and circus sequence.*

minute. There are other abnormal mechanisms which may also produce the same potentially damaging result.

An accelerated rate of electrical activity can be ventricular or supraventricular in origin. In both cases with moderately fast rates (150–200) (VT or SVT) it is possible that a diseased or poorly compliant heart may not be able to respond fast enough mechanically to an electrical drive of this rate. In some cases, there may not be enough time for cardiac filling to occur during diastole. In this case, effective circulatory arrest occurs. However, another patient with exactly the same rate and rhythm may be conscious and alert. It is thus essential always to treat the patient rather than just the rhythm. Pulseless VT should be treated in the same way as VF.

When the rate exceeds 300 times a minute even a normal heart is mechanically unable to respond and so the heart fibrillates, which is to say that it vibrates ineffectively as it tries to contract and expand more than five times a second.

If fibrillation occurs in the atria (AF), the delay of impulse conduction through the A-V node will usually result in only some of the impulses getting through to the ventricles. The ventricular response rate can vary from very fast (greater than 150) to very slow (less than 60). However, the ventricles usually beat relatively normally, the excess driving impulses not being able to get through the barrier of the A-V node; people can survive with atrial fibrillation for many years. Drugs may help control the ventricular rate.

If the fibrillation occurs within the ventricles (VF), there will be no effective cardiac output and the patient goes into a state of cardiac arrest. CPR can maintain life for a short time and, indeed, is necessary whilst a defibrillator is brought to the victim, or the victim is taken to the defibrillator. Without electrical reversion, however, the patient cannot survive.

While spontaneous reversion from VF during CPR has occurred, the best chance for the patient is immediate defibrillation. One of the main reasons for the establishment of coronary care units was to provide immediate defibrillation in the event of a VF cardiac arrest.

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THE AIM OF DEFIBRILLATION

The aim of defibrillation is, by using electricity, to depolarise the entire myocardium at one time. Following repolarisation it is hoped that the next cycle will be a relatively normal one, and that normal activity will resume. It is important to deliver the appropriate amount of energy to the heart, as too little may not be enough to depolarise the heart, and too much may cause myocardial damage. Recent studies suggest that a current of 30–40 amperes is optimal for external defibrillation and that current-based defibrillation may be superior to energy-based defibrillation. However, there are, as yet, no commercially available current-based defibrillators. Further study is needed in this area. The relationships between energy, current, voltage, power and time are shown in Table 10.2.

TRANSTHORACIC IMPEDANCE

Transthoracic impedance is the resistance to the passage of electrical current through the chest. Average transthoracic impedance is from 70 to 80 ohms, but may range from 15 to 150 ohms. If transthoracic impedance is high, a low energy shock may fail to generate enough current to completely depolarise the heart. Factors which can affect transthoracic impedance are listed in Table 10.3. To reduce transthoracic impedance as much as possible the operator should apply firm pressure (approx 12 kg) to correctly placed paddles utilising an appropriate conducting gel or pads and should deliver shocks in rapid sequence. A single shock can reduce transthoracic impedance by approximately 8 per cent for subsequent shocks. Defibrillation with bare paddles and no coupling material will result in very high transthoracic impedance, causing skin burns and failure to deliver appropriate current to the heart. From Table 10.2 it can be seen that a 35 ampere defibrillation across a 75 ohm impedance will require a voltage of

$$75 \times 35 = 2625 \text{ volts,}$$

and with a 200 joule setting, this would be delivered in a time of

$$\frac{200}{(35 \times 2625)} = 0.0022 \text{ seconds (2.2 milliseconds).}$$

Table 10.2 Relationships between energy, current, voltage, power and time

Voltage (volts)	=	current (amps) x impedance (ohms)
(*E*	=	*IR*)
Power (watts)	=	energy (joules)/time (seconds)
Power (watts)	=	voltage (volts) x current (amps)
Time (seconds)	=	energy (joules)/voltage (volts) x current (amps)

Table 10.3 Determinants of transthoracic impedance

Energy selected
Electrode size
Paddle–skin coupling material
Location of paddles
Number of previous shocks
Time interval between previous shocks
Phase of ventilation
Size of chest
Paddle electrode pressure

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ELECTRODE SIZE AND PLACEMENT

It has been shown that transthoracic impedance decreases with increasing paddle size. Large self-adhesive coupling pads have also been associated with lower transthoracic impedance. The optimum diameter for adults is considered to be between 8 and 13 centimetres.

There are two main options for paddle placement. The most commonly used position involves one electrode being placed to the right of the upper sternum just below the right clavicle and the other just lateral to the cardiac apex with the centre of the paddle in the midaxillary line. There are two main variants of the much less commonly used anteroposterior position. The first involves leaving the apical paddle where it is and moving the other paddle to the right infrascapular region. The second involves the anterior paddle being placed over the left precordium with the posterior paddle being over the left infrascapular region. The relative value of these last two positionings, which are much more difficult to achieve than the anterior paddle positions, is unclear. Theoretically, all three positions optimise the current path through the heart.

It is important not to place the electrodes too close together or to allow the gel pads to touch or liquid gel to become smeared between the two paddles. This is because the current will follow the path of least resistance across the chest wall, minimising the amount passing through the heart.

Patients with permanent pacemakers can be safely defibrillated provided the paddles are not placed over or near the pacing generator box. Commonly these are now placed in the abdomen, so this is not a common problem. If the generator box has been placed inferior to the right clavicle, it should be easily palpable.

ENERGY REQUIREMENTS FOR DEFIBRILLATION

Current recommendations for adult defibrillation are based on energy delivered. The American Heart Association (AHA), the Australian Resuscitation Council (ARC) and the European Resuscitation Council (ERC) all have similar guidelines for the first three shocks (Table 10.4). The maximum setting referred to by the ARC usually means a setting between 300 and 400 joules depending on the model of defibrillator. Both the AHA and the ERC suggest that shocks should be delivered in clusters of three to take advantage of decreases in thoracic impedance with repeated shocks.

ASYSTOLE

There is no evidence that there is any benefit in delivering electrical shocks to a heart in asystole (see Figure 10.1). In practice, asystole is encountered particularly in childhood victims of drowning and suffocation accidents. Logically there would seem no reason to

Table 10.4 Recommendations for energy settings for transthoracic defibrillation

	AHA	ARC	ERC
First shock	200 joules	200 joules	200 joules
Second shock	200–300 joules	200 joules	200 joules
Third shock	360 joules	Max. setting	360 joules
Subsequent shocks	360 joules	Max. setting	360 joules

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expect benefit in cases of true asystole. In some patients, however, coarse VF may be visible in some leads but only minor swings visible in others. This is because the true axis of electrical activity occurs in three dimensions, but each lead only records in two dimensions; therefore, if the recording lead is perpendicular to the true axis, minimal deflections may occur. This also accounts for the variation seen in QRS size in different leads in a normal ECG. Therefore, if only one lead is selected, it is possible that the minor visible swings may mimic asystole. This explains the apparent success in defibrillating "asystole". Ideally two leads should be checked as well as the ECG calibration before a confident diagnosis of asystole is made.

Children

In children non-cardiac causes of cardiac arrest predominate and VF is, therefore, uncommon. Children nearly always arrest in asystole. Thus it is unusual for a child to require defibrillaton. The main exceptions to this are children with congenital cardiac disease and children who are the victims of electrical accidents. If VF is present the energy used is usually calculated at 2 joules per kilogram.

BLIND DEFIBRILLATION

Blind defibrillation is the administration of a shock to a cardiac arrest victim without first ascertaining the cardiac rhythm. This was done in the past when the defibrillator involved did not have the ability to display an ECG. Virtually all modern defibrillators now have the ability to display an ECG strip, and the paddles are often marked "APEX" and "STERNUM" to enable them to be the correct polarity to record lead II when using the "paddles" monitor mode. Blind defibrillation is not commonly used today, and must never be used other than by paramedics and doctors trained in its use.

SAFETY AND CARE OF DEFIBRILLATORS

The risk of accidental shock from defibrillators has been reported as between 1 in 1000 and 1 in 1700. The responsibility lies with the operator to ensure that all personnel are clear of the patient before discharging the paddles. Obviously the presence of liquids, and in particular flammable liquids, may be a contra-indication to the performance of defibrillation. Glyceryl trinitrate (GTN) transdermal adhesive patches and ointment should be removed from the chest as theoretically, small explosions can occur (Table 10.5). Daily equipment checks are a vital part of safe practice and it has been recommended that checklists be used with every shift change. These daily checks are separate from the regular service provided by biomedical engineering companies.

Defibrillation has been safely performed in aircraft, both rotary and fixed wing, without interference with avionics equipment. However, it would be wise to test individual equipment electively before putting it into service. In some countries this is a legal requirement.

Currently, defibrillation is not recommended under hyperbaric conditions.

Table 10.5 Care in the use of defibrillators

Do NOT defibrillate over ECG electrodes or GTN patches.
Do NOT defibrillate over an implanted device (eg pacemaker box).
Do NOT have any direct or indirect contact with the patient during defibrillation.
Do NOT charge or discharge the paddles in the air.
Do NOT have the patient in contact with metal fixtures.

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SYNCHRONISED CARDIOVERSION

When a patient is in AF or SVT with a reasonable cardiac output, it may be appropriate to electively cardiovert him in an attempt to regain sinus rhythm. This is not relevant to the first aid or pre-hospital context, but is included here for completeness and interest. It should be clear that it would be unwise to allow the electrical current to pass through the heart when it is partly repolarised and still partly depolarised – that is during the T wave – as this could cause a circus to occur and push the patient into VF. Accordingly, most defibrillators have the capability to provide a synchronised cardioversion. The defibrillator detects the R wave and delivers the shock to avoid the T wave. Thus, after pushing the discharge buttons, there may be a delay of up to a few seconds, while awaiting definite R wave identification, before electrical discharge occurs. Naturally, a conscious patient would require an anaesthetic for this procedure. If the dial setting "synchronisation" is accidentally selected during attempted defibrillation of VF there will be no R wave and the machine may not discharge.

IMPLANTABLE INTERNAL DEFIBRILLATORS

Some patients at high risk of recurrent ventricular dysrhythmias may have a permanently implanted internal defibrillator. These are usually patients who are refractory to drug therapy. These machines have the ability to detect and recognise VF or rapid VT. There is a 5–20 second period of rhythm identification, followed by a 5–15 second period of charging. Following this the unit will discharge on a number of occasions – usually three or four – at between 20 and 40 joules. Obviously, a lower energy is required as the current is delivered directly to the heart itself. It is possible for the unit to discharge while a rescuer is performing CPR. This may be felt by the rescuer, but no injuries to the rescuer have been reported and the theoretical risk is minimal because such small energy is involved.

SEMI-AUTOMATIC EXTERNAL DEFIBRILLATORS

One of the most impressive recent advances is that of the semi-automatic external defibrillator (SAED) (see Figure 10.2). These are only semi-automatic, as some user input is required. This device was initially introduced during the late 1970s but poor performance precluded their widespread use. Recent advances in technology have improved the sensitivity and specificity, and a number of studies have shown them to be safe and effective in the hands of first aiders with minimal training. Shock Advisory Systems have been developed which guide the operator through a number of simple steps.

Adhesive ECG defibrillator pads are attached to the patient in the usual paddle positions. The SAED diagnoses the ECG and then advises the operator whether to shock the patient or not, by displaying messages on a screen, and by voice prompts. The final decision to shock is then taken by the operator. To deliver a shock the operator must press a shock key. This key is only active when a shockable rhythm has been diagnosed. The SAED will then warn everyone to stand clear and finally deliver the shock. Subsequently the sequence is repeated according to a predetermined protocol. These machines have been shown to be very reliable, with 99+ per cent reported accuracy for not advising a shock for non-shockable rhythms (specificity), and 90+ per cent accuracy for advising a shock for shockable rhythms (sensitivity).

Who should use a defibrillator?

The American College of Emergency Physicians suggests that SAED providers should include any first-responder or any level of emergency medical technician. The AHA states that all personnel whose job requires that they perform basic CPR be trained to operate and permitted to use defibrillators, particularly SAEDs. A typical SAED course should last 8–10 hours and be followed six monthly by refresher courses. Recent studies have shown that airline cabin crew, firemen and police can all be

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successfully trained in the care and use of the SAED, and importantly that the time to defibrillation can be significantly lowered. Some highly trained emergency medical technicians may be more efficient using manual defibrillators to increase the accuracy of shocking a shockable rhythm.

Early defibrillation remains the cornerstone of successful treatment of cardiac arrest due to cardiac causes. As the availability of SAEDs increases amongst first responders, along with the increased use of early CPR and the other links in the Chain of Survival, the cardiac arrest victim's chance of survival will be greatly improved.

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CHAPTER ELEVEN

DEVICES FOR EXPIRED AIR RESUSCITATION

G.A. Harrison

The optimal techniques of respiratory management during cardiopulmonary resuscitation (CPR) are intubation of the trachea with a cuffed endotracheal tube and ventilation of the lungs with a resuscitator. The combination enables the rescuer to sustain prolonged periods of ventilation without fatigue whilst the upper airway should remain unobstructed in the absence of technical problems with the tube. The lower airway is protected from regurgitated stomach contents by the inflated cuff and access is provided to suction of secretions or blood etc. However, the equipment required for the two techniques is not commonly available at the moment that a respiratory or cardiac arrest occurs. Both techniques require the development and maintenance of considerable expertise which only comes with frequent practice. In the 1960s and 1970s, these techniques were classified as belonging to the domain of advanced life support and were generally used only in hospitals. Members of the general community were encouraged to learn basic life support with mouth-to-mouth or mouth-to-nose expired air resuscitation (EAR), as the means of ventilation of the victim's lungs.

In the 1980s and 1990s, this simple division of resuscitation skills into basic or advanced life support has been broken down by two developments – the fear of cross-infection with the human immunodeficiency virus (HIV) during CPR, and the desire of highly motivated health professionals to bring the advantages of advanced life support to victims of respiratory or cardiac arrest outside the hospital. Unfortunately, there have developed suppositions in many members of the community, including some health professionals, that technology in CPR equipment in the form of "protective" devices for expired air resuscitation not only prevents cross-infection with HIV but also enhances the ease of airway management and ventilation of the lungs. The former supposition is doubtful and the latter supposition is almost certainly incorrect in the case of the untrained or poorly trained users. Fear, altruism and commercial interest have created a potent mixture of emotions which has driven the development of a large number of ancillary devices for expired air resuscitation (Figure 11.1); some are potentially useful in the hands of trained rescuers, while others are potentially useless in anybody's hands.

Resuscitation should never be delayed until an ancillary device is found and time should not be wasted by an unskilled rescuer's attempts to use a device of a type for which the individual has received no training.

RISK OF CROSS-INFECTION

Very few cases of cross-infection during CPR have been reported. The organisms were *Myobacterium tuberculosis*, meningococcus, herpes simplex virus, shigella and salmonella. At the time this chapter was written, there had been no reported cases of cross-infection with HIV or hepatitis B virus following EAR or its teaching. Despite numerous reiterations of this statement in scientific literature, there is ample evidence that some health-care workers are reluctant to perform EAR on other than a close relative. It is likely that a similar attitude is growing in the general community! This

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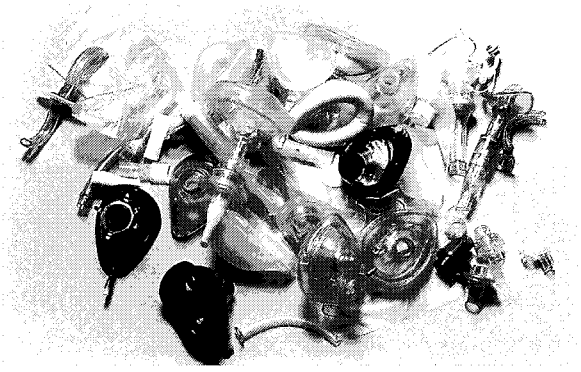


Figure 11.1 *A selection of devices for expired air resuscitation. (Photographs in this chapter courtesy G.A. Harrison.)*

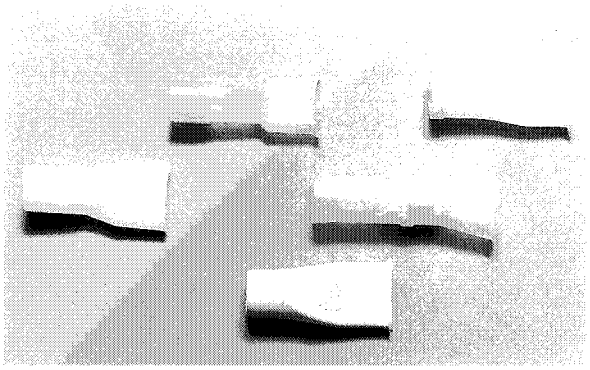


Figure 11.2 *A selection of non-integral mouthpieces for attachment to a face mask. The mouthpiece on the right in the middle row has a valve in it; the others do not contain a valve or filter.*

attitude is likely to have been spread by articles or public statements which encourage the use of ancillary devices for EAR even whilst stating the lack of evidence of cross-infection with HIV. Given the high incidence of vomitus, saliva or blood around the mouth of victims who require EAR, it is not surprising that there are also aesthetic reasons for individuals to prefer EAR with an ancillary device to EAR with mouth-to-mouth or mouth-to-nose contact. Indeed, it seems appropriate that the use of an ancillary device be taught to all individuals who have mastered the skills of basic life support. Such acknowledgement of the availability of ancillary devices for EAR might avoid discouraging the use of mouth-to-mouth ventilation whilst encouraging a more rational and, hopefully, skilled use of the devices rather than the ineffectual fumbling which is likely to be the consequence of a user who has never practised with, or even seen, the device previously.

TYPES OF ANCILLARY DEVICES FOR EXPIRED AIR RESUSCITATION

Approximately 50 individual devices for EAR have been mentioned in the health professional literature in English. There are certainly more. The devices can be classified into the following basic types which may be regarded as the "aesthetic" portion.

1 Devices to be applied to the face without insertion into the airway:
   - **a** Facemasks
   - **b** Faceshields

   There may be an additional integral or detachable mouthpiece (Figure 11.2)
2 Devices to be inserted into the airway:
   - **a** Oropharyngeal airways
   - **b** Nasopharyngeal airways
   - **c** Combitubes and pharyngeal–tracheal airways
   - **d** Oesophageal obdurator airways.
   - **e** Laryngeal masks

To the basic component is often added an integral or detachable part which is the "protective" portion (Figure 11.3). This may be a unidirectional or bidirectional valve or a filter which can prevent the passage of liquids, bacteria or viruses. In the bidirectional valve there is an expiratory port which is intended to direct the expired breath of the victim away from the rescuer. Some filters have the serious disadvantage of a high resistance to airflow which may increase further as the filter becomes moist with the exhaled water vapour of rescuer and victim. This produces potential difficulty of inflation of the victim's lungs by the rescuer and potential expiratory obstruction for the victim. The protective portion and mouthpiece form a suprastructure. Given the exceedingly small incidence of cross-infection during EAR and

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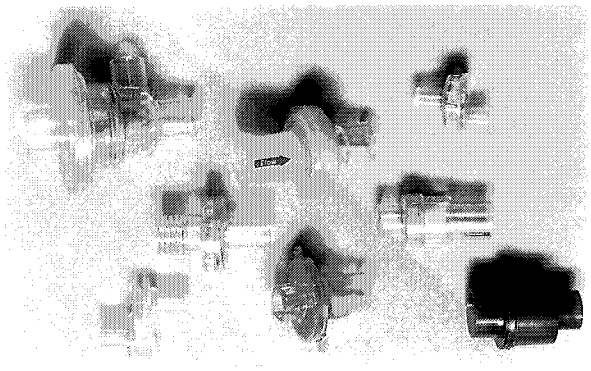


Figure 11.3 *A selection of non-integral bidirectional valves which are intended to be attached to a facemask.*

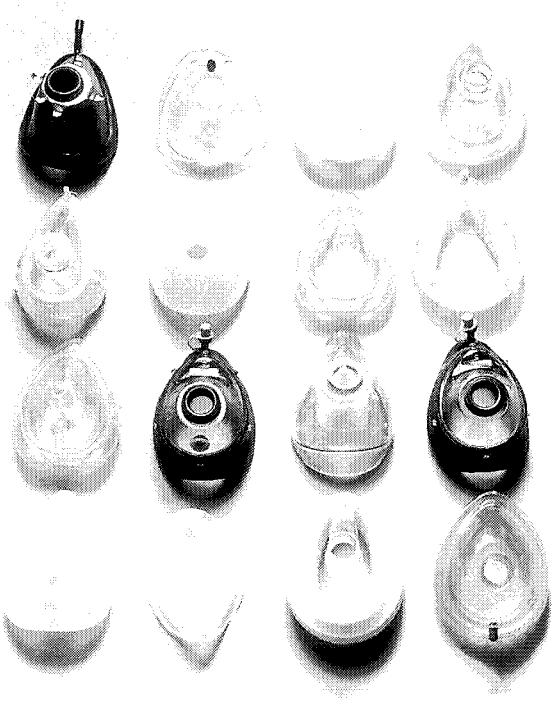


Figure 11.4 *A selection of facemasks. At the top left corner is a representative mask for anaesthesia. The others are suitable for expired air resuscitation. Each facemask has either a 22 mm female or 15 mm male connector for attachment of a suprastructure if the rescuer chooses to do so.*

the lack of evidence of transmission of HIV during EAR, it cannot be accepted that the protective section is an essential part. However, the suprastructure does lessen the possibility of regurgitated material reaching the rescuer's mouth.

Facemasks

The potential efficacy of mouth-to-mask ventilation was established in 1954 by Elam, Brown and Elder. They described the advantages over mouth-to-mouth resuscitation as

1 both hands of the rescuer are available for support of the jaw;
2 the user retains a "feel" for leaks or airway obstruction;
3 mouth-to-mask ventilation overcomes a bystander's reluctance "to come into direct contact with the lips of another person";

and these remarks are still pertinent.

A number of studies on manikins have shown that health professionals produce ventilation of the same order with mouth-to-mask ventilation as they do with mouth-to-mouth ventilation, and the use of either of these techniques produces greater minute ventilation than bag-valve-mask ventilation. Mouth-to-mask ventilation is not difficult to learn once the skills of mouth-to-mouth ventilation have been mastered but without this prior experience the operator's performance is poor.

There are two techniques of use. In one (Figure 11.5), the operator is positioned at the top of the head facing the victim's feet. The mask is held onto the face with two hands with two or three fingers lifting the mandible at the angles with head-tilt and jaw-thrust. EAR is administered through the central connector of suprastructure. In the other technique (Figure 11.6), the operator is positioned at the side of the victim. One hand maintains head-tilt and secures a seal around the nose and upper face, the other hand seals the mask around the chin and lower face and provides jaw support. The first technique generally produces a better seal of mask to face but is more tiring on the fingers. It is less like mouth-to-mouth ventilation with respect to

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Figure 11.5 *The technique of mouth-to-mask expired air resuscitation using jaw-thrust with the operator positioned at the top of the head of the casualty. In this case, the suprastructure contains a bidirectional valve and mouthpiece.*



Figure 11.6 *The technique of mouth-to-mask expired air resuscitation using jaw support from the side of the casualty. In this case, the integral connector in the mask serves the function of a mouthpiece.*

hand position than the second technique and as a consequence may be more difficult to learn. The first technique is less convenient when external chest compression is also required in one person cardiopulmonary resuscitation. There is presently no evidence that one technique is better than the other. The comparative efficacy of each technique may depend on the size of the operator's hands and fingers and the shape of the victim's face.

There is presently no strong evidence that one shape of facemask is better than another. There is also no evidence that masks with air-filled or cushion rims are better than masks without rims. Masks made of very smooth, slippery plastic surfaces may present problems with the achievement of a seal.

Airway management during mouth-to-mask ventilation is greatly enhanced by insertion of an oropharyngeal Guedel-type airway. However, this manoeuvre should be undertaken only by those who have received training in the use of an oropharyngeal airway (see later in this chapter).

Faceshields

These are flexible sheets which cover the victim's face (Figure 11.7). They have a central portion which has a filter or valve. As the potential advantage of the

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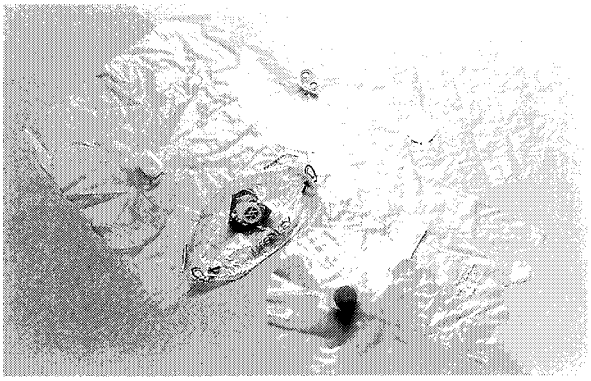


Figure 11.7 *A selection of faceshields. The central portion of each contains a unidirectional valve or filter.*



Figure 11.8 *Technique of mouth-to-faceshield expired air resuscitation. Compare with Figure 11.6. More fingers must exert pressure through the faceshield onto the face and chin to achieve an airtight seal during inflation. The operator's own fingers may hamper access to the central portion of the faceshield.*

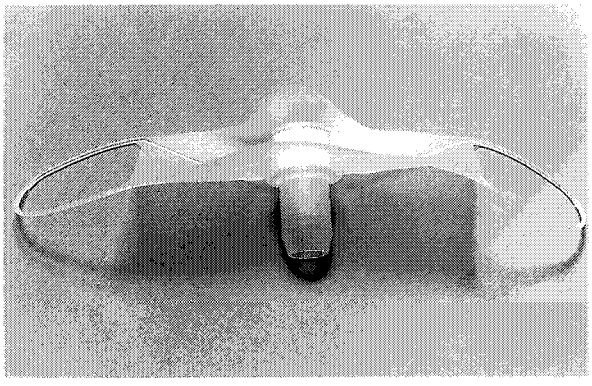


Figure 11.9 *An example of a faceshield with an intra-oral portion (mouth prop). The central portion contains a unidirectional valve which cannot be seen from this view.*

faceshield is its suitability for folding to a very small size for carriage in a pocket or on a key ring, the valve is usually a flat, unidirectional valve.

The operator is positioned at the side of the victim. One hand maintains head-tilt and seals the faceshield over the nose and upper face (Figure 11.8). If possible, this hand also compresses the nostrils. The other hand provides jaw support and seals the faceshield onto the chin and lower face. The operator blows into the central portion. In the case of most faceshields, the operator must be careful to reduce the pressure on the faceshield during expiration.

Some of the central filters have a high resistance. As a consequence of the unidirectional valve or high-resistance filter, most if not all of the victim's expired gases must pass between the faceshield and the victim's face. Some faceshields have an intra-oral portion (mouth prop) (Figure 11.9). Any intra-oral portion introduces an increased risk of expiratory obstruction and it should not be wider than 2 cm in devices designed for use in adults.

There are very few studies of the efficacy of faceshields. There are anecdotal reports of difficulty in effecting a seal of faceshield to face during inflation, especially when the victim's face is wet with perspiration, water or regurgitated stomach contents.

Oropharyngeal airways

Most of the modern oropharyngeal airways (Figure 11.10) are based on the Safar double Guedel airway (Figure 11.11). Modern oropharyngeal airways for EAR have a valve or filter in the suprastructure and a flange which is intended to produce a seal around the mouth. In the hands of an operator who has mastered the insertion technique, oropharyngeal airways are an effective method of airway management. Unfortunately, there is considerable danger that an untrained or poorly trained user will place the airway beneath the tongue, pushing it into the pharynx, or produce obstruction at the level of the epiglottis. To reduce the risk, some manufacturers have produced devices with very short intra-oral portions which, as a result, have the potential to be both ineffective and dangerous in unskilled hands.

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Although oropharyngeal airways can be used with the rescuer positioned at the side of the victim, seal of the flange on the lower face is better achieved if the operator is at the top of the victim's head. Jaw-thrust is used to enhance airway management (Figure 11.12). It is not uncommon for there to be difficulty in sealing the flange around the mouth and it is sometimes not easy to close the nostrils although some airways are provided with a nose clip.

If expiratory resistance for the victim is high in the valve or filter in the suprastructure, the expired gases must pass out between the intra-oral portion and the lips and then between the mouth flange and the face. The risk of expiratory obstruction is considerable.

No formal clinical trials appear to have been undertaken to compare individual makes of airway. There appear to have been no comparative trials between airways and facemasks. Unfortunately, the characteristics of manikin faces and upper airways make the testing, teaching and learning of airways difficult.

Some airways can be used very effectively by health professionals who have been trained on anaesthetised patients but it is doubtful if they have any advantages over a facemask and Guedel-type airway.

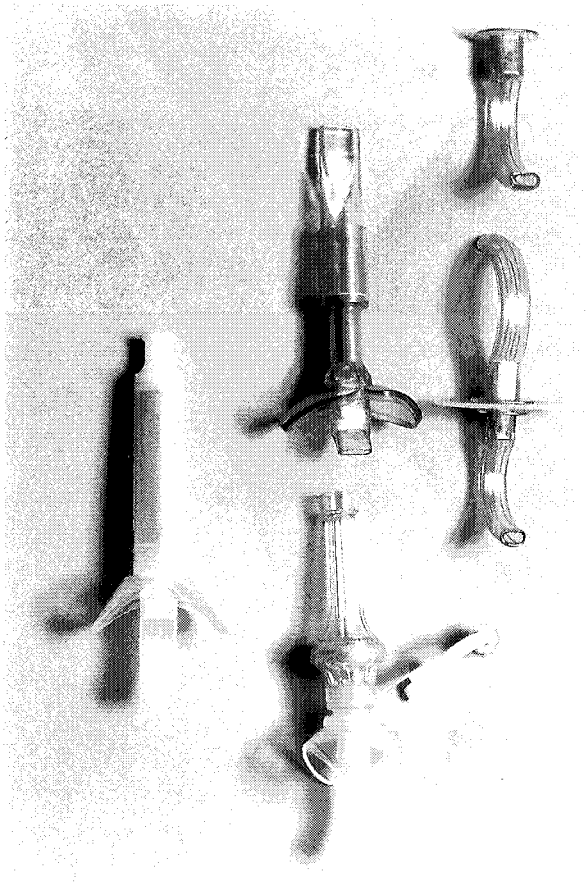


Figure 11.10 *A selection of oropharyngeal airways. The device at the top of the middle vertical row could be classified as a mouth prop. The device on the left has a filter in the supra-structure, and the two in the middle have bidirectional valves in the suprastructure. The devices on the right are a Guedel-type airway (upper) and double Guedel-type airway (lower).*



Figure 11.11 *Technique of mouth-to-airway expired air resuscitation using a double Guedel-type airway.*

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Nasopharyngeal airways

Although potentially valuable in the management of victims of trauma of the face, nasopharyngeal airways (Figure 11.13) have a limited role in CPR because of the risk of nasal trauma and bleeding into the airway.

Oesophageal obdurator airway, Combitube and the pharyngeal-tracheal airway

The skills of endotracheal intubation require intensive training and retraining. Various devices have been designed to facilitate placement of a tube in the pharynx or to facilitate the recognition of fortuitous blind endotracheal intubation. The oesophageal airway has caused concern because of the serious nature of the complications. The Combitube and pharyngeal–tracheal airway have been reviewed recently. Their place in CPR remains to be established.

Laryngeal mask airway

Initially introduced as an alternative to endotracheal intubation during anaesthesia, the laryngeal mask airway (Figure 11.14) has aroused interest as an aid to airway management during resuscitation. It appears easier to place than an endotracheal tube although placement is more difficult than the insertion of a Guedel airway.

The potential for the laryngeal mask during CPR has still to be determined, but early reports are promising.



Figure 11.12 *Technique of mouth-to-airway expired air resuscitation using jaw-thrust. A nose clip closes the nostrils.*

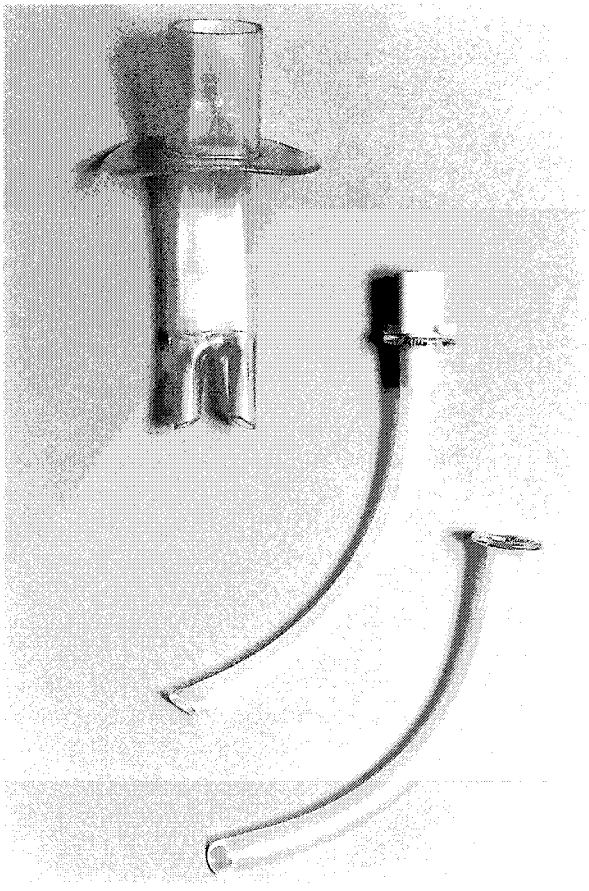


Figure 11.13 *Examples of nasopharyngeal airways.*

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ADMINISTRATION OF OXYGEN

Expired air resuscitation can provide a tolerable partial pressure of oxygen in arterial blood (PaO_2) but the PaO_2 is dependent on the alveolar–arterial oxygen difference which may be considerably widened in victims of respiratory or cardiac arrest. It is highly desirable to increase the concentration of oxygen in the gases which enter the victim's lungs during EAR. The administration of supplemental oxygen is most easily achieved in the case of facemasks as the catheter delivering the oxygen can be placed under the mask. Some masks have a nipple or grummet to direct oxygen into the mask. It is possible to achieve approximately 50 per cent delivered oxygen concentration at supplemental oxygen flows of 8–10 litres per minute. Addition of extra tubing between the mask and operator (Figure 11.15) provides a reservoir which increases the delivered oxygen concentration but adds dead space unless a bidirectional valve is included in the suprastructure close to the mask. For some reason, it has not been usual to include a nipple or grummet in the suprastructures of oropharyngeal airways but there seems no reason why this should not be done. There would be only minor enhancement of delivered oxygen by adding oxygen under a faceshield. The potential reservoir is very low because of the close fit of the faceshield to the victim's face and the presence of the oxygen tubing and oxygen flow might, theoretically, increase the difficulty of achieving a seal on the face.

BUYER BEWARE

Whilst some devices are potentially useful other devices are difficult to use, and others have potential hazards. There is now an Australian Standard 4259-1995 for Ancillary Devices for Expired Air Resuscitation. Some of the more important features are as follows.

- Resistance to airflow across a wet filter or valve of 0.5 kPa (5 cm H_2O) at 50 L/min unless special warnings are provided by the manufacturer in the case of faceshields.
- Maximum diameter of mouth prop of 2.0 cm for adults (1.5 cm for children) and maximum length of 2.5 cm.
- 22 mm female or 15 mm male connectors on devices without integral mouthpieces or suprastructures.
- Minimum ventilation of 9 litres in a test circuit (3 litres for devices intended for children).
- Easy clearance of water or vomitus.
- Resistance to a wide range of temperatures and humidities without loss of performance.
- Substantiation of claims that the device prevents the passage of fluids or microorganisms across it.

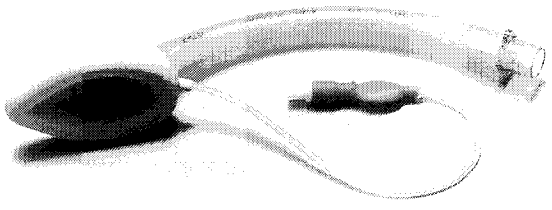


Figure 11.14 *A laryngeal mask airway.*

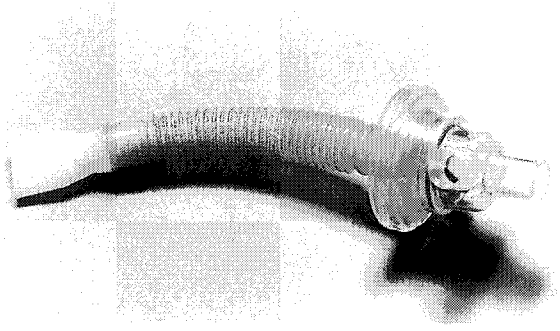


Figure 11.15 *An example of extra tubing between a mouthpiece and a bidirectional valve.*

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Many devices for expired air resuscitation have been evaluated, and the reader should consult studies of these evaluations.

CONCLUSIONS

Individuals over 10 years of age should be able to master the skills of mouth-to-mask ventilation after the attainment of mastery of mouth-to-mouth ventilation. Mastery of the use of a faceshield on manikins should be undertaken by individuals who intend to carry one. The mastery of the use of oropharyngeal airways should only be undertaken by health professionals who can practise on anaesthetised subjects until manikins which permit more realistic practise of airway insertion are developed. All health professionals should be able to perform mouth-to-mask ventilation with or without supplemental oxygen.

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CHAPTER TWELVE

ASSISTED VENTILATION

G.A. Harrison

The re-establishment of an effective minute volume is essential for a victim who has suffered a respiratory or cardiac arrest. During circulatory arrest and cardiopulmonary resuscitation (CPR), carbon dioxide (CO_2) accumulation occurs on the venous side of the circulation due to the reduced pulmonary blood flow. It is essential to reduce the partial pressure of CO_2 in the arterial blood ($PaCO_2$) to minimise the rise in venous PCO_2 and the potential or established intracellular acidosis. The partial pressure of oxygen in arterial blood (PaO_2) depends not only on an adequate minute volume but also on the delivered oxygen concentration, the mixed venous PO_2 and the alveolar–arterial oxygen difference. Of these variables only ventilation and delivered oxygen concentration can be readily manipulated by the rescuer. The rescuer should achieve clearly visible normal expansion of the chest at a rate not less than 12 per minute and the delivered oxygen should be maximised as soon as feasible, preferably to 80–100 per cent concentration.

A skilled rescuer can achieve these objectives by the use of a resuscitator. The International Standards Organisation's definition of a resuscitator is a device used in emergency situations to provide lung ventilation to individuals whose breathing is inadequate. Whilst effective expired air resuscitation (EAR), with or without an ancillary device, can provide high minute volumes to the victim, there are some problems.

- It is tiring for the rescuer if prolonged ventilation is required.
- Some rescuers may find the position for EAR (sometimes crouching, kneeling or lying fully prone) very uncomfortable.
- If there is a need to compensate for leaks between the rescuer's mouth or an ancillary device and the patient's mouth or face, the minute volume which must be produced by the rescuer may be very high, leading to the rapid onset of apnoea or a sense of respiratory distress in the rescuer.
- The need to swap roles in two person techniques of CPR can arouse aesthetic concerns or even produce fear of potential cross-infection from the other rescuer.
- The delivered oxygen is only approximately 16 per cent during EAR which may not be sufficient to achieve an appropriate PaO_2 in some victims.

A resuscitator has the potential to overcome these problems and is particularly useful in prolonged resuscitation.

TYPES OF RESUSCITATORS

Resuscitators can be classified according to the power source which provides inflation of the lungs.

1 Operator-powered resuscitators
- **1.1** Not dependent on an oxygen supply
- **1.2** Dependent on an oxygen supply
  - **a** Partial rebreathing systems
  - **b** Circle system

2 Gas-powered resuscitators
- **2.1** Manually cycled
- **2.2** Automatic pressure cycled
- **2.3** Automatic time or volume cycled

3 Miscellaneous

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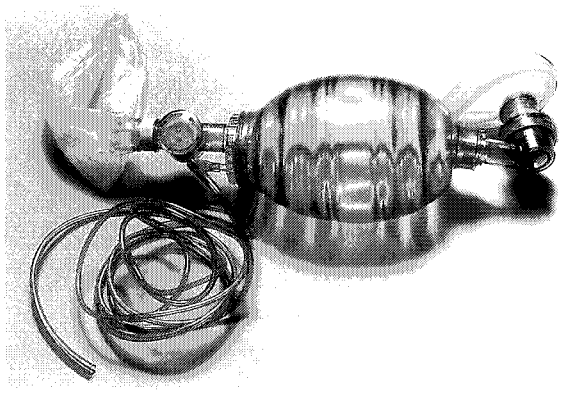


Figure 12.1 *An example of an operator-powered resuscitator not dependent on an oxygen supply. There is a central compressible bag attached to a bidirectional patient valve on the left. Oxygen may be added through the tubing seen at the left hand end. A non-integral reservoir for oxygen with low pressure positive and negative pressure valves can also be seen at the left hand end. (Photographs in this chapter, unless otherwise attributed, are courtesy of Professor G.A. Harrison.)*



Figure 12.2 *Technique of use of an operator-powered resuscitator not dependent on an oxygen supply. With one hand the operator produces head-tilt and jaw support and effects a seal of the mask to the face. The other hand squeezes the compressible bag.*

The potential ease of use, effectiveness and complications of each class depend on whether or not the patient is intubated with an endotracheal tube with the cuff inflated. In the non-intubated patient, the potential problems are

- failure to ventilate the lungs because of airway obstruction or leaks between the facemask and the face;
- distension of the stomach because of high pharyngeal pressures; and
- delay in the achievement of ventilation because of technical problems with the resuscitator.

In the intubated patient, the potential problems are

- barotrauma, especially pneumothorax; and
- airtrapping with circulatory embarrassment due to poor design or malfunction of the patient valve in the resuscitator.

It is important that the victim be able to breath spontaneously from the resuscitator during recovery from the respiratory or cardiac arrest. In that event, the potential complications are

- excess subatmospheric pressure in the airway. If the patient is intubated, this encourages the development of pulmonary oedema. If the patient is not intubated, it encourages the development of regurgitation of gastric contents and aspiration into the trachea.
- sudden fall in delivered oxygen even though supplemental oxygen is being delivered to the resuscitator if the victim inhales predominantly atmospheric air through the patient valve.

AIRWAY MANAGEMENT

Although definitive airway management is intubation of the trachea, a resuscitator may be used with jaw support or jaw-thrust only or with a Guedel-type airway. In that case, a facemask will be needed as well. A resuscitator may also be attached to the suprastructure of some oropharyngeal airways, an oesophageal obdurator tube, a Combitube, a pharyngo-tracheal airway or laryngeal mask airway. However, when used with these devices the

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risk of distension of the stomach is very high if the device is misplaced in the patient's airway.

OPERATOR-POWERED RESUSCITATORS NOT DEPENDENT ON AN OXYGEN SUPPLY

A number of brands are available (Figure 12.1). Recently, single-use, throw-away versions have become available but most are reusable. The principles of design are much the same in each brand. There is a compressible bag, which is squeezed by the hand of the operator, moving air to the victim through a valve whilst the other hand maintains head-tilt and jaw support (Figure 12.2) if the victim is not intubated. The volume delivered into the victim's lungs depends on the patency of the airway, the effectiveness of the seal of the mask on the face, the volume of the bag, its shape, the material of which it is made, the size of the operator's hand and the vigour of the squeeze. The International Standard requires that it must be possible for a resuscitator intended for use on adults to deliver a tidal volume of greater than 600 millilitres (ml) in a test lung system with a compliance of 0.2 litres per kilopascal (kPa) (20 ml/cm H_2O) and a resistance of 2 kPa/litre/second (20 cm H_2O/litre/second). In the case of fairly soft bags, the operator generally has a "feel" that an adequate volume has been delivered into the lungs but this should also be assessed by rise of the chest. With the less soft bags, this "feel" is lost. It is essential that the design of the resuscitator be such that the operator can achieve a peak inflation pressure of at least 6 kPa (60 cm H_2O).

During the expiratory phase, when the operator's hand releases the squeeze, the victim's expired gases pass to the atmosphere through the patient valve. There should be no back leak into the bag. At the same time, air enters the bag through a bag inlet valve which is usually positioned at the opposite end of the bag to the patient valve. The respiratory frequency which can be achieved by the operator depends on the inspiratory time and the expiratory time. The latter depends on the inspiratory tidal volume, the speed with which the bag can resume its unsqueezed position and the resistance in the bag inlet valve or any other valve which might restrict air entry to the bag. The International Standard requires potential minimum frequency of 20 per minute.

To most resuscitators of this type, it is possible to add oxygen (Figure 12.3). The addition of oxygen is not essential to the ventilatory performance.

The delivered oxygen concentration depends on the minute volume delivered and the flow rate of supplemental oxygen. It should be possible to achieve a concentration of at least 40 per cent with a supplemental flow of oxygen of 15 litres per minute. The addition of a reservoir bag or length of tubing at the site of the bag inlet valve will increase the delivered oxygen concentration so that it is possible to achieve a delivered oxygen concentration of 80–100 per cent. The addition of oxygen at the patient valve end of the bag may produce problems of valve jamming in some resuscitators if a rapid flow of oxygen is added. This is not a problem if the victim is being ventilated by mask but may be serious if the resuscitator is connected to an endotracheal tube in which case barotrauma may result.

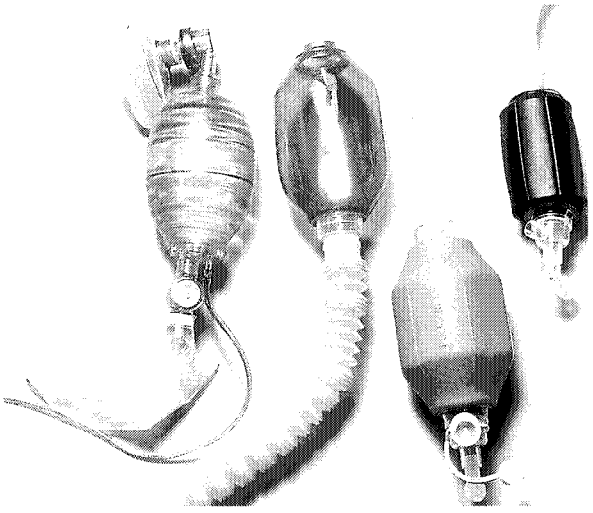


Figure 12.3 *Four examples of operator-powered resuscitators not dependent on an oxygen supply. It is possible to add oxygen to the four resuscitators. Each has either a bag or length of tubing which functions as an oxygen reservoir.*

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To some operator-powered resuscitators, it is feasible to add a PEEP valve. Whilst this is not usually indicated in the context of CPR, it may be useful to do so in patients who require emergency ventilation whilst suffering from pulmonary oedema or atelectasis.

In the hands of an expert, bag-valve-mask ventilation can be very effective but, in the hands of non-expert health professionals, the ventilation which is likely to be achieved is inferior to mouth-to-mouth or mouth-to-mask ventilation. The difficulty arises from the need to maintain with one hand head-tilt and jaw support which requires an upward pressure of the operator's hand, and seal of the mask on the face which requires a backward pressure. The usual result is a large leak and/or airway obstruction.

Ventilation can be improved if the resuscitator is operated by the two hands of one person, whilst another uses two hands to maintain the patency of the airway with head-tilt and jaw-thrust and the seal of the facemask on the face. It is not usual to have two skilled rescuers available and the use of hand-powered resuscitations by untrained or unskilled individuals should be discouraged.

If the tidal volume is delivered to a victim with an unobstructed airway over approximately 1–2 seconds, the peak airway pressure is unlikely to exceed 20 cm H_2O which is the opening pressure of the oesophagus. Gastric distension is no more likely than with mouth-to-mouth or mouth-to-mask ventilation.

When the patient has a cuffed endotracheal tube in place, ventilation is easily performed by one rescuer and barotrauma is unlikely unless there is serious patient valve malfunction. To avoid this unusual but dangerous complication, the resuscitator must always be checked before use by several squeezes of the bag whilst the patient valve is inspected.

It is essential that the victim should be able to breathe spontaneously through the device with the development of no more than normal subatmospheric pressure. In many bag-type resuscitators, the patient will inspire during spontaneous ventilation a similar oxygen concentration to that delivered during intermittent positive pressure ventilation. Unfortunately, in some resuscitators during spontaneous ventilation the inhaled gases come predominantly through the patient valve from atmosphere at low oxygen concentrations barely above 21per cent.

OPERATOR-POWERED RESUSCITATORS DEPENDENT ON AN OXYGEN SUPPLY

Partial rebreathing system

A simple, relatively safe system of delivering high concentrations of oxygen to an intubated patient is a valveless partial rebreathing system (Figure 12.4). The operator partially occludes the tail of the bag whilst squeezing the bag to inflate the patient's lungs. During expiration, gases pass into the bag and are partially washed out of the bag through the tail. Provided an oxygen flow of greater than 8 litres per minute is supplied to the bag, the delivered oxygen concentration should quickly rise to 90–100 per cent. Carbon dioxide elimination from the bag depends on the fresh gas flow of oxygen, and is adequate above 8 litres per minute oxygen supply.

The system is not suitable for ventilation by mask as leaks around the facemask can quickly lead to depletion of the contents of the bag before an adequate tidal volume is achieved. It is not easy to control the tail of the bag and squeeze the bag at the same time. Once the

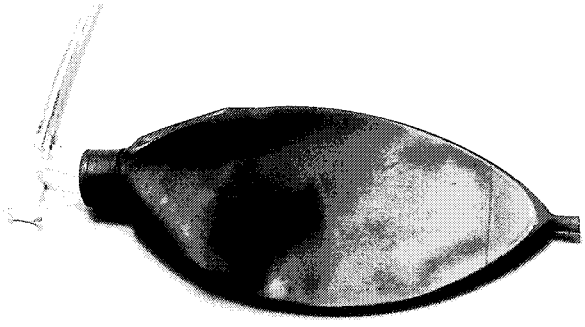


Figure 12.4 *An example of an operator-powered resuscitator dependent on an oxygen supply – partial rebreathing system.*

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patient is intubated, barotrauma is unlikely unless an inexperienced operator completely occludes the tail throughout a major part of the respiratory cycle. This may occur because the operator loses the "feel" of optimal chest expansion in a fully closed system.

Circle system

A small version of the standard circle system for anaesthesia can be used as a resuscitator. It is possible to achieve a delivered oxygen concentration of 100 per cent oxygen early in resuscitation. Ventilation of the patient by mask with low flow rates of oxygen may be difficult as leaks around the facemask may deplete the contents of the compressible bag before an adequate tidal volume is achieved. A low fresh gas flow in a non-intubated patient may limit the frequency of ventilation as it takes time for the bag to refill. However, the provision of an oxygen bypass flush which delivers temporary flow rates of oxygen in excess of 30 litres per minute overcomes these difficulties. Once the patient is intubated and the trachea sealed by the cuff, ventilation can be performed at low oxygen flows (eg 0.5 litres per minute) as expired carbon dioxide is absorbed in the soda lime. At these low flows, the rescuer has a good "feel" of satisfactory ventilation. Care must be taken that inappropriate settings or function of the blow off valve do not lead to barotrauma.

The system is well suited to spontaneous breathing. The system is potentially conservative of oxygen supply but, as is the case for all resuscitators dependent on an oxygen supply, care must be taken that sufficient is available for the expected duration of the resuscitation.

OXYGEN-POWERED RESUSCITATORS

Manually cycled

Oxygen is admitted at a high pressure to a valve system which drops the pressure to a safe working level. The peak flow level is set by the manufacturer or, in some models, may be adjusted by the user. The flow of oxygen is initiated and terminated by the operator pressing and releasing a button or lever. Rise of the chest suggests that a sufficient tidal volume has been delivered. When ventilation is performed with a facemask, a high flow rate has the advantage that ventilation can be satisfactory in the presence of a substantial leak. However, in the presence of an obstructed airway, the same flow rate can produce gross distension of the stomach as the airway pressure is likely to exceed the

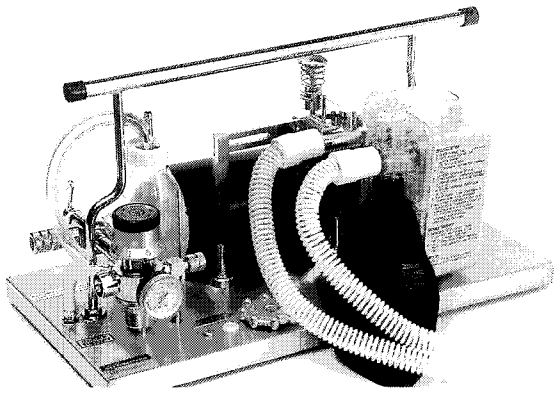


Figure 12.5 *An example of an operator-powered resuscitator dependent on an oxygen supply – circle system. (Photograph courtesy of Dr David Komesaroff.)*



Figure 12.6 *An example of an oxygen-powered manually cycled resuscitator.*

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opening pressure of the oesophagus. Generally, relatively low flow rates of 40–60 litres per minute are appropriate but a pressure limiting system at 6 kPa (60 cm H_2O) is also essential.

In a closed system, as applies in the case of the patient intubated with a cuffed tube, the risk of barotrauma is high. Manually cycled oxygen-powered resuscitators are suitable for use only by highly trained individuals. The rescuer has no "feel" of the lung ventilation and the rise and fall of the chest must be scrutinised with every breath.

Most of these resuscitators deliver 100 per cent oxygen but some entrain air and lower concentrations may be delivered. These resuscitators vary in response to spontaneous breathing. Some function as demand devices providing a flow rate of oxygen which matches the patient demand created by subatmospheric pressure in the airway. Others simply permit the victim to inhale ambient gases. Oxygen-powered resuscitators which do not entrain ambient gases are suitable for resuscitation in toxic atmospheres.

Automatically cycled

To reduce the risk of operator-dependent complications with oxygen-powered resuscitators and to free one of the operator's hands, automatically cycled resuscitators have been developed. These may be pressure, volume or time cycled at the end of inflation. As the flow rate is usually fixed, time and volume cycled devices are very similar. Pressure cycled resuscitators have two major disadvantages. Significant underventilation can occur in the face of increased stiffness of the lungs or chest wall or an increase in airway resistance. Even worse underventilation may occur during external cardiac compression as each stroke cycles the resuscitator. Automatic time or volume cycled resuscitators do not compensate well for leaks around a facemask but may be satisfactory with a tightly fitting facemask or endotracheal tube. Those which do not entrain ambient gases are suitable for use in toxic atmospheres and may be set up as a back-pack to facilitate movement of the victim.

CONCLUSION

A resuscitator is a very useful device in the hands of an expert. In the hands of the untrained or poorly trained, it is likely to be useless and even dangerous. In the case of individuals who have mastered the skills of jaw-thrust, but do not have the opportunity to practise ventilation of the unconscious human frequently, a two person technique is more likely to produce satisfactory ventilation. With the exception of the highly trained, mouth-to-mouth or mouth-to-mask are better options for ventilation especially as 50 per cent oxygen can relatively easily be achieved during mouth-to-mask ventilation. If a resuscitator is used, it is highly desirable that the highest delivered oxygen for that type be administered as soon as possible.

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PART

III

TRAUMA

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CHAPTER THIRTEEN

BURNS AND SCALDS

J. Fred Leditschke

Destructive injuries to the skin and underlying tissue caused by heat, cold, chemicals, electricity, friction and radiation are common. Most injuries are minor and can be managed by the casualty himself or herself, or by the first aider or paramedic without reference to a medical practitioner and without the requirement for hospital admission. First aid measures promptly applied may mean the difference between a moderate and a major burn or a moderate and a minor burn. In the long term, prompt first aid influences the resultant appearance and the degree of scarring or deformity.

ANATOMY OF SKIN

Skin consists of an upper portion known as the "epidermis" which is separated from the deeper layers by a germinal layer which is constantly producing new cells (Figure 13.1). These cells progressively move to the surface where they die and are rubbed off or washed away. New cells take from 14 to 21 days to reach the surface. The thickness of the epidermis varies from the thick horny or keratinised layer on the sole of the foot or palm of the hand, to the thin skin of the upper inner arm, back of the hand or top of the foot. Due to this variation in thickness, a person who walks through ashes may sustain a full thickness burn to the skin on the top or dorsum of the foot, which will require grafting, whilst the thick sole will only sustain a partial thickness burn and heal from surviving deeper tissues.

The dermis of the skin is the portion that lies between the germinal layer and the underlying fat. The dermal layer contains the sebaceous glands which secrete an oily material that helps to keep our skin waterproof. It also contains the hair follicles which vary in number from one area of the body to another. On the surface near the hair shafts and follicles are the sweat glands which have the ability to open up and lose water and salt. This helps to maintain the body's fluid balance as well as the concentration of salts or electrolytes in the body. In this layer is also a large mesh of blood vessels which branch progressively to form a mesh of capillaries as they approach the germinal layer. Nerve endings are also situated in the dermis – the greatest concentration being close to the epidermis. By opening and closing the dense network of blood vessels in the dermis, one is able to regulate the amount of heat retained in the body. By constricting the vessels (vasoconstriction), heat is conserved; conversely, by opening up the vessels (vasodilatation), heat is lost. Provided the air around the body is cooler than the body, heat is lost by convection from the body to the surrounding air.

The pigment cells in the germinal layer help to protect the body from ultraviolet light. The nerve endings in the skin also act as a protective barrier to warn the individual of danger. These danger-signal receptors coupled to the joint-response system of the muscles protect the individual from continual burn exposure.

Bacteria exist on the skin surface and in the hair follicles but the intact skin acts as a barrier to bacteria invading the body. A breach of the skin surface produced by burn injury or by a traumatic abrasion will allow bacteria, fungi and other organisms to invade the body.

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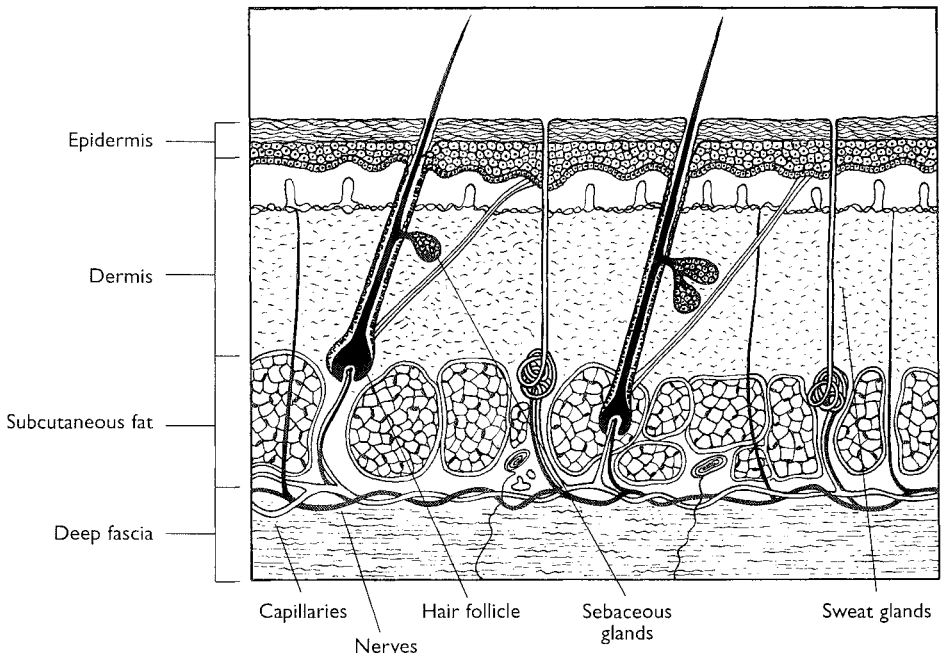


Figure 13.1 *The layers and structure of normal skin. The flakes of keratin and the hairs themselves are dead tissue. The dermis consists of living cells. After a burn, surviving living cells divide and form new skin and scar tissue.*

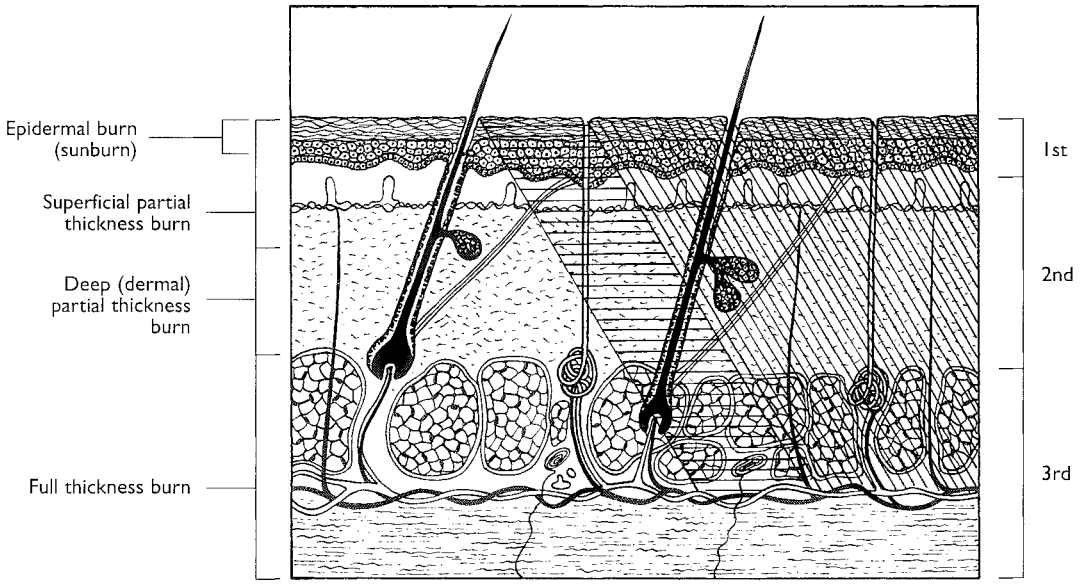


Figure 13.2 *Burns can be classified into four types, from first to fourth degree (the latter being the most severe), depending on the depth of tissue which is destroyed. In the case of full thickness burns, all skin cells in the region of the burn are destroyed and there are no remaining cells to generate new tissue to resurface the wound. Grafts from another part of the body are needed to cover the affected part.*

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BURN INJURY

Some examples of burn injuries to children may be seen in the colour plates section of this book. The depth of a burn depends on the temperature applied or generated in the tissues and the duration of application of the heat source (Figure 13.2).

First degree or superficial burns result from irritation to the epidermis. The commonest example is ultraviolet light producing sunburn. The capillaries open up and the nerve endings are irritated. If the process continues, some fluid may leak from the capillaries into the epidermis producing swelling and minor blistering. A similar erythema or reddening occurs following the application of hot water at a temperature of 43 degrees Celsius (°C).

Superficial partial thickness burns occur when the upper layers of the dermis are injured resulting in leakage of fluid from the capillaries into the tissues, producing blistering. Tissue and cell damage releases histamine, kinins and other chemicals which are responsible for stimulation of the nerve endings which the victim feels as pain.

Deep partial thickness burns involve the deeper layers of the dermis and may be dark red, purple or white in colour. If the capillaries are damaged, red cells can leak out into the dermal tissue producing a blotchy red or purple appearance which does not blanch on pressure. The deeper into the dermis is the burn injury, the more the nerve endings are damaged. Although there may be a painful response to sterile pin-prick in the first 24 hours, this response to pin-prick is often lost in deep partial thickness burns in the ensuing 24–28 hours as the swelling (oedema) increases.

Full thickness burns arise when all of the tissues in the dermis are irreversibly damaged or destroyed. In these cases the superficial layer will usually be dead-white or charred by the injury and the surface will be pain-free to pricking by a sterile pin or needle. Because the capillaries and vessels in the dermis are now damaged and no longer operational, fluid pours out beneath this dead layer. If the full thickness burn encircles the limb or the chest or the neck, the fluid poured out will act like a tourniquet and impair the circulation to the fingers. If it involves the neck, this swelling will limit the ability to breathe or cause obstruction in the neck and airway. Full thickness burns may involve complete destruction of the skin, fat and muscle, down to the bone. This occurs particularly in high voltage electrical injuries (over 11 000 volts).

A burn injury will show three zones, either laterally or in depth (Figure 13.3). At the outer margin will be the zone of erythema or hyperaemia where the damage is minimal and the blood vessels are opened up and blistering is occurring. Clinically, the area is pink, blistered and painful. The next zone is the area of stasis where the vessels have been damaged but, with good first aid, the changes are able to be reversed with a reduction of tissue damage, as in the zone of the erythema. The third zone of coagulation is where the tissues have been killed. If it is more than a centimetre or two across, the area will require excision to remove the dead tissue, and the use of skin grafts or skin flaps to cover the defect and heal the wound.

Destruction of the skin barrier creates an open wound through which bacteria from the patient's own hair follicles, sweat glands or gastro-intestinal tract, can enter the body. Similarly, the first aider or health professional can contaminate this injured tissue by touching it, or by droplet transmission as he or she talks or breathes heavily over the burn-injured part.

FIRST AID TO THE BURN WOUND

Following the application of the St John Ambulance DRABC action plan for first aiders (Danger, Response, Airway, Breathing, Circulation), attention turns to the burn wound to institute the three Bs, and the three Cs.

Three Bs

- Burn extinguished by stop, drop, roll
- Breathing – institute appropriate respiratory or cardiac assistance
- Body injury check

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Three Cs

- Cool the burn wound
- Cover the burn wound
- Carry the casualty to medical aid

Cooling the burn wound

The prompt cooling of the injured part, usually in cold water, is one of the oldest recorded methods of treating burns. As Surgeon Extraordinaire to the King and His Majesty's Household, James Earl in 1799 recorded several case histories noting the rapid and sustained relief of pain that following the application of ice packs or cold water. He wrote of the reduced severity of the tissue damage and the reduced scarring, and regretted "that this practice was so little known". Since that time, many experimental studies have been performed on a great variety of animals as well as human volunteers using both hot liquids as well as hot metal to produce a burn injury. People in Iceland have traditionally used cold water (10–20°C) in the treatment of burns and scalds. One of the most valuable reports described a 40- year-old woman who, as a child aged two years, had scalded her arm up to the axilla in a pot of boiling water. A bystander had thrust the child's limb into a bucket of cold water in which the water level reached only to her elbow. Thirty-eight years later the hand and forearm were quite normal apart from a smooth superficial scar on the back of the hand. From the elbow to the shoulder the arm was disfigured by ugly brown and white scars.

Since that time a great deal of experimental evidence has been accumulated which shows that cooling will rapidly reduce the tissue temperature, and that cooling will reduce the fluid or oedema formation in the burn wound. Cooling is excellent at providing pain relief and its application reduces the release of enzymes and other damaging substances, such as histamine, into the tissues.

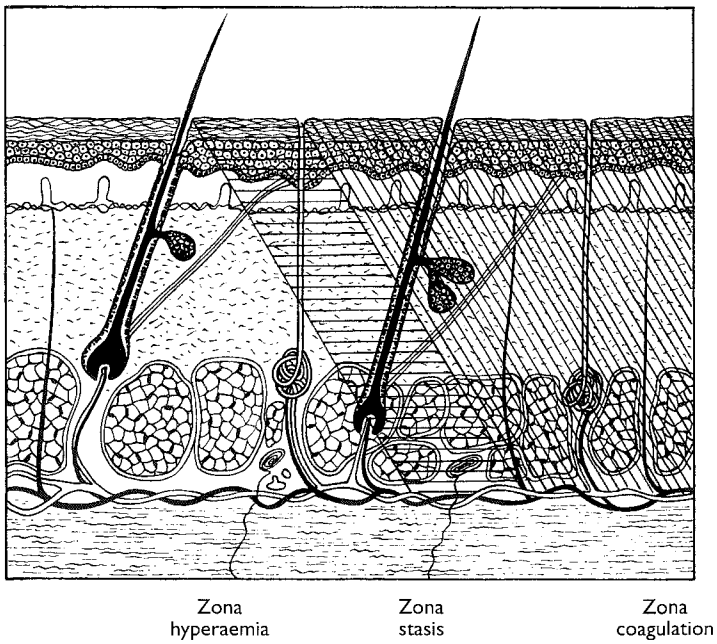


Figure 13.3 *When tissue is burnt it coagulates, and the chemical structure of proteins and fats changes (right). Beside or beneath this is a zone (the zona stasis) where the blood vessels have been damaged and capillary blood flow has stopped. The skin cells themselves may still be alive and, with correct first aid, healing will occur. The normal adjacent skin (zona hyperaemia) will be hot and red, and fluid will accumulate causing oedema.*

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- What temperature is best to cool the burn?
- How long should cooling be applied?

The underlying science of first aid provides some indication of the answers to these two crucial questions. Experiments have shown that tissues scalded in water (at 83°C for 30 seconds) returned to normal temperature or below within 30 seconds of exposure if the tissues were promptly cooled in iced water or immersed in water at temperatures between 20°C and 30°C. Thermally insulating material, such as clothing, covering the scalded tissue will delay the fall in tissue temperature. Sugar solution at 120°C, equivalent to hot jam or sticky toffee spilt onto the skin surface, has the potential to cause third degree burns. If tap water (at 10°C) is rapidly applied, the temperature in the dermis falls quickly to normal levels. If the skin is heated with a metal block at 100°C applied for 10 seconds, it takes several minutes for the temperature of the dermis to fall to normal in room air. However, the prompt application of ice cubes to the burns area reduces the temperature to normal in half the time. It also results in less severe burns, and in a marked reduction in the time to healing. There is less scarring.

However, from all the experimental evidence it is difficult to ascertain with precision what the maximum time may be following injury during which cooling is still beneficial, or how long the patient should be cooled, or at what temperature the cooling solution should be. The best experiments suggest that the lowest post-burn mortality is found when the cooling water temperature is between 20°C and 25°C, or the average water temperature at the tap from a domestic supply. Colder water is associated with a higher mortality, the increased death rate presumably as a result of whole body cooling or hypothermia.

Pain relief by prompt cooling has been known for well over 100 years. The threshold burning temperature for cutaneous pain sensation is approximately 43°C. A temperature of 56°C is required before the tissue proteins commence to coagulate. The greater the temperature the greater the damage, and the greater the duration the greater the damage (Figure 13.4). As a guide, a full thickness burn results from:

10 minutes contact with 49°C water

30 seconds contact with 54°C water

2 seconds contact with 66°C water

1 second contact with 70°C water

In children and infants with thinner skin, a shorter duration at the same temperature will produce a full thickness scald-injury.

Most electric hot water services are set in the vicinity of 65°C.

The capacity of the skin for heat transfer is considerable. If 43°C is the threshold temperature for cutaneous pain sensation, then cooling the surface with water will reduce the skin temperature quickly below that associated with cutaneous pain sensation. The application of iced water below 8°C has been associated with cessation of cutaneous nerve conduction and hence of perception of pain. Caution must, however, be expressed, for cooling temperatures near 0°C are potentially liable to produce frostbite or, in the case of extensive burns, equally serious total body hypothermia.

Cooling also slows the release of substances which cause pain such as histamine and bradykinin. Below 52°C, histamine release from cells does not occur. Above 56°C, large quantities of histamine, which is associated with inflammation and pain, are liberated. Cooling will inhibit the release of histamine and histamine-like products which cause some of the vasodilatation and leakage of tissue fluids from the blood vessels.

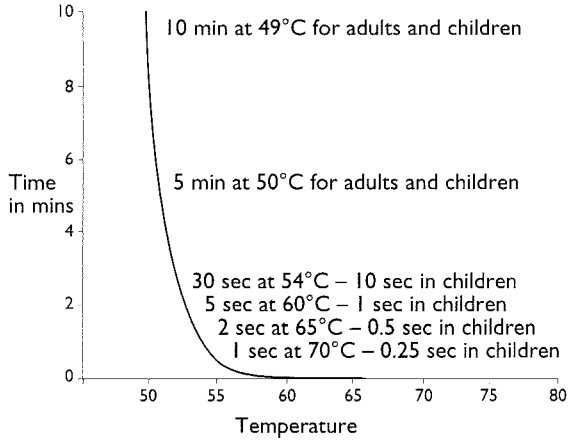


Figure 13.4 *The time:temperature curve leading to scalding of human skin.*

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The findings of a considerable body of animal experimental work and research performed on human volunteers leads to rational first aid procedures. From all the studies, cool water at 20–25°C appears to be as effective as iced water without the potential hazards of frostbite or hypothermia induced by temperatures near 0°C. Tap water is commonly the most readily available cooling fluid and even in hot weather, tap water is seldom above 20°C. Cooling must be immediate and to the injured part. The duration of cooling should be long enough to optimise pain relief and reduce tissue injury. There have been some conflicting articles suggesting that chronic swelling following prolonged cooling has occurred in some burn injuries associated with decreased wound healing. In small area burns, it is quite safe to keep the part immersed in cold water until such time as pain is relieved on removal from the water. Cold compresses need to be changed every 2–3 minutes, as heat transfer occurs from skin to compress. In the larger area burn, burns should be cooled for 10–20 minutes and then the cooling influence removed. This will avoid any drop in core temperature which might otherwise lead to hypothermia and the potential for cardiac arrhythmias, especially in children and the elderly group at special risk from major burn injuries.

Covering the burn wound

A sterile surface is created initially by the burning influence itself, whether this be from hot liquids, hot metal or strong chemicals.

After cooling the burn with tap water, which is for all intents and purposes sterile, the burn wound must be covered. Although the damaged skin may have been sterilised by the hot liquid or the flame, it is now a wound readily able to be colonised by either the victim's or the first aider's bacteria or microorganisms. All people have bacteria on their skin surface and in their hair follicles. Washing and drying the hands will reduce the number of bacteria, but will not eliminate them. Likewise the mouth contains a large number of organisms. When one breathes out and, in particular, when one coughs or shouts, these organisms are potentially expelled as droplets onto the burnt wound surface.

Tap water is chlorinated but, if tap water is not available, alternatives like cold milk, beer or fruit cordials can be used which do not contain harmful bacteria. In the absence of a sterile fluid, any source of water – even green stagnant – is preferable to allowing the heat to be retained on the surface of the body, particularly one covered with smouldering clothing. In the presence of a small area burn, cold water compresses can be used continuously until medical aid is available. In burns covering more than 10–15 per cent of the body surface and certainly over 20 per cent, these cold compresses must be removed and replaced by a clear or sterile covering after 10–15 minutes or earlier if the patient shivers or complains of cold. This covering may be a cotton sheet, clean towels or even newspapers are effective. Plastic garbage bags make an excellent agent to exclude the burn wound from the environment. The use of kitchen plastic film such as Cling or Glad Wrap applied directly to the wound surface is used extensively in hospital burn units. In the first aid situation it may be applied direct to the wound surface or, in the case of small burns, used to secure the wet compresses in place.

The passage of air over an open wound causes pain which is distressing to the casualty, and to see the wound may be distressing to the first aider and bystanders. Thus to prevent contamination of the wound, to prevent air movement over the wound, and to reduce the psychological impact, burn wounds should be covered.

Many proprietary preparations are marketed to treat burns. These range from extracts of *Melaleuca alternifolia* to extracts of aloes. Many are in popular lay use but their benefits are anecdotal and scientifically unproven. In the factory workplace, watergel blankets have been advocated in place of cold water.

Topical treatment

Prior to the 1960s, infection of the burn wound was a major problem and one could always tell the burn unit by the smell of infected wounds emanating from the ward. Deep partial thickness burns were often converted to full thickness burns by infection at the interface of the dead and living tissue.

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In 1915, Leonard Bickle, honorary surgeon at the Adelaide Hospital, gave an excellent review of the products available up to that time. Carron oil (a mixture of limewater and linseed oil) was the recommended treatment for many years. It was kept at all industrial sites. Its main disadvantage was that the oil spoiled everything with which it came in contact and secondly, the smell was nauseating and objectionable. It was suggested that sprinkling lavender water or eau de Cologne or one's favourite perfume would overcome some of the objections.

Picric acid ousted carron oil treatment but its great disadvantage was that, being a permanent yellow dye, it stained everything. It was also associated with case reports of poisoning resulting from absorption when applied to extensively burned surfaces. Another treatment was lead lotion which had a soothing effect but an objectionable smell. Liquid lead acetate was said to have had no adverse effects, and has been used through the ages; when used as a dressing for burns it was kept moist by regular irrigation. Other treatments included boric acid ointment and petroleum jelly, the latter being used especially in the Royal Navy. As recently as 1934 a first aid book recommended covering the burned part with a clean cloth or cotton wool soaked in carron oil or olive oil. It was suggested that oil of eucalyptus be added to make the product more antiseptic. Other soothing applications were petroleum jelly or castor oil. It is difficult to understand why the application of cold liquids was not more widely publicised at that time.

The breakthrough in the prevention of the burn wound sepsis was the development of topical antimicrobial agents in the 1960s. These limited the proliferation of bacteria which were known to colonise and invade the burn wound. This decreased markedly the incidence of invasive burn wound infection or sepsis and eliminated the smell of wounds infected with *Pseudomonas* bacteria. The three products which came on the market in the 1960s were 0.5 per cent silver nitrate solution, mafenide acetate (Sulfamylon) and 1 per cent silver sulphadiazine (Silvazine).

The 0.5 per cent silver nitrate solution was promoted by Carl Moyer of St Louis in the United States, being applied as a multiple layer occlusive dressing saturated with the solution. It was changed two or three times a day. It was extremely effective against bacteria, there was an absence of hypersensitivity reactions, it was painless to apply and it helped in the control of the elevated temperature frequently seen in patients following major burn injury. The disadvantages of its use were electrolyte abnormalities and the black staining of the patient, the staff, the sheets, the floor, the walls and everything which came into contact with the silver nitrate. Its use has now been largely superseded by the development of silver sulphadiazine.

Silver sulphadiazine is an insoluble salt obtained by mixing sulphadiazine and 1 per cent silver nitrate. It has a broad antibacterial spectrum, is not painful to apply and is effective against most organisms. Its disadvantages are that it is of no use once infection has been established in the burn wound. Silver sulphadiazine is applied to the burn wound following deflating and/or removal of the blisters and the debris at a definitive care centre, and applying a low adherent dressing. It is washed off each day in a saline or chlorhexidine bath and a 1–2 mm layer reapplied. The development of silver sulphadiazine has markedly reduced the frequency of deaths from burn wound sepsis.

In minor wounds the application of Vaseline gauze or chlorhexidine-impregnated gauze called "Bactigras", or the use of Betadine Cream in some countries, covered with multilayer gauze has proved to be very satisfactory. The dressing can be changed every 3–4 days. In India, Keswani has used boiled potato peelings as a dressing for partial thickness burns and, in Thailand, steamed banana leaves have also been applied. In countries where Silvazine is not available, honey has had its advocates.

In uncomplicated superficial burn wounds of less than 10 per cent of the body surface, in a patient with good body defence mechanisms and who is not debilitated by associated illness or diseases, it is probably immaterial what topical agent is used. Proprietary agents such as Ungvita has had its advocates, as has Savlon. In looking at topical agents for the management of burns, silver sulphadiazine stands out as the product above all

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others which has been most effective at reducing burn wound sepsis and its associated morbidity and mortality.

Mafenide acetate (Sulfamylon) is able to penetrate the dead tissue and is excellent at controlling bacterial growth on the burn wound surface. It is also of use in treating deeply colonised or infected wounds. Its major side effects are that it causes severe pain following its application to most patients, and many develop severe metabolic acidosis, or upsets in their electrolyte balance. For these reasons it is now no longer used in Australia.

Products such as butter, margarine, olive oil and other greasy substances should not be applied. These will limit heat loss and will be required to be washed off on arrival at the burn unit prior to the application of silver sulphadiazine cream and the covering with a low adherent (non-stick) dressing.

RESPIRATORY AND INHALATIONAL INJURY

Early and adequate intravenous fluids have eliminated shock from fluid loss as a cause of death; likewise, deaths from burn wound infection have been much reduced by the development of the topical antibacterial agents. Inhalational injury remains the most significant cause of death in patients with burn injuries. The intensity of the heat may cause burns to the face but it is the inhalation particularly of smoke, with its chemical products of incomplete combustion, which leads to the major damage. Hot air and in particular hot steam in industrial accidents will cause damage above the vocal cords, while the lower airway is usually damaged by the inhalation of smoke and the toxic gases given off during the fire. Smoke contains hot solid particles was well as certain chemicals which dissolve in the tissue fluids causing damage to the airway and lungs.

Hypoxaemia is probably responsible for most deaths from smoke inhalation. Carbon monoxide is produced by the incomplete combustion of carbon and occurs when a fire develops in a confined space. Carbon monoxide is a tasteless, odourless gas. It also has an affinity for haemoglobin 200 times greater than that of oxygen. The carbon monoxide displaces the oxygen from the haemoglobin making it difficult for the body tissues to extract oxygen from haemoglobin. The lack of oxygen may damage any organ but particularly has an effect on the central nervous system, with symptoms ranging from disorientation to coma.

Other injuries resulting from smoke inhalation during a fire may include cyanide poisoning from burning fabrics or polyurethane, life-threatening swelling of the face, mouth and oropharynx, and lower airway obstruction resulting from the inflammatory oedema fluid which is poured out into the alveolae and smaller air passages.

In short, every burn victim rescued from a smoke-filled environment or an enclosed space containing hot gases must be assumed to have sustained an inhalational injury. Treating any burn wound is, therefore, only a part of emergency care in such cases. Hypoxaemia being one of the major threats to life, oxygen should be administered to all casualties with potential inhalational injury, and continued until it is confirmed later in hospital that oxygen therapy is no longer needed. Ideally, 100 per cent oxygen should be delivered, but this is very difficult to achieve with a facemask. A high oxygen flow of 15 litres per minute will help. The higher the oxygen content of the inspired air the sooner the carbon monoxide will be removed from the blood and the hypoxaemia reversed. The administration of bronchodilators in conjunction with the oxygen will often help overcome the airway obstruction.

A FINAL COMMENT

Whilst advances in medical and para-medical technology for the control of infection and the reversal of hypoxaemia have been important in treating victims of the various heat-related injuries dealt with in this chapter, it remains vital for the first person on a burning or scalding scene to remember, and implement, the two Big Cs – Cool and Cover.

More importantly still, it must be remembered that almost all injuries of this kind are eminently preventable.

The best treatment for burns is prevention.

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CHAPTER FOURTEEN

CRUSH INJURY

Harry Oxer

The pre-hospital management of the crushed victim has been one of the most controversial subjects in the teaching of first aid. In no subject has there been a greater requirement for the application of science rather than dogma and tradition.

Three definitions are used in this context.

Crush injury a general term used to cover any injury caused by crushing or compression.

Compartment syndrome the local manifestations and sequelae of neuromuscular ischaemia due to increased pressure within an osteofacial compartment.

Crush syndrome the delayed systemic manifestations of muscle necrosis resulting from crush injury or compartment syndrome, which includes acidosis, hyperkalaemia, myoglobinuria, shock and acute renal failure.

Pressure on a limb may cause

- direct damage to skin, muscle, vessels, bones and other tissues;
- ischaemia, by pressure interrupting the blood supply; and
- interference with circulation due to gross swelling.

EARLY RELEASE OF CRUSHED VICTIMS

Crush injury was first described by Von Colmers in the Messina earthquake of 1909 and the syndrome was first reported in the official German handbook on medical studies in the First World War.

In the early 1980s concerns about the effects of release of toxic substances into the circulation on releasing a crushed limb led to varying recommendations on how or when to release the crushing force. Much debate ensued, with the development of doctrine that believed that the crushing force should not be released until a medical team had arrived at the accident site. The team would then presumably apply some "medical magic" that would prevent the patient dying. Tourniquets were suggested, to "keep the toxins confined to the injured limbs" and, presumably, to prevent sudden death.

An extensive literature search, mostly in the English language, has failed to find any reports of patients who died immediately upon release of the crushing force. In the greatest human catastrophe of the twentieth century – the earthquake of 28 July, 1976 in the city of Tangshan, on China's north east coast, thousands of people were crushed. Deaths due to crush syndrome in the pre-hospital phase of management were not reported. Of the 164 800 injured who survived, survival rates of trapped and crushed victims fell dramatically as entrapment times were prolonged. For this reason, and from a distillation of the world's recent medical literature on this subject, the current first aid doctrine is now to release an entrapped or crushed victim as quickly as possible.

HISTORICAL REVIEW

After Von Colmers' 1909 paper first defining the crush syndrome, an understanding of the pathophysiology soon followed.

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The damaged, underperfused muscle and soft tissue becomes acidotic and releases substances into perfusing blood and lymph vessels when the circulation is restored. The raised potassium level may disrupt cardiac rhythm. The free and circulating myoglobin is filtered at the glomerulus into an acid and concentrated urine, and precipitates in the distal convoluted tubules of the kidney. This reduces reabsorption of water and leads to greatly increased intrarenal pressure by this and other mechanisms. Kidney failure may ensue.

As early as 1941, it was found that renal damage could be prevented by copious oral administration of alkaline fluids containing sodium bicarbonate. This advice was eventually officially issued in March 1943 as a Ministry of Health memorandum. In March 1943 over 200 people were crushed when jammed together for over two hours on the stairs of an underground bomb shelter in London. Many died, but 60 survived and were quickly removed to local hospitals. The clinical details of 12 patients were carefully recorded. One of these died within 45 minutes of admission from pulmonary and cerebral haemorrhage due to suffocation. Although they showed evidence of crush injury, each of the others made an eventual complete recovery with treatment.

Little advance seemed to have been made in the understanding of this injury until around 1987 when Stewart wrote that limb compression can injure through either ischaemia or muscle destruction. It was found that a crushing pressure of 40–60 millimetres of mercury (mm Hg) will start functional degeneration in nerves and muscles after 15 minutes, and can lead to ischaemic necrosis within 4–8 hours. Systemic manifestations of muscle damage and necrosis can follow prolonged pressure even from a patient's own body weight – this can occur if the patient is unconscious after drug overdosage, or is undergoing an operation. Such damage can result from pneumatic antishock (MAST) garments, if applied for too long. Muscle, severely damaged by entrapment, releases its contents into the circulation on being freed, and causes rising blood urea, high serum concentrations of potassium, phosphate and uric acid.

It was Stewart's report that stated that the high serum potassium concentration, especially if associated with a low serum sodium concentration, could cause cardiac arrhythmias and arrest soon after release; he quoted Allister, writing in the British Medical Journal in 1983, as the source of this belief. It was on these reports that many first aid bodies in the world changed their doctrine, and recommended that rescue workers and first aiders do not release trapped victims quickly.

This, however, was wrong advice. It transpired that Allister quoted only one case of "crush syndrome" – that of a 47-year-old man who was crushed in a building collapse, and not released for 8 hours. His cardiac arrest occurred one hour after his release from crushing, and he was successfully resuscitated. Nevertheless, rescue and first aid doctrine continued to be influenced of the fear of causing harm by the release of a trapped victim. One report suggested that a tourniquet should perhaps be applied before freeing the limb, and not released until arrival in hospital.

In 1984, Michaelson *et al.* described seven patients trapped in bombed houses, all with severely crushed legs. In three cases the crush injury involved the pelvis as well. All were trapped for at least 6–12 hours, and it took up to 12 hours to bring them to hospital after being rescued. None had more than 1 litre of isotonic crystalloid intravenous solution before arrival at hospital. Although all were hypotensive and dehydrated, and two had a systolic blood pressure below 80 mm Hg, there were no deaths, either on release or later after treatment.

Seven more cases were reported by Reis and Michaelson from Haifa in Israel. All patients who survived did so because they escaped injury to the head, neck and trunk. When the abdomen and chest are crushed, death ensues rapidly from cardio-respiratory failure.

The length of time that the crushing force acts is vital information as regards therapy and prognosis. Kikta *et al.* reported four patients with crush syndrome due to prolonged limb compression following drug overdosage. They found that peripheral pulses were usually intact, and often remained so in these cases despite massive oedema and tissue swelling. The "third space" losses into the injured extremity may, however, be

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sufficient to cause hypotension, although other causes of hypotension must also be considered in patients with multiple trauma. Hypotension is almost always due to volume deficit.

Reis and Michaelson from Israel noted that the length of time that the crushing force acts is vital information as regards therapy and prognosis. They reported a series of 15 people with crush injury of the lower limb. None of the victims were acutely hypotensive or at risk of sudden death after removal of the crush injury.

Animal experiments with total limb ischaemia showed myoglobinuria develops after 4–8 hours and irreversible nerve and muscle injury after 12 hours. Thus rapid release of the trapped victim is imperative.

This historical review has been presented because seldom in the history of rescue and first aid work, has a single reported case so distorted the development of correct doctrine. In cases of the reports mentioned above, when the authors spoke of the risk of sudden death after release of a crushing force, they cited the case from Glasgow, reported in the 1983 *British Medical Journal* article. What was this case?

Dr Charles Allister reported *one case* of cardiac arrest and successful resuscitation which occurred *one hour after release* from prolonged crushing. He suggested that biochemical abnormalities could be a cause of sudden death after release from severe prolonged crushing. In this case the victim was a 47-year-old man crushed when three floors of a derelict building collapsed. He was trapped by a large mass of debris, and only his head, right arm, and shoulder were exposed. He was fully orientated, his pulse was 90 beats per minute and ventilation was adequate.

He was released after 8 hours. There were no obvious major injuries. Blood pressure was 90 mm Hg systolic and pulse 100 beats per minute. A cardiac arrest occurred one hour after release from crushing, and blood taken at that time showed that he was acidotic pH 7.15, and that his serum potassium was 8.0 mmol per litre. Resuscitation successfully restored sinus rhythm, normal blood pressure and spontaneous breathing, and he was given 50 ml of 50 per cent dextrose and 10 units of soluble insulin. His potassium concentration fell steadily over the next two hours to 5.0 mmol per litre, which is normal.

The crush syndrome is a very real phenomenon, but it is an issue for the doctors in accident and emergency departments of hospitals and those in intensive care units – not one to delay rescue, release or first aid at the accident site.

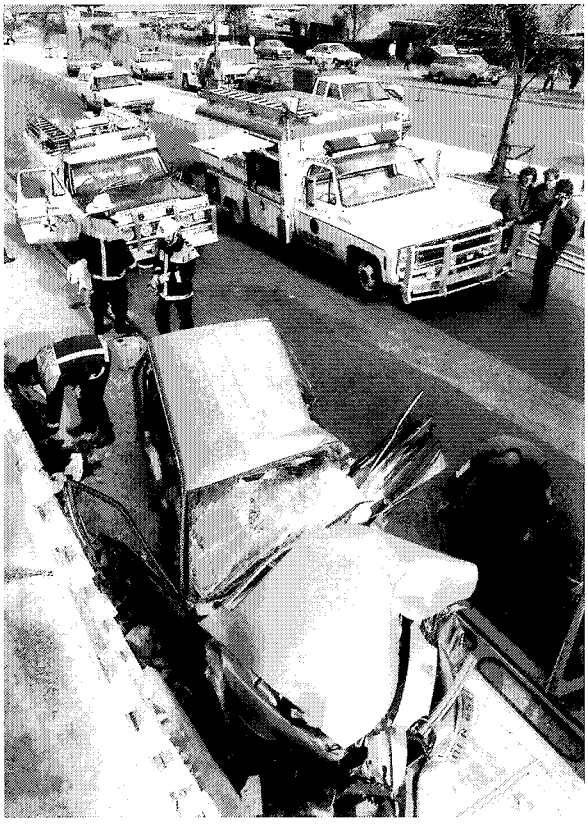


Figure 14.1 *One of the most common scenarios involving crush injury – the two-vehicle head-on collision. In such cases it may not always be physically possible for unequipped first aiders to remove the crushing force, but they should certainly not be deterred from doing so merely because of earlier teaching about the supposed effects of crush syndrome. (Photograph courtesy of the Ambulance Service of New South Wales.)*

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CRUSH INJURY – SUMMARY

Applying a crushing force to a limb may

- cause direct death of some tissues by disruption;
- damage tissues, causing swelling, inflammation and loss of function; and
- restrict or cut off blood supply by pressure on vessels, causing tissue ischaemia and hypoxia.

Damaged and hypoxic tissues

- develop capillary leak, and oedema; and
- release myoglobin, potassium, lactic acid, creatine, kinase, and other substances.

On release of a crushing force, there may ensue

- rapid swelling and oedema;
- bleeding from ruptured vessels; and
- release of large quantities of potassium, metabolic acids, and myoglobin into the circulation.

These may cause

- early (one to two hours after rescue):
  - hypotension,
  - shock,
  - heart irregularities,
  - pain, swelling.
- late (days after rescue):
  - rising blood urea, uric acid, potassium phosphate,
  - renal damage and failure.

CONCLUSION

First aid is all about doing what you can, with what you've got, to help the injured, before the arrival of professional medical help. Its teaching should be aimed at doing the best you can for the current problem.

Crush injury is rare. It will be rarer still for the first aider.

Crushing forces involving the head, chest or abdomen, *must* be removed as soon as possible – otherwise they will cause death.

Crushing forces involving limbs should also be removed as soon as possible. The risk of sudden death following removal of the crushing force is extremely small, if it exists at all.

The longer a crushing force is left in place, the more damage is done to the tissues, and this may become irreversible.

There is no justification in the literature for delay in removing crushing forces from a trapped victim and commencing first aid, unless medical help will be available within minutes. However, a common sense approach must still be adopted, as in all good first aid. For example, if it is evident that on-site amputation will be needed, the first aider may well decide to wait for the arrival of the experts. In the meantime, there are always the essentials of the St John Ambulance DRABC action plan to observe – especially the monitoring of the casualty's airway, breathing and circulation.

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CHAPTER FIFTEEN

DIVING ACCIDENTS, PULMONARY BAROTRAUMA AND GAS EMBOLISM

John Williamson

Understanding what may have happened to a distressed diver is an important theme for advanced first aiders, paramedics and the attending physicians. Millions of people, world-wide, hold certificates to dive using compressed air. Diving accidents are common. The ability to administer first aid to SCUBA (Self-Contained Underwater Breathing Apparatus) or SSBA (Surface Supply Breathing Apparatus) divers is thus an important skill for all who take their place in first aid, rescue and emergency care teams.

An understanding of the scientific basis of first aid for specific diving injuries requires familiarity with the following principles.

1 Buoyancy as applied to solids, liquids or gases immersed in liquids (eg Archimedes' Principle)
2 Pressure transmission within liquids (eg Pascal's Principle)
3 The "ideal" gas laws:
   - Boyle's Law
   - Charles's Law
   - Dalton's Law
4 Henry's Law of solubility of gases
5 The functional anatomy of the human cardio-respiratory systems

CLASSIFICATION OF DIVING ILLNESSES RELEVANT TO THE FIRST AIDER

A *Non-specific diving illnesses and injuries*

- near-drowning
- trauma from knives, propellers, deck machinery, compressed gas accidents, and marine animal bites
- head injury
- hypothermia and hyperthermia
- sea sickness
- seafood poisoning
- concurrent illnesses (eg asthma, infections, ischaemic heart disease, diabetes)
- marine envenomations

B *Specific, hyperbaric and hypobaric ("dysbaric") related illnesses*

1 "Squeeze" phenomena
2 Decompression illnesses
   - decompression sickness, or "the bends" (outmoded terminology)
   - gas embolism (venous or arterial)
   - pulmonary barotrauma:
     emphysema and complications,
     pneumothorax and complications,
     arterial gas embolism.
3 Inner ear barotrauma
4 Contaminated breathing gas
5 Dysbaric osteonecrosis

BAROTRAUMA

A human body can be considered as a soft-walled bag of fluid, containing a bony skeleton. This bag of fluid contains various air-filled or gas-filled cavities, all of which have communication with the outside world,

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under conditions of normal health and function. The cavities are of two kinds. One kind has rigid walls, and resists compression, expansion, and deforming. The other kind has soft, compressible walls, collapses and/or deforms readily, but will not expand (stretch) beyond a certain limit.

Examples of the rigid-walled kind are the facial air sinuses, middle ear cavities, and any dental cavities, all located in the head. Another "created" example is the space behind the diver's facemask, in front of his eyes and nose (Figure 15.1). Examples of the collapsible cavities are the alveoli and smaller air passages of the lungs, and any gas spaces in the bowel lumen. Bowel gas-related complications of diving are very uncommon, and seldom of significance to the first aider.

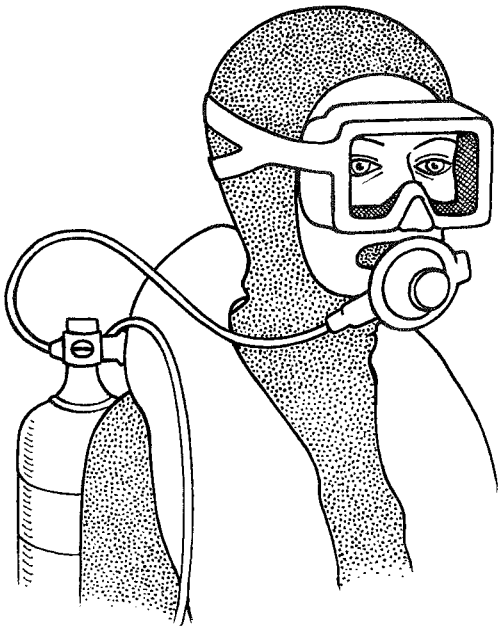


Figure 15.1 *The air space behind a diver's facemask. The pressure in this space can be equalised during descent simply by the diver exhaling through his nose. Inexperience, distraction or nasal obstruction during descent may result in the soft tissues of the diver's face enclosed by the mask being forced into the space, with resultant bruising and congestion of the tissues – especially the conjunctivae (see Plate 4). This results in a "mask squeeze". (Illustration courtesy of Yvonne Van Pelt, Medical Illustration Unit, Royal Adelaide Hospital.)*

A diver floating upon the surface of a body of water at sea-level has an ambient pressure acting upon his or her body of 1 atmosphere absolute (ATA) – the same as that acting upon the reader of this text, unless he or she is significantly above sea-level as, for example, in an aeroplane or on a mountain.

When the diver descends beneath the surface, the ambient pressure acting upon his or her body increases with depth, at a rate of 1 ATA for every 10 metres (m) increase in depth of sea water below the surface (slightly more than 10 m in fresh water). Thus at 20 m depth in sea water the ambient pressure has tripled to equal 3 ATA – a very significant increase! It should be noted that in diving the magnitude of ambient pressure changes far exceeds those in aviation; the latter can never be greater than 1 ATA!

Conversely, when the diver begins to ascend towards the surface, the ambient pressure will decrease, until on his or her reaching the surface (safely or otherwise), the ambient pressure will once again be 1 ATA.

If the air pressure in a rigid-walled body cavity in the diver (eg frontal sinus) is unable to equalise with the changing (increasing during descent, and decreasing during ascent) ambient pressure, pain and injury in that cavity will occur. This risk will only exist if communication between the rigid-walled cavity and the pressure "outside" the diver's body is interrupted (eg blocked sinus ostium or, in the case of the middle ear, a blocked Eustachian tube) at any stage during the descent or ascent.

On the other hand, a non-communicating soft-walled or collapsible gas cavity will simply undergo reduction in volume during descent (increase in ambient pressure), and re-expand back towards its normal starting (ie surface) volume during ascent (decreasing ambient pressure), in accordance with Boyle's Law of gases. This is what happens in the lungs of a breath-hold diver or snorkeller, and is virtually without risk at customary depths for breath-hold divers with healthy lungs. However, if the diver is breathing air or any other gaseous breathing mixture underwater (eg SCUBA or SSBA), a different situation applies.

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It is necessary to appreciate that, when a diver breathes a gas mixture underwater, the "second stage" of his breathing regulator delivers the breathing gas to his mouth, throat and lungs at ambient pressure at all times, irrespective of what depth below the surface he may be. This is essential to enable the diver to take a breath underwater against the water pressure acting upon his chest. Thus during SCUBA or SSBA diving, the partial pressures of all the gases making up the breathing mixture will be increased with depth, and the sum of these partial pressures will at all times equal the ambient pressure acting upon the diver's body.

In the case of a rigid-walled cavity, such as the middle ear, a blocked Eustachian tube during descent in either a breath-hold diver or a diver breathing underwater will result first in the ear drum (with pain) and the round window (without pain) being pushed towards the middle ear cavity. This will be followed by inflow of blood (which, as described by Pascal's Principle, always remains at ambient pressure) into the layers of the ear drum and the mucous membrane lining the cavity of the middle ear. This is a "squeeze". Conversely, if the tube becomes blocked after descent, or during ascent, the pressure in the cavity will be greater than ambient as ascent continues. This is a "reverse squeeze", which may also produce pain. Exactly the same sequence of events may occur in the facial sinuses if their ostia are blocked, resulting in pain in the face, usually overlying the location of the affected sinus. Similarly, dental cavities due to caries or faulty fillings may result in "dental squeeze" or "reverse squeeze", from pressure on the dental nerve.

In the case of the soft-walled cavity (pulmonary alveolus), obstruction of the air passage leading to it during descent (natural during breath-hold diving, unnatural in SCUBA or SSBA diving) carries no particular risk of pressure damage. It will simply tend to decrease in size and volume, in both kinds of divers. However, the significant situation from the first aid point of view is the SCUBA or SSBA diver who descends without incident, but whose air passages become obstructed following descent (eg mucous plugging with a chest infection or possibly asthma), or during ascent (eg unwitting breath holding during ascent). In this situation the gas trapped in the obstructed alveoli will continue to expand during ascent (decreasing ambient pressure, Boyle's Law), until the maximally stretched alveolar walls tear, releasing gas into one or more dangerous places.

Finally, it should be appreciated that the above considerations apply equally to all persons exposed to any dysbaric environment, either hyperbaric or hypobaric (eg divers, astronauts, aviators, caisson workers, hyper- and hypobaric chamber attendants).

1 "Squeeze" (descent) and "reverse squeeze" (ascent) phenomena

Cavities affected

- facial sinuses (maxillary, frontal, ethmoid, sphenoid)
- middle ear cavity
- any significant dental cavities
- facemask "cavity" (see Figure 15.1)
- pockets of trapped air under a badly fitting wet suit

A middle ear squeeze may also put the inner ear at risk of barotrauma.

Signs and symptoms of a squeeze

As the blood is always at ambient pressure, it will flow into the capillaries in the mucous membrane lining the walls of a squeezed cavity, down an increased pressure gradient. The capillaries and the lining will become first congested and swollen, then haemorrhagic, and then blood and blood-stained serum will leak out of the damaged, overdistended capillaries, into the blocked cavity itself. As this accumulates it will occupy an increasing volume in the blocked cavity, until the gas trapped in the blocked cavity and the blood together reach a pressure equal to ambient.

Thus divers will first experience pain from the cavity if they continue to descend, which will tend to disappear once they reach a constant depth. Subsequent ascent will often see a recrudescence of the pain. If the now reverse squeeze "blows" the obstruction clear during or shortly after ascent, this may release a sudden flow of

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blood-stained fluid (say into the facemask from a nostril, in the case of a sinus squeeze), usually accompanied by an equal reduction in the pain. If the squeezed cavity is the middle ear, symptoms of pain (earache), hearing loss, and even tinnitus and giddiness may occur or recur during ascent.

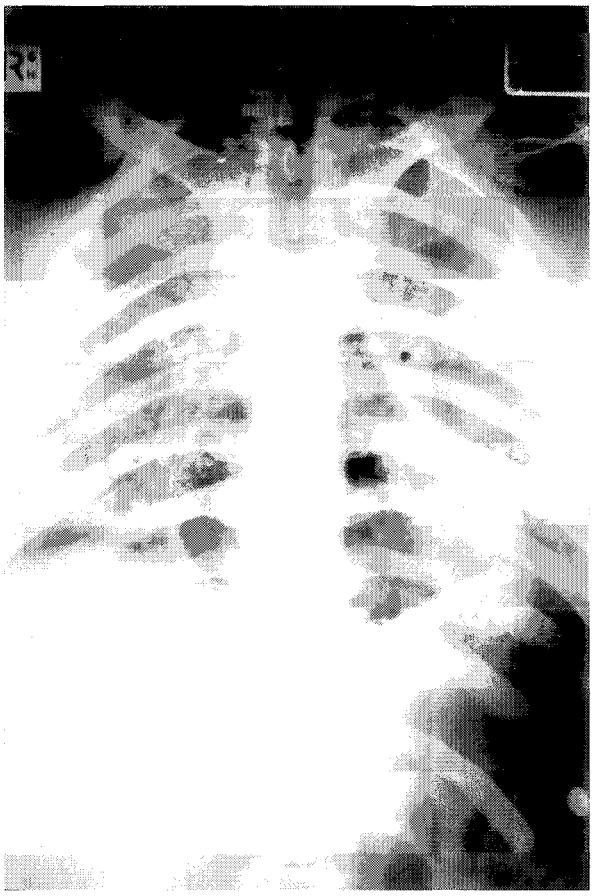


Figure 15.2 *Erect plain chest radiograph of a deceased diver, within hours following a panicked ascent resulting in pulmonary barotrauma and massive arterial gas embolism. Note the cardiac chambers empty of blood and distended with gas. Note also the grossly distended stomach (a common feature following a period of bare-handed resuscitation in the field) and the absence of any pneumothorax. Gross distension of the internal jugular veins with gas is also visible. In such a situation external cardiac compression will not generate a palpable carotid pulse. (Radiograph reproduced with the kind permission of the editors of the* Medical Journal of Australia.*)*

Incidence of squeeze phenomena among divers

Squeeze during descent is far more common than the reverse squeeze of ascent involving rigid cavities. Middle ear squeeze is by far the commonest diving malady requiring first aid and medical attention. It may occur in the untrained and inexperienced, divers with upper respiratory tract infections (colds), hay fever, upper airway pathology (trauma to nose, nasal polyps, etc.), smokers, and "bounce" divers (ie divers who engage in repeated or partial ascents during diving).

First aid principles

This involves management of a painful, inflamed, bleeding, and potentially infected middle ear or sinus, or a severe toothache, or congested upper face with "bloodshot" eyes (mask squeeze – see Plate 4). If barotrauma of the middle ear has been sufficiently severe, actual tympanic perforation may have occurred. Immediate cessation of diving, resting in the head-up position and referral for qualified medical or dental care are essential. If concomitant barotrauma to the inner ear is suspected, the same treatment principles apply, but are more urgent.

2 Decompression illnesses

Some diving and hyperbaric related illnesses have recently been reclassified under the heading "decompression illnesses" (DCI). They comprise all medical misadventures associated directly with decompression, and involve all hyperbaric and hypobaric conditions. Theoretically the decompression illnesses would include the reverse squeeze phenomena, but for convenience these have been considered under squeeze phenomena, above.

Prior to this reclassification it was customary to consider the two remaining major decompression illnesses as separate diseases, viz:

a decompression sickness, or "the bends", and

b arterial gas embolism.

It is now clear that this differentiation is contrived, and impractical for the following reasons.

- Tissue and intravascular bubbles are known to exist both in divers with decompression sickness, and in

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asymptomatic divers following normal dives.

- Arterialisation of venous gas bubbles occurring in decompression sickness, or even following a "normal" dive, may give rise to cerebral arterial gas embolism (CAGE). Resulting symptoms may be clinically indistinguishable from what was called "decompression sickness".
- Decompression sickness and CAGE can and do co-exist.
- The long-term neurological effects of decompression sickness and CAGE are frequently indistinguishable.
- In submarine escape trainees who develop acute symptoms of CAGE, it is not possible to exclude arterialisation of venous gas.

Thus for the purposes of this discussion, and pending further advances in understanding of the pathogenesis of the decompression illnesses (DCI), these two conditions (the former "decompression sickness", and CAGE) will be considered as one illness (DCI) having differing grades of clinical severity. There does appear to be a form of massive AGE (arterial gas embolism) resulting from pulmonary barotrauma, in which the diver suffers massive ingress of breathing gas directly into his circulation via over-pressured lungs. This syndrome is instantly fatal (Figure 15.2), is always a complication of ascent, and is usually associated with diver panic. Resuscitation from such a catastrophe is not possible, and it will not be considered further here.

Pathogenesis of the decompression illnesses

The tissues of a diver's body are exposed to an increased partial pressure of nitrogen. Depending (among other things) upon the magnitude of this partial pressure (depth of the dive), the time the tissues are exposed to this gas at increased pressure (the "bottom time" of the dive), the solubility of the gas in the different body tissues (central nervous tissue can slowly take up large amounts of inert gas, such as nitrogen), and the rapidity with which the partial pressure is lowered following exposure (the speed of ascent), this nitrogen may come out of solution within the tissues, instead of remaining in solution and being harmlessly unloaded from the blood via the lungs. If this happens, bubbles will form and grow in the tissues, causing pressure on nerve cells, muscles, and even blocking the flow of blood through the lungs in serious cases. Their presence also initiates chemical changes within blood vessels and the blood itself, which may potentiate vessel obstruction from thrombi. All this will produce signs and symptoms of DCI. Bubbles of nitrogen actually in the blood are called gas emboli, and their contact with cerebral vascular endothelium, even when transitory, can produce endothelial damage, white cell accumulation, and subsequent vascular luminal obstruction, resulting in delayed neurological sequelae to CAGE.

It is thought that bubbles first form in tissues, then in veins. It is possible for bubbles to form *de novo* in arteries, but most arterial bubbles get there either from veins by passing through (or bypassing) the lungs, or from pulmonary barotrauma.

Should large quantities of gas bubbles gain access to a diver's arterial blood stream, by whatever means, these bubbles will distribute themselves in the larger arteries according to their buoyancy, and not according to the direction of blood flow. As all divers ascend in the head up position, these gas bubbles will travel straight to the brain, producing cerebral arterial gas embolism (CAGE). The only way to prevent this from continuing is to lay the diver down with the head at, or a little below the level of the heart, and keep him there. This has important connotations for first aiders rendering assistance to affected divers.

Signs and symptoms of decompression illness

Table 15.1 shows the presently accepted classification of DCI on a clinical basis.

A diver with DCI may have any of a very wide range of signs and symptoms, partly because the disease particularly affects the central nervous system. However, the commonest symptoms are:

Less severe

- pain in a limb, joint or trunk
- paraesthesia
- headache
- excessive lassitude
- skin itch

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More severe

- actual numbness
- unsteadiness on the feet
- muscle weakness
- visual disturbance

Most severe

- paraparesis or paraplegia
- hemiparesis or hemiplegia
- acute respiratory difficulty
- acute urinary retention
- impaired consciousness

Divers most severely affected by the CAGE form of DCI will develop impaired consciousness immediately upon surfacing, or within minutes afterwards. Divers suffering from the less fulminating forms of DCI will usually develop their first DCI symptoms after 2–6 hours following the dive; some may take even longer.

First aid principles for DCI

In divers with conscious state affected, normal basic rules of resuscitation (the St John Ambulance DRABC action plan – Danger, Response, Airway, Breathing, Circulation) always apply, and take priority.

Any diver who has, or who has had, their conscious state affected must be placed in a horizontal position immediately, and kept there as far as possible. This will prevent further CAGE. As for any other casualty, the lateral, stable side, recovery, or coma position will be necessary for the spontaneously breathing but unconscious diver. There is no evidence to date to indicate that one side is safer than the other.

The administration of 100 per cent oxygen is important (Figure 15.3). This will accelerate the reduction in bubble size, as well as relieving any hypoxia more efficiently. Anything less than 100 per cent oxygen will contain nitrogen, which will oppose bubble resolution.

Nitrous oxide containing gases, for example Entonox, must not be administered to diving casualties with proven or suspected decompression illness. The nitrous oxide will enter any bubbles faster than nitrogen exits, cause them to enlarge, and prolong their existence.

As fluid loss from the blood stream is a consequence of decompression illness, the administration of fluids – orally in fully conscious casualties, or intravenously in the presence of impaired consciousness – will have a beneficial effect.

Affected divers (and well divers who have dived within the preceding 24 hours) should not be taken to altitude, where the atmospheric pressure is below that at sea-level. Maximum permissible altitude is 300 metres. A reduction in ambient (atmospheric) pressure will increase bubble size and worsen symptoms.

Other effects of pulmonary barotrauma (Interstitial emphysema and complications; pneumothorax and complications)

Lung overpressure producing alveolar tearing does not always result in arterial gas embolism. The gas may track along the lung airways and blood vessels to the mediastinum (pneumomediastinum), thence up into the neck and under the skin of the neck and shoulders (surgical emphysema), or even down into the abdomen, behind the bowel (uncommon). This may produce central chest discomfort, swelling of the neck, palpable gas under the skin (crepitus), and a voice change. Such effects, although alarming, will usually produce no serious harm. The diver should be rested, reassured, and administered 100 per cent oxygen, to reduce the volume of the air in the tissues. Medical referral is essential.

Pulmonary barotrauma may also result in a pneumothorax (air in the pleural cavity). This will

Table 15.1 A clinical descriptive classification of the decompression illnesses. (Note that no attempt is made to distinguish cerebral arterial gas embolism from decompression sickness. Thus any "evolution" term in combination with any "organ system" term is possible.)

Evolution	Organ system
progressive	neurological
static	cutaneous (skin)
spontaneous	musculo-skeletal
relapsing	pulmonary (lung)

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usually produce chest pain on the affected side, pain on inspiration, and possibly some breathlessness. Again, rest and 100 per cent oxygen is the best first aid, while the DES/DAN emergency distressed diver telephone hotline is contacted.

If a pneumothorax develops in a diver who is still underwater, as he ascends the pneumothorax will expand, and can result in his heart and great chest vessels being pushed across to the other side, inside his chest (Figure 15.4). This is called a tension pneumo-thorax, and is dangerous. It can quickly cause hypotension and shock. Other clinical signs that may be present are chest pain, breathing difficulty, shift of the trachea in the neck away from the midline towards the unaffected side, and distension of the large veins in the patient's neck. Immediate administration of 100 per cent oxygen will help reduce the volume of the trapped air, but urgent DES/DAN telephone consultation is advised.

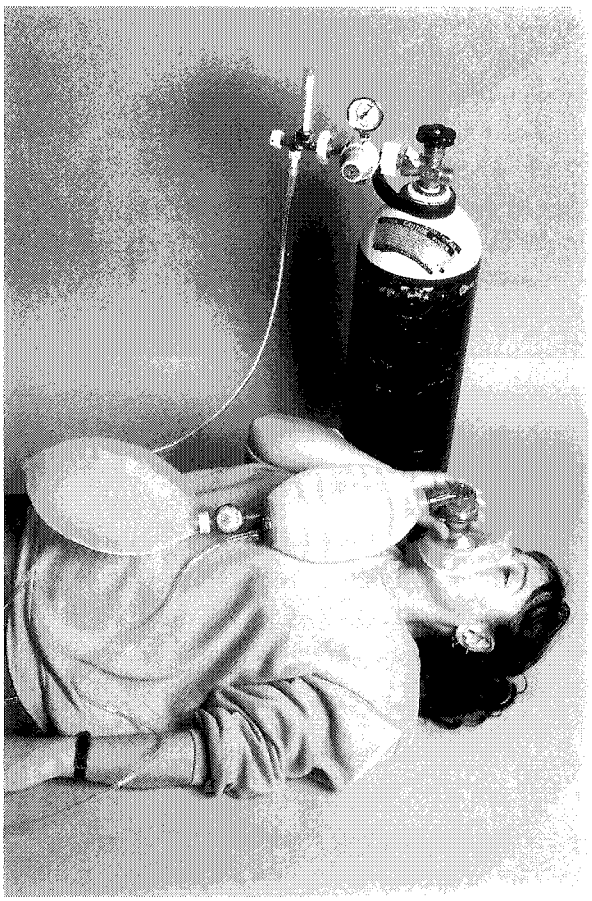


Figure 15.3 *An apparatus for the administration of 100 per cent oxygen (FIO_2 = 1.0), using a continuous flow oxygen catheter from an oxygen source (here a cylinder). The presence of the second, collapsible reservoir bag is essential to compensate for the peak inspiratory flow rate of the patient. A fully conscious, co-operative diver can self-administer his or her own 100 per cent oxygen, following instruction.*

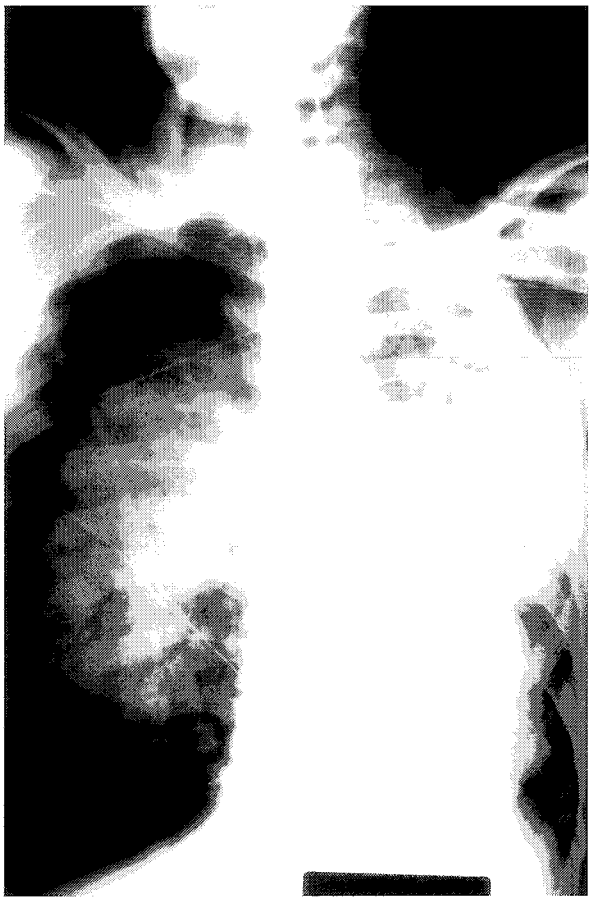


Figure 15.4 *Erect plain chest radiograph of a patient (not a diver) with a right tension pneumothorax. Note the partially collapsed right lung, and the displacement of the mediastinum to the left. This is a potentially dangerous situation for a diving casualty. (Photograph courtesy of Dr P.D. Thomas.)*

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Divers who develop surgical emphysema and/or pneumothorax seldom have coexisting arterial gas embolism, and *vice versa*. The reasons for this are not completely clear.

3 Inner ear barotrauma

This results from rupture of the round or oval window, allowing the leak of inner ear fluids into the middle ear cavity, with the attendant production of hearing loss, vertigo (sometimes quite severe), tinnitus, and often nausea and vomiting. These symptoms will persist as long as inner ear fluid loss occurs.

First aid principles

Cessation of diving and of attempts at equalisation are essential. The diver should be rested in a head up recumbent position to reduce inner ear fluid loss. Avoid coughing and straining. Oral fluids, if the casualty can accept them (do not induce vomiting) may help, while rescuers contact a diver telephone hotline for advice. Urgent medical referral will be necessary to salvage hearing in the affected ear, and may involve surgery.

4 Contaminated breathing gas

This is usually carbon monoxide poisoning. It may be caused by poorly serviced air compressors, which allow partially combusted fuel within the engine to enter the cylinder (SCUBA) or air hose (HOOKAH) of the diver. (Carbon monoxide is always a product of incomplete combustion of organic material.) It may also occur by the entrainment of exhaust fumes from an internal combustion engine into the clean air intake of a compressor filling SCUBA cylinders. This is particularly likely when the exhaust is upwind of the clean air intake (eg following a change in wind direction on a dive boat at sea).

Carbon monoxide's precise actions in the human body are still under study, but the gas is tasteless, colourless, non-irritant, and probably odourless, and a diver will, therefore, have no idea of its presence in a contaminated breathing gas mixture. Even tiny amounts of carbon monoxide in the breathing gas, under the increased partial pressure conditions of diving (see above), may have dangerous effects. Classically, the diver is affected upon ascent, because the falling oxygen partial pressure permits the carbon monoxide already absorbed to exert greater effect. When this relatively uncommon diving mishap does occur, it is not uncommon for more than one diver to be affected. This is because SCUBA cylinders are often filled in "banks" or groups.

Carbon monoxide affects mainly the human nervous system, causing headache, impaired intellectual function, and in serious cases, unconsciousness. Thus the differential diagnosis between contaminated breathing air and DCI can be very difficult for the first aider. Fortunately, that diagnostic distinction is not essential, as the first aid management of carbon monoxide poisoning is, for practical purposes, identical to that for DCI.

First aid principles for contaminated breathing gas

- Surface all divers, and have them breathe atmospheric air.
- Remove the casualty from the water, and resuscitate as indicated (DRABC).
- Place the casualty in the horizontal position (just to be safe, in case the casualty has CAGE).
- Administer 100 per cent oxygen as soon as possible to affected persons. This will more rapidly remove the carbon monoxide from each casualty's body.
- Telephone the nearest national DES/DAN Distressed Diver hot-line.
- If possible, retain the suspect SCUBA cylinder for later gas analysis.

GENERAL REMARKS ON FIRST AID FOR DIVERS

First aid for divers can be approached in the same basic way as first aid for any other casualty. However, the value of a horizontal posture, 100 per cent oxygen, and avoidance of both altitude and the use of analgesic gases such as Entonox are points of difference of which rescuers should be aware.

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Diving equipment worn by the casualty can be removed by persons unfamiliar with such gear, once the casualty is removed from the water. This equipment removal should not be permitted to compromise or interrupt resuscitation at any stage.

In-water expired air resuscitation (EAR) takes the greatest skill and practice, and the presence of positive buoyancy of the casualty. Mouth-to-mask EAR is preferred. It is virtually impossible, even for the skilled, in rough weather conditions. In-water external cardiac compression is a physical impossibility. Such manoeuvres should never be allowed to delay the prompt removal of the diving casualty from the water into a boat or onto dry land, where effective assessment and resuscitation can commence.

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CHAPTER SIXTEEN

DROWNING

John Pearn

Care of the apparently drowned can involve first aiders, paramedics and doctors unexpectedly at any time, and in a variety of situations ranging from the local sailing club, a holiday beach across the world, the neighbours' swimming pool, the local ambulance station or the local surgery. Management of the near-drowned involves the pragmatic issues of rescue, extraction from the water, rescue-site resuscitation, transport, emergency room management, intensive care therapy, and the making of realistic predictions about the prognosis in survivors.

A central theme, inescapable in near-drowning, is the fact that no two situations are the same. Variations in the type of water inhaled, questions of alcohol or drugs, attempted suicide, immersion hypothermia, the possibility of epilepsy, how soon after rescue resuscitation was started, whether CPR was skilled or not – all may summate to produce an infinitely variable clinical picture. The most important variable of all – the duration of hypoxia – is usually unknown and unknowable.

Because of the potential of these confounding variables, it is essential to have a clear picture of the "hypoxic march" which leads to brain death, and then to somatic death (Table 16.1). Management of the near-drowned victim depends on an understanding of this pathophysiology in the immersion victim.

THE SUBMERGED VICTIM

Although death comes quickly and in a very unsubtle way, there are several ways in which subjects die while in the water. Brain death is the end result, but the first link in the chain may be cerebral hypoxia, carbon dioxide narcosis, laryngeal spasm, reflex lung changes, or vagal cardiac inhibition. In a pedantic sense, some define true drowning as death caused by the aspiration of fluid into the air passages. Death caused by laryngeal spasm, reflex pulmonary changes, or vagal cardiac effects might be more correctly termed "death from submersion", rather than from drowning. In fresh-water drowning, it has been known for 30 years that large volumes of water can pass across the alveolar–capillary interface.

The volumes of water inhaled by a submerged victim may be relatively small. Depth is no guide; children, intoxicated and epileptic subjects, and those with cervical and head injuries can drown in very shallow water. In some cases, the submerged victim inhales large volumes of water; however, there is no evidence that the increase in blood volume is beyond the capacity of the heart and kidney to compensate for this potential fluid overload. The heart compensates for this with the basic reflexes of Starling's Law and the Bainbridge Reflex.

All forensic pathologists subscribe to the theory of "dry drowning" in humans. This is a condition of fatal cerebral hypoxia brought about by inadequate ventilation which is not caused by mechanical blockage of the bronchi by the drowning fluid, usually water, but rather by laryngeal spasm, or deleterious lung reflexes, or mechanical blockage of the airway by mucus and froth. The frequency of dry drowning in all drowning episodes is variously estimated to be from 10 per cent to

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Table 16.1 The hypoxic march which constitutes the pathophysiology of drowning

Sequence	Pathophysiology	Notes
1 Involuntary submersion	Voluntary apnoea, tachycardia, hypertension, hypoxia, hypercarbia, acidosis.	Diving reflex occurs in infants and toddlers (blood shunting, bradycardia). Voluntary apnoea is biphasic.
2 Break-point involuntary inspiration	Arterial hypoxaemia, tachycardia, tissue hypoxia, tissue acidosis.	Breakpoint determined by both hypercarbic and hypoxic drives which are synergistic.
3 Water enters lungs	Increased peripheral airway resistance.	Laryngeal spasm occurs in 10–15 per cent.
	Reflex pulmonary vessel vasoconstriction.	Pulmonary hypertension and shunts develop.
	Decreased lung compliance.	The "stiff-lung syndrome" leads to resuscitation difficulties.
	Fluid shifts occur across the alveolar membrane.	Water flux occurs in the direction of alveolus to blood in both salt- and fresh-water drownings.
	Surfactant diminished.	By loss (salt water) or denaturation (fresh water).
4 Decompensation	Gasping occurs with further inhalation. Swallowing occurs with secondary emesis. Secondary apnoea. Consciousness lost.	Froth and foam production in airways. Water flux continues.
5 Neuronal	EEG becomes flat. Blood–brain barrier breaks down.	Different neurones show selective vulnerability.
6 Cardiac dysfunction	Bradycardia, arrhythmias, asystole.	Temperature-modified.
7 Brain death		Confounding influence of drugs, alcohol, and hypothermia.
8 Somatic death		Occurs within 1–60 minutes after submersion, with a median of 3–10 minutes. Dependent on age, water temperature, and degree of tissue hypoxia.

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20 per cent. There is good experimental evidence to support this phenomenon.

When dry drowning occurs, it appears that a small amount of water enters the larynx or trachea, and sudden laryngeal spasm (as a vagal reflex) occurs. It is followed by an immediate outpouring of thick mucus. Foam and froth develop and, in some cases, a physical mucus plug may form. It seems likely that, when the spasm relaxes shortly before death, water may be prevented from entering the trachea and lungs by the foam and froth which act as a physical barrier, and by bronchiolar spasm. Whether or not such lung reflexes are facilitated by a raised blood alcohol level is unknown, but it has been suggested as one explanation for the greater number of dry drowning cases in adults, compared with that reported in series of child drownings.

THE DROWNING EPISODE

When a sober victim first submerges, there is an initial period of voluntary apnoea. From personal observations in infants and toddlers in so-called "drown-proofing" classes (well-meant but in my view misguided), one is struck by the failure of an infant to struggle as the head goes below the surface. Usually the child simply holds his breath; makes automatic, but ineffective, paddling type movements; and calmly sinks to the bottom. It is almost certain that the same pattern is followed in the accidental drowning of children.

At the age of peak risk of drowning (1–2 years), it is apparent that the atavistic diving reflex still operates to some degree. Its presence can still be demonstrated in adult human volunteers. Before consciousness is lost, it may be predicted that, within seconds, the submerged victim manifests bradycardia and shunting of blood from the cutaneous and splanchnic vascular beds to the cerebral and coronary circulations. Blood pressure promptly starts to rise. These reflex changes are independent of baroreceptor and chemoreceptor inputs, and depend both on sensory afferents in the trigeminal nerve which supplies the face, and on reflex or voluntary inhibition of the medullary respiratory centres. Variation in water temperature above 20°C (the most common situation in the case of real life drownings) does not influence the brain-protecting diving reflex, but progressively lower temperatures augment it. I have speculated that the brain-sparing effects of cold water may be mediated through an augmented diving reflex as much as through physical chilling of the body core.

It is known that the diving reflex is more intense in the frightened or startled mammal, compared to that observed in an animal which dives or submerges voluntarily. If this is true in humans, a case can be made on physiological grounds against the practice of so-called "drown-proofing" of young infants, which is condemned by most paediatric bodies throughout the world.

The breath can be held voluntarily until the breaking point is reached. The period of breath-holding (and the onset of the breaking point) is determined both by hypercarbic and by hypoxic drives. The influence of high carbon dioxide levels and low oxygen concentrations is mutually synergistic; the breaking point occurs at $PaCO_2$ levels below 55 millimetres of mercury (mm Hg) when there is associated hypoxia.

When the submerged victim reaches the breakpoint, and an inspiration has to be made, tachycardia and arterial hypoxaemia have already developed. Gasping follows; glottal spasm may then occur. Besides gasping, large quantities of water are usually swallowed, and, even before consciousness is lost, vomiting with aspiration of gastric contents is likely to occur.

In older children and adults, the dangerous practice of hyperventilation before underwater endurance games or dives can change the dynamics of the breath-holding breakpoint. If the hypercarbic drive to breathe is lost, unconsciousness from cerebral hypoxia may occur before any breakpoint is reached. Under these circumstances, breathing (with fluid aspiration) may recommence after the submerged person loses consciousness, with disastrous consequences.

Within seconds of the first submerged breath being taken, a phase of secondary apnoea occurs. This is

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followed by involuntary gasping under water which may continue for several minutes before final respiratory arrest occurs. Arrhythmias are inevitable in all cases of near-drowning and, in the absence of ventilation, lead within minutes to brain death if rescue and resuscitation are not effected. The spleens of drowned victims are relatively bloodless; hypoxia in the submerged victim causes reflex constriction of splenic vessels.

The duration of hypoxia to cause death is age-dependent. After extensive interviews with parents, neighbours and rescuers in Australian studies (using a "bracket method"), it was suggested that children immersed for 3 minutes or less are likely to survive; and that the necessarily crude estimates of immersion time for human fatalities lie between 3 and 10 minutes, provided that the immersion occurs in warm water not below 15–20°C. Long immersion times (5–20 minutes) are still occasionally compatible with survival and the preservation of normal intellect, even in warmer water. Very long immersion times (5–40 minutes) are certainly compatible both with survival and with restoration of normal neurological function in children who have been immersed in very cold water (0–15°C). In animals there is evidence that survival times are marginally longer after immersion in salt water.

Consciousness is always lost within 3 minutes of involuntary submersion. Even in swimming and diving animals, such as mink and beaver, consciousness is lost within 2.5 minutes, and the EEG becomes flat within 4.5 minutes. In practice, when the human victim loses consciousness, this is almost always because of cerebral hypoxia. In some boating accidents, in accidents involving falls through ice, and in cold-water accidents involving victims who can swim, immersion hypothermia can occur in the absence of primary drowning asphyxia. Consciousness is lost when the core temperature falls below 34°C.

INHALED FLUID

The salinity of sea water varies, but typically sea water contains 34.48 grams per kilogram (g/kg) (3.5 per cent) of dissolved salts, of which 29.54 g/kg (2.9 per cent) is sodium chloride. Fresh water contains variable amounts of organic material, dissolved salts, and free and nascent gases.

Most victims who drown do so in the sea, in chlorinated fresh-water pools, or in bathtubs with variable concentrations of soap. The chemical effects of chlorine and soap in fresh water are currently believed to be of no consequence in the pathophysiology of lung syndromes in survivors; laboratory studies have shown that the effects of chlorinated fresh water and of simple fresh water on the surface tension of lung surfactant are similar. However, compared with the effects observed after salt-water aspiration, both types of fresh water considerably elevate the minimum surface tension of tracheal and lung surfactants. Sea water, and water with sodium and chloride concentrations which are approximately iso-osmolar with plasma, do not denature pulmonary surfactant, but may dilute it or wash it out.

In "wet" drownings, there is evidence that, even if large amounts of fluid are aspirated as a single event, spontaneous breathing (albeit with a grossly altered ventilatory pattern) will continue. One implication of this is that, in a typical childhood drowning (say, that of a 13 kilogram, 2-year-old toddler), the amount of inhaled water needed to drown the child would probably exceed 0.3 litres, in a true "wet" drowning.

THE LUNGS

The "march of events" in the lungs of a drowning victim is summarised as follows.

1 Increased peripheral airway resistance.
2 Variable degrees of laryngeal spasm.
3 Reflex pulmonary vessel vasoconstriction, leading to pulmonary hypertension.
4 Decreased lung compliance.
5 Ventilation–perfusion ratios fall.
6 Fluid shifts occur across the alveolar membrane.
7 Surfactant loss occurs (salt water), or its properties alter (fresh water).
8 Foam and froth production.
9 Anatomical changes in alveolar epithelial cells.

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When fluid is inhaled, vagal efferents cause obstruction of the peripheral airways. Fresh water inhalation is particularly effective as a trigger for this reflex; osmolality and not volume is the more important trigger. Even when very small amounts of fresh water are inhaled, aspiration is followed by pulmonary vasoconstriction, with the immediate development of pulmonary hypertension induced by a parasympathetic reflex. With the aspiration of larger volumes of water, intrapulmonary reflexes lead to the blood perfusion of non-ventilated areas. This phenomenon (combined with the loss or inactivation of surfactant, and compounded by alveolar collapse) causes a significant reduction in mechanical compliance. In normal lungs, about 5–18 per cent of blood perfuses non-ventilated areas; within minutes of the aspiration of even small amounts of fresh water this rises to 75 per cent. Clinical studies in humans have shown that, even in near-drowned children who rapidly (within minutes) become virtually "normal" from the clinical point of view, the redistribution of blood perfusion takes at least several days to revert fully to pre-accident levels.

Inhaled water moves across the alveolar epithelium, through a basement membrane, and finally through the endothelial capillary lining into the capillary lumen before haemodilution occurs. This flux causes rapid and severe distortion of pulmonary ultrastructure. Histological examination shows damage both to type 1 and to type 2 epithelial cells (pneumocytes). The latter cells are believed to produce surfactant. Endothelial changes consist of microvesicle formation, swelling, detachment from the basement membrane and disruption of cells. It is probable that this phenomenon is intensified if alcohol and other gastric contents are inhaled.

Denaturisation of surfactant can continue even after an apparently successful initial resuscitation. This phenomenon is known as "secondary drowning", and is potentially fatal if not recognised and treated. Clinically evident, secondary drowning occurs in about 5 per cent of survivors, but it is probable that some degree of hyaline-membrane formation occurs in most cases of near-drowning. An outpouring of a proteinaceous exudate which is relatively cell-free occurs and may take several days to resolve.

Mechanical blockage of the bronchial tree is produced by a combination of bronchial spasm, changes in the elastic properties of lung tissue, and an increase in non-elastic resistance caused by physical blockage of the airways by fluid and foam.

THE PROGRESS OF HYPOXIA

The cause of death in an acute drowning episode is irreversible cerebral anoxia. Brain death in the drowning victim may occur before or after cardiac asystole. In about 5 per cent of survivors who persist in a totally "vegetable" state, it may occur without any long-term evidence of coronary hypoxia.

After consciousness is lost, there remains a short period of reversibility of the altered neuronal metabolism, before intracellular hypoxia and acidosis cause permanent damage. Not all neurones are equally vulnerable, and those associated with more subtle forms of intellectual activity are affected first. In survivors of childhood fresh-water immersion accidents, I have observed that skills which require visual–motor co-ordination are particularly affected. The EEG becomes flat during this stage, but is reversible. Oxygen depletion arrests neuronal activity. It is probable that the order of deleterious influence is hypoxia, acidosis, and hypercarbia.

The blood–brain barrier breaks down and both fluid and macromolecules pass into brain parenchyma; functional, if not anatomical dissolution of vascular and cell-wall integrity leads to intracellular and intercellular increases in osmotic pressure. If the patient survives to be resuscitated, brain oedema develops. It is thought that cerebral oedema caused by ischaemia is harmful only in that it may cause swelling of the hemispheres sufficient to result in mass effect and herniation. There is no evidence to date to indicate that oedema *per se* increases the extent of neuronal death. In survivors, the speed with which significant cerebral oedema develops remains uncertain, but this phenomenon has been

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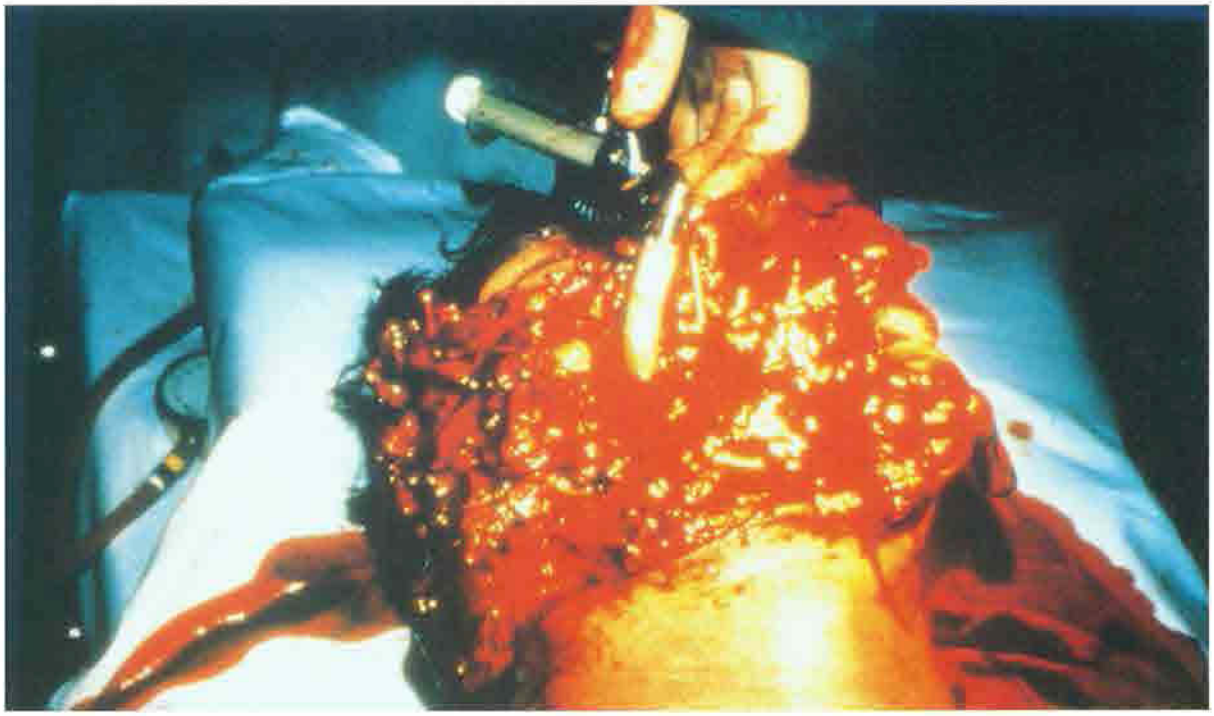


Plate 1 *Gunshot wound to the face. An example of extreme difficulty for the first aider in the all-important task of maintaining an airway.*



Plate 2 *Flame-caused burns.*

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Plate 3 *Burns caused by spark igniting firework in pocket.*



Plate 4 *A diver's eyes showing the congested, haemorrhagic, "conjunctivitis-like" appearance following a mask squeeze. (Photograph courtesy of Dr William Douglas and Dr Robert Thomas.)*

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reported in several recent studies as not being a real problem in the first 24 hours after rescue.

The final stage is that of permanent cerebral death. In cases of true brain death there is no recovery, although patients supported by mechanical ventilation can exist in a state of true brain death for months, or longer.

FIRST AID

Cardiopulmonary resuscitation (CPR) is standard whether it is undertaken at the rescue site, during transport, or in the emergency room (casualty department) while intubation, monitors, defibrillators, ventilators and other hardware are being assembled. The airway must be cleared, initial breaths given, carotid pulse checked and if it is absent combined expired air resuscitation (EAR) and external cardiac compression (ECC) administered. In over half the cases of immersion accidents the heart can be restarted by mechanical stimulation and, provided that submersion has not occurred for extreme lengths of time (eg longer than 10 minutes), it is possible to restore circulation. Even in victims who are truly dead when extracted from the water, correctly administered CPR should cause some lessening of pupillary dilation, and the body should become pink to some degree.

Approximately 80 per cent of victims who do survive will make their first respiratory gasp within 5 minutes of rescue. Many do so within the first minute after extraction from the water.

The mechanics of EAR may be difficult because of the reduced compliance of the water-containing, mechanically altered lungs. The pressure used to inflate the lungs of the near-drowned subject has to be greater than that used for victims who are apneic from other causes. The reduced pulmonary compliance follows tracheal aspiration of both salt and fresh water. The usual danger of over-inflation during EAR remains, however, and interstitial emphysema and air embolism can be produced relatively easily by over-inflation.

It is very important that an unconscious but breathing near-drowned victim be managed (both at the rescue site and during transport to hospital) in a stable side position (ie the semi-prone position with the head to one side). This general rule of first aid is crucial in the case of a near-drowned child, because regurgitation of large amounts of water from the stomach is very common.

Of all patients pulled unconscious from the water who do survive, less than 5 per cent will be brain-damaged. All victims extracted unconscious from the water must be given the benefit of skilled resuscitation in spite of the apparent hopelessness of the situation or fears about possible prognosis.

A small but unknown percentage of children who are unconscious and apnoeic when rescued from the water nevertheless make a successful recovery in spite of the absence of skilful resuscitation. However, poor technical CPR, and effective CPR not commenced within at least 10 minutes after rescue, are two unfavorable prognostic factors. There is usually no need to be concerned about re-warming at the rescue site when victims are rescued from very cold water (less than 15°C); ventricular fibrillation due to hypothermia is not a problem until the body temperature falls to approximately 28°C. It is known from both experimental and clinical evidence that, if technically good resuscitation is applied, the hypothermic brain can withstand long periods of hypoxia, in extreme cases up to one hour. There is good clinical evidence that the hypoxic brain of the near-drowned victim suffers less permanent damage if the body core temperature is reduced until adequate ventilation in the emergency room can be re-established.

In practice, in most parts of the world there is usually no need to be concerned with re-warming the victim in the first aid situation. However, if the temperature is below 28°C, ventricular fibrillation and asystole will occur, and attempts to re-start the heart by CPR in the field will be in vain. Under these circumstances, CPR must be continued indefinitely until the victim can be re-warmed under controlled conditions in an intensive care unit. It is essential that a diagnosis of death is not made in the field, if the victim is cold. Prolonged

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submersion (even up to 40 or 60 minutes) can be compatible with complete recovery even when complete cardiopulmonary arrest is present at rescue, in those cases of submersion hypothermia in which the core temperature falls below 30°C. Involuntary aspiration or ingestion of cold water has the same effect as an intravenous injection of cold water. Core chilling is brain sparing. Brain temperatures fall at the rate of up to 1°C per minute during agonal respirations of drowning animals submerged in fresh water at a temperature range from 20°C to 22°C.

How long should one persist with CPR? If the victim has a core temperature above 33°C, and drugs or alcohol are not involved, I have yet to see a survivor without significant brain damage who did not make their first respiratory gasp within 40 minutes of rescue. For this reason, provided that the body is not chilled, if asystole persists after one hour, I feel that persistence in field CPR is fruitless. None the less, as already indicated, the decision to stop CPR should be made in a properly equipped emergency room.

Often the first sign of successful CPR is a convulsive abdominal–diaphragmatic heave with a flood of vomitus or swallowed water. The majority of patients who are going to survive manifest some returning respiratory activity (usually incoordinate gasping) quickly. Indeed, the majority (at least in the case of children) become conscious within 20 minutes or so, and many are able to talk and move by this time. Because of the danger of secondary drowning, it is essential that all immersion victims be admitted to hospital for observation at least overnight. This post-rescue deterioration may occur because of surfactant loss or denaturation by the inhaled liquid, or because of alveolar cell damage due to osmotic or anoxic causes. After inhalation of relatively small quantities of water (2.5 millilitres per kilogram), within minutes the intrapulmonary shunt is known to increase from its normal level of perhaps 10 per cent to levels which may be as high as 75 per cent. Even in victims who are conscious, alert, and clinically normal after near-drowning and successful CPR, this shunt takes several days at least to revert to pre-immersion levels.

In some circumstances (eg surfing or diving into creeks) there may be a significant probability of spinal injury, and the near-drowning may in fact be secondary to this. The possibility of cervical spine fracture or dislocation is a very important consideration in the first aid management of the near-drowned.

All first aiders, paramedics and doctors have a responsibility to promote the teaching of CPR, and example is the first step. In the United States for the last decade, 66 per cent of general practitioners stated, in reply to a questionnaire, that they were not required to take a practical course in CPR at their medical schools. There is good evidence that trained first aiders can turn potential fatalities into survivors. A trained person is also more likely to start resuscitation earlier, giving the victim a better chance of absolute survival and less residual neurologic damage. In a retrospective analysis of childhood survivors of near-drowning, undertaken as part of the Brisbane Drowning Study, the survival rate in the presence of a trained resuscitator was 70 per cent. In the absence of a trained resuscitator the survival rate was only 40 per cent. It is inescapable that this difference is not simply due to chance, and that trained resuscitators do in fact turn potential fatalities into survivors.

CLINICAL HISTORY

In the emergency room or casualty department, while the victim is being assessed and life support instituted and maintained, it is important that as complete a history as possible is obtained. The rescuer or first aider can help significantly in this context. Key factors of the history include: (1) documented or estimated time of accident; (2) type of water (salt, fresh) and degree of contamination; (3) estimated temperature of water; (4) estimated duration of the immersion or estimated bracket of time during which the victim is believed to have been immersed; (5) details of the rescue; (6) documentation of resuscitation attempts, time before CPR was attempted after extraction from the water, and the presence of vomiting during resuscitation; (7) time to the first spontaneous gasp after apnoea; (8) details of transport to hospital and maintenance of CPR (or

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otherwise) during transport; (9) specific features surrounding the immersion incident.

An estimate of the time when the submersion occurred is obviously important, and questions about the water osmolality are now believed to be of relative inconsequence. A scientific debate that started some 30 years ago has persisted in clinical circles about whether or not water osmolality is of relevance in the practical context of decisions concerning emergency care of the near-drowned. Modell's work in the 1960s indicated that if the individual survived near-drowning, the electrolyte changes were likely to be transient and to revert spontaneously to normal. This work and a number of subsequent case series have shown that initial management of the critically ill survivor can discount whether salt or fresh water was involved. Management is governed by clinical assessment and by the results of tests performed on the victim after arrival at the emergency room and initial CPR.

In a small percentage of victims there will be other coincidental illnesses (epilepsy, asthma, etc.) It is important that the admission history documents these illnesses, as subsequent management may be influenced by them. Alerting emergency room physicians to such coincidental diseases may assist them to anticipate complications. The history should also indicate whether medication or alcohol is related to the drowning incident. The history should also state whether or not physical injuries have been sustained. Although uncommon, injuries nevertheless are reported in most case series, and it is essential when the near-drowning is precipitated by a closed head injury (eg from a fall at the edge of a swimming pool) that the medical team be aware of this complication. The possibility of a cervical spine dislocation caused by a diving accident is included in this category.

The admission history should also contain details of the health of other members of the family because they can sometimes provide information about the current drowning accident. The usual social history is also particularly important in this type of case, because certain families are more likely to be represented in such case series. The drowning accident itself is caused by one or more acute triggers, which are superimposed on a vulnerable baseline predisposition. It is known that accidents in general can be attributed to a lowering of a particular threshold because of identifiable intra-familial factors such as coincidental illness or marital discord. Finally, and especially in the case of child victims, it is essential to document the prior developmental history of the child. There is good evidence to indicate that, at least with respect to toddlers (who form the major group of drowning victims in children), it is particularly the bright and adventurous one- and two-year-olds who get themselves into potential drowning situations. If questions of subsequent neurologic damage arise, it is desirable to have documented the pre-accident developmental history as a baseline.

TRIAGE

Primary triage has long classified near-drowned victims into three groups (popularised in 1980, by Conn and Modell for prognostic purposes) – those who are apparently normal when they reach a treatment facility ("A" for "awake"), those who are conscious but obtunded ("B" for "blunted") in the Modell–Conn classification, and those who are comatose ("C"). Those in groups A and B require a chest X-ray, and those in B may also require baseline biochemical and respiratory function studies. Careful clinical monitoring of these two groups should obviate the danger of over-investigation and poly-biochemistry, as the great majority will make a successful recovery with good nursing care.

NOTE ON MEDICAL MANAGEMENT

Mechanical ventilation is required when near-drowned survivors cannot maintain their arterial oxygen and carbon dioxide levels within normal limits by their own efforts. A PaO_2 of less than 60 mm Hg (8.0 kPa) breathing air, and a $PaCO_2$ tension of 56 mm Hg (7.5 kPa) is an indication for mechanical ventilation. Most

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intensivists try to keep the PaO_2 at 100–200 mm Hg, on empirical grounds in the belief that blood-to-cell diffusion will be facilitated. Ventilator mode should be CPAP/PEEP (constant-positive airway pressure/positive end-expiratory pressure). Although virtually all near-drowned comatose children will require intubation and PEEP, a percentage of victims (especially those who are older) will tolerate a mask and will benefit from CPAP rather than the simple breathing of oxygen-enriched air in a face-box, mask or tent. The discovery and use of PEEP has been hailed as one of the significant therapeutic milestones in the history of medicine. Its use in the near-drowned has now been well established for well over a decade, and is not controversial. Weaning to IPPV (intermittent positive pressure ventilation) with ZEEP (zero end-expiratory pressure) should occur as soon as possible to reduce the risk of pneumothorax and tracheomalacia.

COMPLICATIONS IN THE POST-RESCUE PERIOD

Management includes giving a prognosis to parents and relatives of the near-drowned. If the victim has made a first respiratory gasp within 30 minutes of rescue, and is continuing to improve (vital signs, level of consciousness

Table 16.2 Ten prognostic guides, in the early phase of management of the near-drowned

Factor		Notes
1	Site of drowning	Fresh-water risks worst; surf best.
2	Water temperature	Cold water best.
3	Immersion time	Usually unknowable; estimate by "bracket method".
4	Time to first spontaneous respiratory gasp	Within 15–30 minutes after rescue, less than 10 per cent of survivors have mental retardation or spastic quadriplegia. Within 60–120 minutes or later, 50–80 per cent of survivors will show serious neurological sequelae.
5	Whether CPR administered, and whether given by a trained operator	Some 30 per cent of potential fatalities turned into survivors by skilled rescue-site CPR.
6	The presence of coma on arrival in intensive care unit	Usually it takes 1–2 hours post-submersion, before the victim is in an intensive care unit, even in the best retrieval systems.
7	The presence of fixed dilated pupils on arrival in intensive care unit	An ominous sign.
8	Intensive care therapy, especially PEEP	Role of barbiturate rescue, hypothermia and ICP control uncertain.
9	The first measured arterial pH	Usually not undertaken in practice prior to 1–3 hours post-rescue. An arterial pH below 7.00 is a bad prognostic sign.
10	The first measured arterial oxygen tension	Usually not measured prior to 1–3 hours post-rescue. A PaO_2 below 60 mm Hg (8.0 kPa) in air is a bad sign.

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etc.) over succeeding hours, the prognosis is good. If the victim is a child and the first gasp was made within 20 minutes of extraction from the water, the prognosis is excellent. Approximately 50 per cent of children who are pulled apparently lifeless from fresh water will survive and some 70 per cent who are rescued from the surf will do so. Of all children who are rescued and who survive, some 3 per cent only will exist in a persistent vegetative state. Another 2 per cent manifest chronic hypertonic quadriplegia and other neurological complications such as extrapyramidal signs and peripheral neuropathies. Of all child survivors who appear to function normally, approximately one-third have wide subscale discrepancies on formal psychometric testing (minimal cerebral dysfunction). Growth and hearing are not affected by this type of acute hypoxic insult. It is important to appreciate that sequential recovery over the ensuing 4–6 months, even in the severely affected victim, is possible.

In the case of child victims who are still apnoeic with fixed dilated pupils when the intensive care unit is reached (usually 1–2 hours post-hypoxia) the prognosis is not as good. The further survival of this group of selected cases may be as low as 50 per cent. Fixed dilated pupils in the intensive care unit are a bad sign as regards survival and neurological salvage. I believe the best prognostic indicator is the time to first gasp, and this is best recorded by the attending first aider.

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CHAPTER SEVENTEEN

ELECTRIC SHOCK AND LIGHTNING STRIKE

Christopher Andrews

This chapter presents the science underlying the immediate treatment of electrical and lightning injuries. It will be seen that the primary concerns are for the cardiorespiratory consequences of the passage of electric current, together with burns, and certain immediate neurological sequelae. A grasp of basic physical concepts is necessary, however, to appreciate those factors governing current flow and consequent current damage within the body. The first section of the chapter therefore outlines fundamental electrical principles and their application to the human body. (See also Chapter 10 on Defibrillation.)

BASIC ELECTRICAL PHYSICS

Ohm's Law – EMF, electric current, and resistance

It is assumed that the reader has a knowledge of the basic structure of an atom – in principle, an atomic nucleus consisting of positively charged protons and neutrons of no charge, with electrons orbiting this nucleus in various well defined states of energy. While modern atomic theory has refined this picture dramatically, it is sufficient for our purposes. If energy is applied to an atom, electrons may be dislodged, and move away from it. The electron's subsequent behaviour is governed by the nature of the applied energy.

When energy is applied to a given mass of a substance, the matter can undergo a process where electrons are dislodged from many atoms simultaneously and "flow" through the material. This bulk flow of electrons constitutes an *electric current*. The energy which causes their dislodgement and motion is imparted by an *electric field*.

The smallest known unit of charge is that carried by an electron. It is extremely small, and in SI units a larger unit, the coulomb (C), is used for the fundamental unit of charge. A coulomb is a quantity of charge equivalent to that carried by 6.24×10^{18} electrons. An electron's charge is 1.602×10^{-19}C. A current of 1 ampere (amp, A) passes a point if 1 coulomb passes that point per second.

An electric field, E, is a conceptual thing without immediate physical realisation. It is an abstraction, where the "field" is defined by its observed effects. Its *magnitude* is defined by the force experienced by a unit charge in the area of influence of the field. E is a vector quantity, that is, it acts in a given direction with a given magnitude. The direction of movement of a positive charge under its influence defines the *direction* of the field.

An electric field is capable of imparting a given amount of energy to a charge, the amount differing with the position of the charge in the field. Each position in the field, therefore, has a potential energy, or simply electrical potential. The potential energy of a point in a field is the amount of energy necessary to transport a unit charge from infinity to the point, the movement being "against" the field and thus requiring energy input. Electrical potential in a field is measured in volts (V), and is often loosely referred to as the voltage at a point in the field.

How is an electric field established? It may be established in a piece of matter by the application of an electromotive force, EMF, to a region within the matter. EMF is a historical term, implying a source of force to "move electrons electrically". EMF is measured in volts, and therefore the magnitude of field established in a

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region of width d in the matter is given by V/d, where V is the voltage difference across the region established by the source of EMF.

The concept of electric current is now re-examined. It will intuitively be felt that once electrons are dislodged from an atom and flow away under the influence of a field, this current cannot keep flowing indefinitely, since electron numbers are firstly finite in a given piece of matter and, secondly, while outer shell electrons may be easy to dislodge from an atom, subsequent electrons require more and more energy to be freed for motion. As defined so far the current generated under the influence of an electric field must ultimately cease. This brings us to the concept of an electric circuit. If it is arranged that electrons mobilised from a piece of matter flow "off one edge", are collected, and re-introduced at the "opposite edge" then a continual flow of current through a material will be produced, as long as energy is supplied by the field. This circular process occurs in an electrical circuit. Thus a source of EMF can be thought of as having several roles – the establishment of a field in the space between its terminals, the supply of energy to keep electrons in motion and, when connected in a circuit, the ability to recirculate electrons through itself.

In summary then, a source of EMF (loosely termed voltage) establishes an electric field between its terminals in whatever medium is connected between its terminals. Electrons are dislodged from atoms in the material, and flow, producing an electric current, i, in the circuit thus made. The source provides energy for the process to continue.

Ultimately when a source of EMF (E) is connected across a piece of material of resistance R, a current, i, will flow in the material. This law is termed Ohm's Law, and is expressed classically as

$$i = \frac{E}{R}$$

In terms of the effect of electric current on human beings, we will discover that the body has a certain resistance, and that a circuit established through the body will cause an injurious current to flow when the body is in contact with a source of EMF. The magnitude of the current is relatively predictable.

Earth may be idealised as an infinite source of zero resistance and zero potential Thus a circuit can be established through a person either between the two terminals of an EMF source, or between one terminal of an EMF source and earth, when the other terminal of the EMF source is connected to earth. In the household distribution system in many countries (including Australia), one terminal of the generated supply is deliberately connected to earth – the so-called Multiply Earthed Neutral system (MEN). While there are good technical reasons for doing this, the practice adds a possibility of establishing an extra injurious electrical circuit through a person from generator to ground.

Sources of voltage and current

An EMF source (loosely, a voltage source) can be defined as any piece of apparatus capable of producing a potential difference and, therefore, an electric field between its terminals. The most common forms of EMF source encountered in daily life are dry or wet cell batteries where the potential difference is established by virtue of an internal chemical reaction, and electrical generators. A generator is a device which makes use of the phenomenon that a conductor moving through a magnetic field, or alternatively a conductor stationary in a moving magnetic field, will have an EMF generated across its two terminal ends. It is in this manner that household electric current is generated, and then transformed and distributed to households for use in the many applications we are used to.

The above sources of EMF are properly called *voltage sources*. A circuit, of resistance R, connected to a voltage source will have a current flowing through it governed by Ohm's Law. It is emphasised that for all practical purposes, the applied voltage is the independent variable in this equation, and the current is the dependent variable, with R as the parameter. Household electric outlets, and batteries, are voltage sources.

There do exist sources of *current* however. That is, they produce a constant current emanating from their terminals regardless of the resistance connected to them. The governing equation for circuits connected to these sources is now a transformation of Ohm's Law:

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$V = iR$

where i is the current (the independent variable) produced by the source, R is the resistance of the circuit connected to its terminals, and V is the voltage (dependent variable) which is forced to appear across the resistance material connected to the current source. Lightning is a good example of a current source.

Thus voltage sources are ideally sources which produce a constant voltage across their terminals regardless of the current flowing through them or the resistance connected across them. Current sources are devices which produce a constant current regardless of the resistance connected across them and the voltage appearing across the circuit to which they are connected. The degree to which these idealisations are approached in practice is variable but the distinction is important.

Direct and alternating current

Two major types of electric current will concern our later deliberations, namely direct and alternating current.

Direct current (d.c.) is current which only flows in one direction for all time. Current caused to flow by a battery is an example. Alternating current (a.c.), of which household current is an example, is a current which flows in one direction then the other, and is usually assumed to have regular alternations.

In the above statement of Ohm's Law there is no dependence on time, and indeed this is true for most circuits of import to human injury. So the current through a resistance will follow the form of the voltage applied across it.

Why is alternating current used? The reasons why household current is a.c. are too many to mention, but

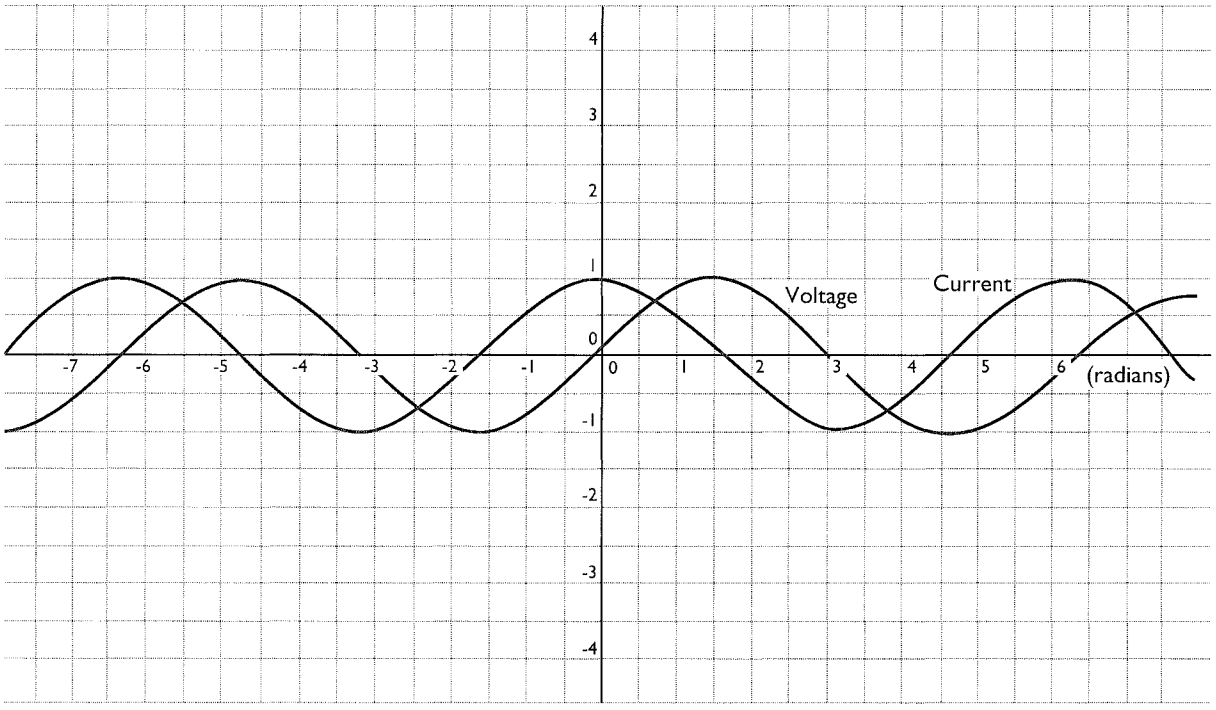


Figure 17.1 *Relationship between voltage applied across a capacitance and the current flowing through it. The current leads the voltage by a phase angle of 90°.*

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primarily revolve around ease of generation, transformation from one voltage level to another, and efficiency of transmission over long distances. The body responses to direct and alternating currents are quite different and thus the distinction is necessary.

Resistance and impedance

The characteristic of resistance is that the current flowing through a pure resistance has the same form as the applied voltage. The current flowing through a *capacitance,* on the other hand, is proportional to the rate of change of the voltage applied across it. The relationship between voltage and current for a capacitance is shown in Figure 17.1. The current "leads" the voltage by 90°.

A capacitance consists of two parallel conducting plates separated from each other by an electrical insulator called a "dielectric". This is shown in Figure 17.2. As the voltage in sine form rises just a little from zero, there is a big inrush of electrons onto the "empty" top plate of the capacitance. There is a corresponding outrush of electrons from the bottom plate to maintain electrical neutrality. As more electrons are added to the upper plate with rising voltage it becomes more negatively charged and opposes the addition of these further electrons. Thus the current falls and reaches zero when the applied sinusoid changes no more, that is at its maximum point. As the applied voltage decreases back to zero, charge is removed increasingly quickly from the capacitor and the flow reaches its maximum as the voltage reaches zero, the value where the rate of change of the voltage is maximum. The reverse process applies during the negative half cycle of the applied sinusoid.

From the external viewpoint current still flows in the external circuit in response to the time varying voltage applied to the capacitance. The electrons flowing "out" of the capacitance, however, are not the "same" electrons as entered the capacitance. Those leaving the bottom plate are electrons reacting to the addition of electrons to the top plate. The current is thus termed "reactive" current. The current is inversely proportional to the size of the capacitance and directly to the angular

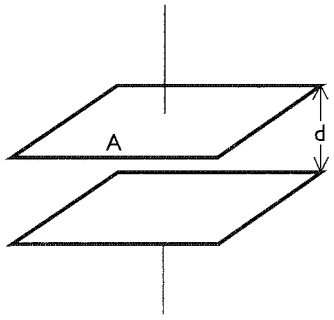


Figure 17.2 *Physical construction of a capacitance. Two parallel conducting plates separated by an insulating dielectric.*

frequency, ω. That is, a capacitance is a device that can "conduct" a.c. only and, for a given capacitance passes more current at higher frequencies than lower frequencies. The SI unit of capacitance is the farad (F). This is a large unit and, in practice, most capacitances are only a small fraction of a farad in size.

A pure capacitance has an infinite resistance and therefore, in a circuit is said to have pure capacitive reactance. When reactances and resistances are connected together in circuits the resulting combination, made up of mixed reactance and resistance, is termed the circuit *impedance.* Advanced methods of circuit analysis are needed to predict the behaviour of these circuits as they are both time and frequency dependent, and contain multiple phase lags and leads.

Heating effects

As current is conducted through a resistance, the resistance rises in temperature. The power at which the electric current works is given in watts, and the energy dissipated in the resistance is given by joules (watts × time). The temperature rise that this engenders in, for example, tissue depends on its specific heat.

For a given current higher resistance tissues absorb more energy, and tissues of lower specific heat capacity reach higher temperatures. Thermal tissue burns are thus possible, and are a feature of electrical injury, but not of lightning injury where the time of current passage is very small.

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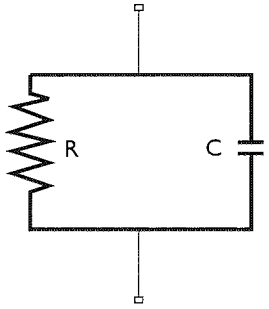
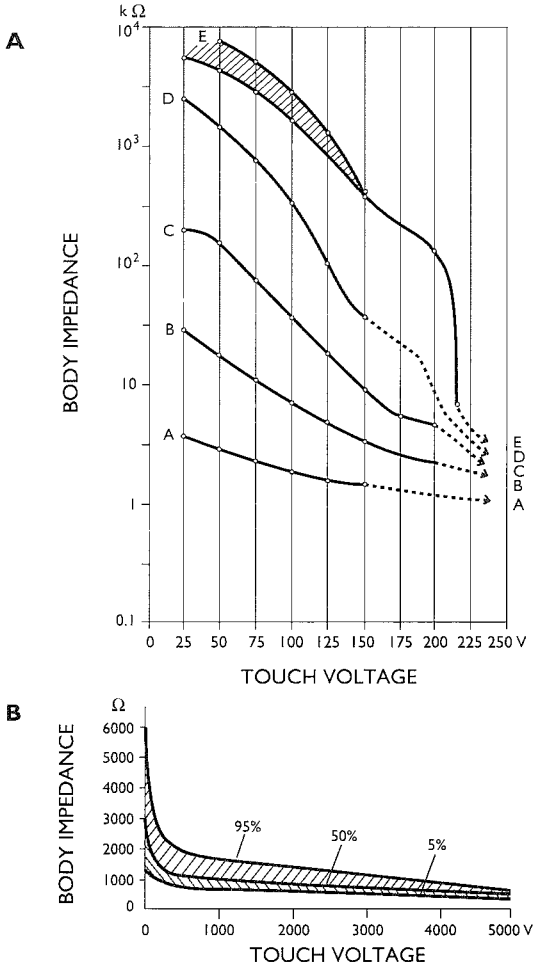


Figure 17.3 *Skin modelled as a parallel resistance and capacitance.*



Figures 17.4 A and B *Representative curves for total body impedance under varying conditions, showing:* ***A*** *the variability for contact voltage 0–250 V, and contact areas 8200(A), 1250(B), 100(C), 10(D), 1(E) mm²; and* ***B*** *body impedance as a percentile diagram for various voltages of contact. (Adapted from Bridges* et al. *with permission.)*

Body impedance

The effects of electrical current on the body can now be examined. The first examination will be in physical terms, and later the clinical implications will be examined.

When a voltage is applied to the body, current flows through a circuit including body components – firstly through skin, then through the body's internal milieux, then through skin a second time. The skin impedance is by far the largest component of the total body impedance, and is also the most complex.

The internal milieux constitute a quite ionic medium, and represent a relatively pure resistance. A value from 500 to 2000 ohms for the internal resistance is commonly assumed, and depends on the length of path through the body, and to some extent on the particular tissue through which the current passes.

EPIDEMIOLOGY OF ELECTRIC AND LIGHTNING SHOCK

Electric shock

Electric shock is usually divided into two categories – high-voltage shock (due to contact with greater than 1000 V) and low-voltage shock (less than 1000 V). Recent work would suggest that estimation of the electric field within the body is more relevant than the absolute voltage involved. A low voltage operating over a short distance (for example, when a child places a domestic extension cord in its mouth) may be just as harmful for the intervening tissue as an industrial accident where a workman contacts 11 000 V between an outstretched arm and a foot. Further subdivision of electrical injuries into domestic (usually low-voltage) and industrial (more likely to be higher voltage) circumstances is often made.

It is estimated that in the United States 10 000 workers will be killed annually in the course of their employment. Ten million will be accidentally injured. Death from electric shock ranks fifth in the causes of these deaths in the workplace.

A Virginian study gives representative figures for the industrial arena. Over the nine-year period 1977–85,

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there was a total of 277 electrical deaths in the State. Industrial deaths, as per death certificate records, numbered 196, a rate of 0.9/100 000 workers per year. Of these 186 were recorded in the Workers Compensation system as being occupational electrical deaths. The distinction is not altogether clear and may relate to recording techniques. Of the latter 186, 179 were due to electric shock and 7 were due to lightning. In the five-year period, 1980–85, of 341 workers sustaining an electrical injury, 72 were killed, giving a fatality percentage of 21 per cent.

Examining industrial deaths first, males in utility companies predominated (10/100 000 per year), with mining (5.9/100 000 per year) and construction (3.9/100 000 per year) industries following. These groups, however, only accounted for 50 per cent of total electrical deaths. The most common mechanism for the fatal shock was with power line contact (53 per cent), and this was followed by contact arising from machine or tool usage and/or repair (22 per cent). In this latter category, approximately half occurred when a worker was operating an apparently safe machine under usual operating conditions. Alcohol, in the industrial setting, involved only from 1.5 per cent to 2 per cent of 101 subjects tested. Multiple deaths from single exposures were relatively rare – in five cases two people were killed, and in two cases three were killed, the remaining being single subject incidents. Sixty-five per cent of deaths occurred in summer months, a seasonal trend which is not fully explained. The age groups 20–29 and 30–39 accounted for 42 per cent and 22 per cent of fatalities respectively. The most dangerous times of day were around 10 am and 3 pm, and the most dangerous days were Monday, Tuesday and Thursday. Tradesmen and

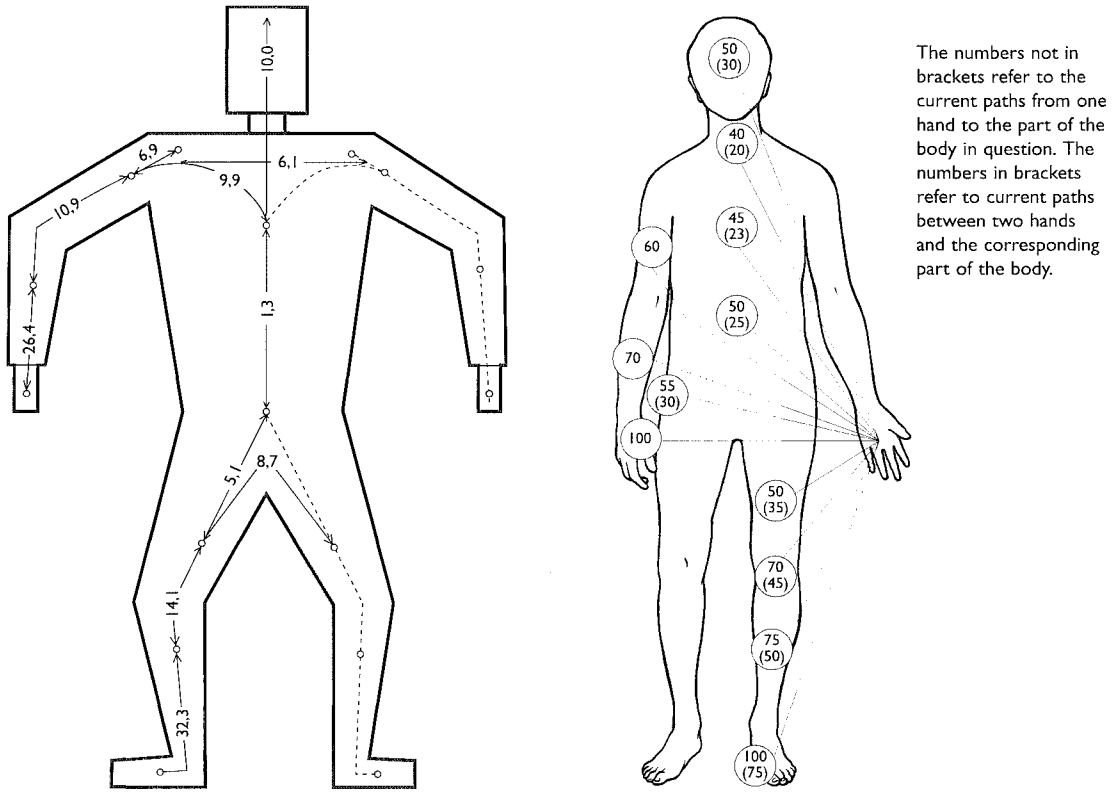


Figure 17.5 *Variation in body impedance for differing pathways, and the distribution of internal resistance in the body. (Part by permission of Standards Association of Australia and part adapted from IEC Draft International Standard Doc TC64 234.)*

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labourers accounted for most deaths (101/178) as opposed to sales (0/178), clerical (0/178), and professional (6/178) categories.

In examining individual practices at the time of accident, the number of incidents involving the inadvertent contact of metal equipment with overhead lines was noteworthy, and deserves attention in formulating safe work practices. Another study suggests that as many as 10 per cent of total deaths are involved with metal ladders, alone, contacting overhead lines. Similar contact, for example, with metal ladders held aloft to tend antennae with subsequent power line contact is also disproportionately represented in domestic accidents.

Turning now to incidents involving the general public, it is difficult to discover information of a similar nature to that available for industrial incidents, perhaps due to different reporting requirements. The following information has been kindly provided by the Queensland Electricity Commission, Australia. They also note, very importantly, that domestic electrical deaths have decreased in number dramatically since the widespread installation of compulsory Residual Current Devices (RCDs) for protection *(vide infra)*.

In Queensland, with a population of approximately 2 million, there was an average of 11.7 deaths per year among the general public between 1984 and 1994. In 1994, 50 per cent of these involved power line contact, and 33 per cent involved faulty wiring. Only 16 per cent involved appliances and 16 per cent involved faulty power cords. This compares with 20 per cent, 10 per cent, 50 per cent and 20 per cent respectively in 1983. The reduction in domestic deaths may be attributed to RCD installation. The majority of power line contact was with overhead mains, with a small contribution from overhead service wiring and fallen conductors. Cabling errors accounted for the majority of faulty wiring fatalities. The major areas of danger from power cords were incorrect connections, and damaged and frayed cords allowing exposure to conductors. With appliance contact, the major danger was contact with exposed conductors. It may be seen that most of these causes of death could be avoided with care to avoid overhead contact, requiring wiring to be installed by licensed tradesmen only, and attention to correct maintenance and repair of electrical equipment.

Non-fatal accident statistics are harder to derive, owing to lack of standardisation of reporting requirements. As expected, however, percentages within the categories mentioned above for fatal accidents are mirrored in non-fatal accidents. The average number of non-fatal accidents per year in Queensland over the same period was 134.9. It may be seen that injuries outnumber fatalities by approximately 10:1.

Lightning shock

The incidence of death from lightning has shown significant changes in its distribution over the past 50 years, and this has been extensively studied in the Australian literature. The annual lightning-caused death rate is expressed in numbers per million of population, and Table 17.1 shows typical values.

As there are significant fluctuations in annual death rates, a "running average" is the figure usually adopted. The decrease in mortality rates has been attributed to increasing urbanisation, and possibly greater awareness of the hazard of lightning strike. There may also be an association with decreasing thunderstorm activity over that period. The ratio of the number of lightning injuries to lightning deaths has been variously estimated from as low as 2:1 to as high as 5:1. The generally accepted figure for lightning strike mortality is 30 per cent, and this is surprisingly low given the violence of the event.

Other features of lightning strike mortality include a sex bias towards men, with men accounting for 1.67 times the total population lightning death rate, and women accounting for 0.33 times. Bias in age group places the age group 20–29 at risk in most studies.

The location of persons killed indicates dangerous environments for individuals. These include sheltering under trees, on open water, on tractors, and playing golf. In the United States a small number are killed annually using a telephone. Outdoor and indoor activity when struck is shown in Table 17.2 which gives Australian figures.

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The changes in pattern over the periods indicated may reflect the change in work pattern with less outdoor unmechanised manual labour being performed in recent years, and also the trend to shorter working hours with more time available for outdoor recreation.

The seasonal distribution of lightning fatalities follows the occurrence pattern of lightning itself, notably peaking in summer months where summer storms are the norm. Further, the time of day of fatal strikes follows the same pattern with times midday to 6 pm being peak times.

The thunderstorm activity level for a given region is estimated by its "thunderday level" or "keraunic level". This is a crude measure, and is defined as the number of days per year on which thunder is heard at a given locality. Typical values are 30 for Brisbane in subtropical eastern Australia, and 90 for Darwin in tropical northern Australia. Mackerras has inferred the mean annual death rate vis-a-vis keraunic level on a world-wide basis and this is shown in Table 17.3.

Table 17.1 Changes in the lightning mortality rate per million of population over a 50-year period

	1920	1970
Singapore	2.5	1.5
Australia	1.4	0.3
United States	1.6	0.3
England/Wales	0.4	<0.1

Table 17.2 Circumstances of lightning strike over two 12-year periods

	1945–57	1957–69
Outdoors, work	67%	45%
Outdoors, recreation	28%	50%
Indoors, work	3%	5%
Indoors, recreation	2%	0%

HUMAN RESPONSE TO ELECTRICAL AND LIGHTNING SHOCK

Electrical thresholds

The response of the human body to electrical current will now be outlined, and it will be noted that it is possible to predict with a degree of certainty the effect of current, once calculated, on the body. This is not to say that it is entirely certain just what the best index of injury to the body is. For example, it is accepted fairly widely that the important factor for derangement of cardiac function is the current passing through the heart. None the less a body of opinion maintains that the important parameter is the charge transferred to the body that is important, and another body of opinion suggests that the energy transferred is the important parameter. We will not pursue this debate here.

As the current passing through the body increases, various thresholds are passed. These are now examined.

Threshold of perception

The first threshold that is passed is the perception threshold. It is defined as the minimum value of current which causes any sensation for the person through which it is flowing. It depends on several parameters, much the same as body impedance, which is not surprising as it is body impedance that determines the magnitude of current flow. A general value of 0.5 mA is accepted for current frequencies of 15–100 Hz, regardless of the time for which it is applied. For currents above 100 Hz, a multiplicative factor is applied, and this is shown in Figure 17.6. Above 10 000 Hz the threshold rises linearly to become 100 mA at 100 kHz. Above that frequency perception changes from a feeling of tingling to a feeling of warmth and requires several hundred milliamperes of current.

The above refers to alternating current. As is true for many sensory modalities, stimulation of body sense

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organs only occurs when the stimulus is changing. Thus the situation for direct current is different. In general, deleterious effects of direct current occur only at instants of change of direct current and require magnitudes some three to four times greater than corresponding 50 Hz a.c. values. For perception, only the making or breaking of current is felt, and the threshold is approximately 2 mA.

Let-go current

The next threshold which is passed is the threshold of let-go. This is the maximum value of current at which a person holding electrodes can let go of the electrodes. It is a consequence of induced electrical muscle spasm. A value for a.c. of approximately 10 mA is typical. Figure 17.7 indicates the variation of this value with frequency up to 10 000 Hz. Above 10 000 Hz there are no data available on this parameter. Presumably current of this frequency is not able to stimulate muscle or nerve membranes easily.

For d.c. there is no real definable let-go threshold, as the muscle tetanus giving rise to inability to let go occurs significantly only at make or break of current. A value of 300 mA is often taken to indicate a value where let-go is not possible, or only after several seconds of current flow leading to muscle adaptation.

Table 17.3 Inferred lightning mortality rate vis-a-vis keraunic level

Keraunic level days per year	Mean death rate per million population
5–20	0.2
20–40	0.4
40–80	1.0
80–200	1.7

Fibrillation thresholds

Anticipating a later discussion the reader is asked to accept that the most dangerous consequence of electric shock is the induction of ventricular fibrillation. The final threshold the body passes with increasing current is the threshold of ventricular fibrillation – that minimum value of current flowing "through the heart" which causes ventricular fibrillation. The current through the heart is approximately that which flows in a left hand to left foot circuit during the shock. We will return to other paths later.

The threshold for a.c. is set out first. An important concept is that of the cardiac "vulnerable window". We will return later to the meaning of this term, but suffice it to say that the early part of the electrocardiographic T wave is a period of maximal randomness in the excited/recovered cycle of myocardial muscle cells following contraction. An electrical stimulus need only be relatively small during this period compared with other parts of the cycle to induce fibrillation, and also causes maximum dysfunction during this window. Thresholds of fibrillation therefore aim to give conservative values based on this interval.

Given that all humans show individual variation, thresholds for fibrillation are expressed in probability terms. For the range 15–100 Hz, the threshold of fibrillation is shown in Figure 17.8. It is expressed in terms of a time/current diagram implying that a given current attracts a different likelihood of inducing fibrillation if it is applied for differing times. This may indicate that it may be energy rather than current which is the injuring agent in electric shock.

Above 100 Hz, if a shock longer than a cardiac cycle is experienced, then the effect of frequency is as shown in Figure 17.9. For shock durations less than one cardiac cycle in length, and for frequencies above 1000 Hz, there are no fixed data available.

While the current/time curves are accepted as the standard prediction vehicle, some writers have attempted to derive analytical equations for the fibrillation threshold. The most famous is that of Dalziel.

$$I = \frac{K}{\sqrt{T}}$$

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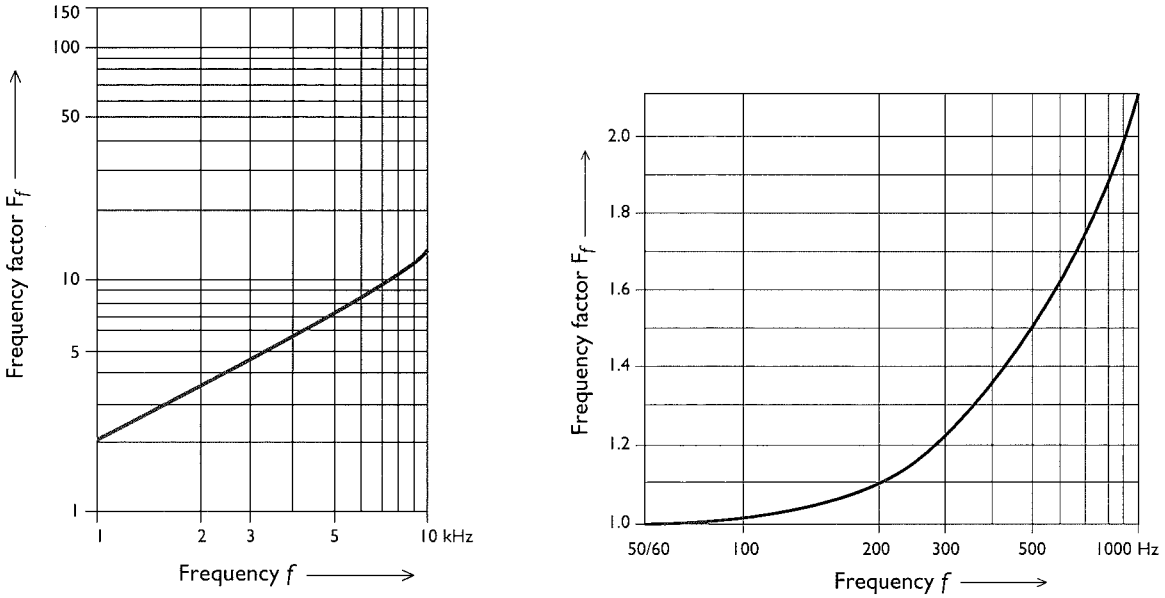


Figure 17.6 *Variation of perception thresholds for currents of frequency 100–10 000 Hz. (By permission of Standards Association of Australia.)*

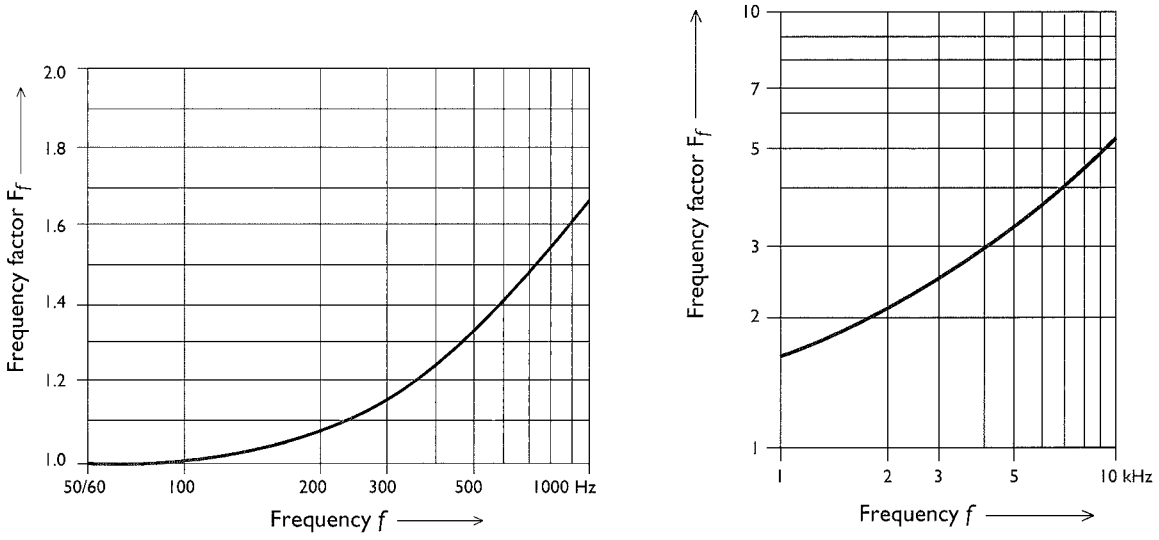


Figure 17.7 *Variation of let-go thresholds for currents of frequency 100–10 000 Hz. (By permission of Standards Association of Australia.)*

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Zones	Physiological effects
Zone 1	Usually no reaction effects.
Zone 2	Usually no harmful physiological effects.
Zone 3	Usually no organic damage to be expected. Likelihood of muscular contractions and difficulty in breathing, reversible disturbances of formation and conduction of impulses in the heart, including atrial fibrillation and transient cardiac arrest without ventricular fibrillation increasing with current magnitude and time.
Zone 4	In addition to the effects of Zone 3, probability of ventricular fibrillation increasing up to about 5% (curve c_2), up to about 50% (curve c_3) and above 50% beyond curve c_3. Increasing with magnitude and time, pathophysiological effects such as cardiac arrest, breathing arrest and heavy burns may occur.

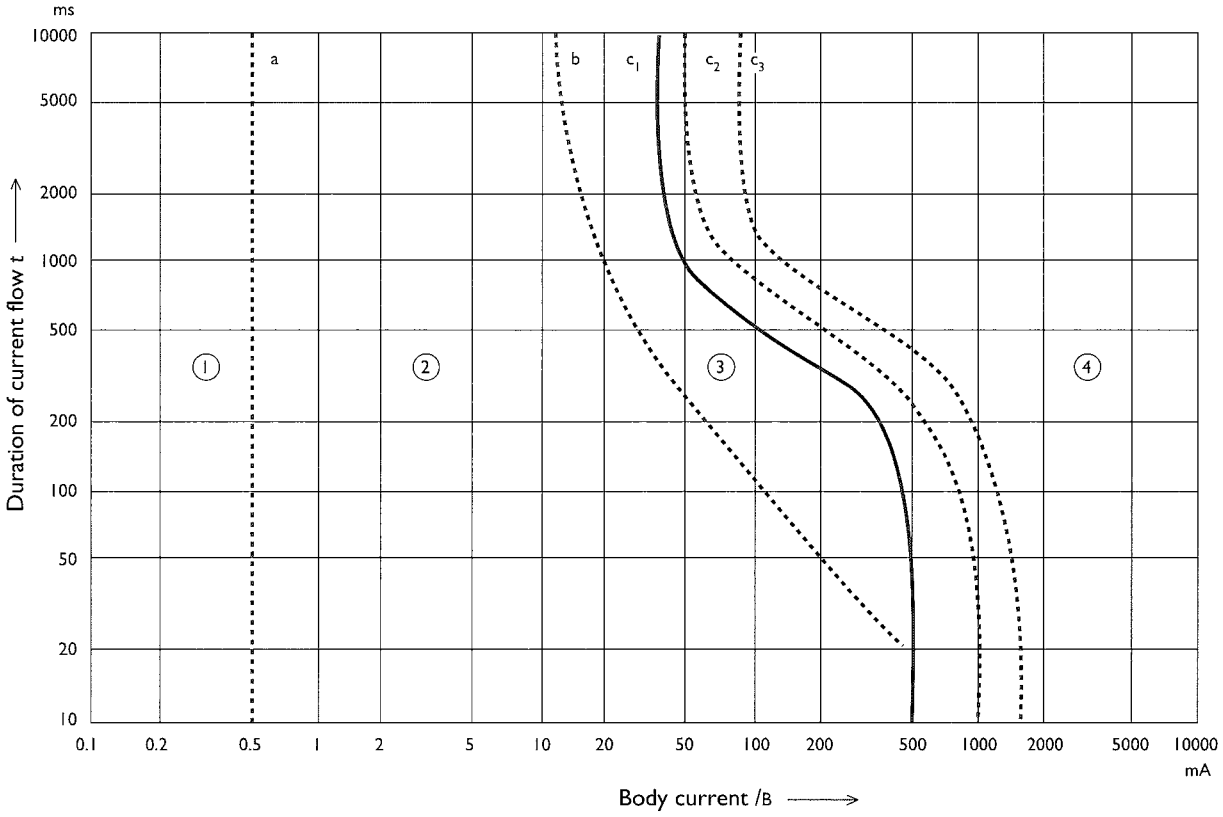


Figure 17.8 *Cardiac threshold for ventricular fibrillation for a.c. in the range 15–100 Hz. (By permission of Standards Association of Australia.)*

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Plate 5 *Entry/exit burns from lightning strike.*



Plate 6 *Linear burns from lightning strike.*

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Plate 7 *Marathons, half-marathons, and so-called "fun runs" are accompanied by heat stress, cardiac problems and soft tissue injuries requiring first aid. (Photograph courtesy Inform Public Relations, Sydney.)*



Plate 8

Plate 8 *Severe frostbite in the fingers of a Himalayan climber. The outcome was the loss of the tips of eight fingers. (Photograph courtesy of Mr D. Capps.)*

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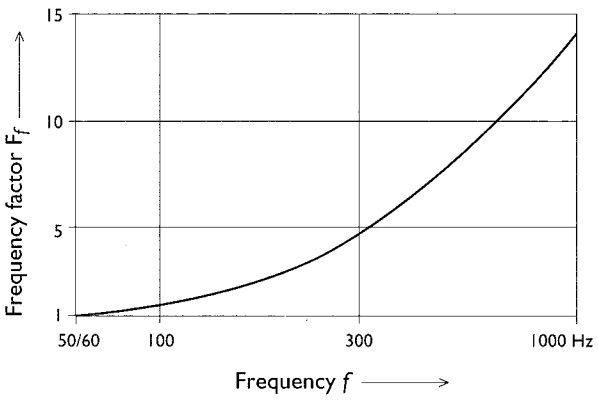


Figure 17.9 *The effect of frequency on the fibrillation threshold for a.c. for frequencies above 100 Hz and shock durations greater than one cardiac cycle in length. (By permission of Standards Association of Australia.)*

where I is the 0.5 percentile threshold current for fibrillation at 50–60 Hz in mA, and T is the shock duration in seconds. K is an empirical constant and is 116–183 as originally derived, depending on individual body variation. The range of applicability was stated to be shocks from 8.3 msec to 5 sec duration.

For d.c. a current/time curve is available and is shown in Figure 17.10. This curve must be interpreted with caution. The direction of current flow is important, with the threshold for current which flows from left hand to foot, with the foot being the negative polarity electrode (a "falling" current), being twice that for current flowing in the opposite direction (a "rising" current). A hand to hand path is considered unlikely to cause fibrillation. For shock durations shorter than 200 msec, the d.c. threshold is about the same as that for a.c. (rms). For shock durations longer than the cardiac cycle, the threshold is significantly higher than that for a.c. Figure 17.10 is drawn for a rising current for the left hand–feet circuit.

Current pathways and current factors

It has been implicitly assumed that the heart current above is approximated by that current which flows through the body in a left hand to left foot path. Various factors are available to determine the heart current resulting from current flowing in other pathways.

RESPONSE TO LIGHTNING SHOCK

Mechanisms of shock

There are four basic mechanisms by which lightning can impinge on the individual. The first is self-explanatory and is by direct strike where the individual takes the full force of the lightning flash as the only object struck. The second is termed a "side flash". An individual may be standing next to an object which is struck, such as a building or a tree. The main brunt of the flash is borne by the object, but a proportion of the current "jumps" from the object to be conducted to earth via the individual. The third mechanism is via contact potential and is similar to side flash. An individual may be in contact with a stricken object and receives a shock when current is conducted through the individual directly from the object.

The fourth mechanism is termed "earth potential rise" (EPR). As an idealisation it is assumed that the earth is an infinite source of zero potential and is of zero resistance. In practice this is not so. The earth has a finite resistance of its own and when current from a lightning flash is injected into the ground, remembering that lightning represents a current source, it flows away from the impact point of the flash setting up a potential gradient from the base to infinity. Two points on the earth, in a radial direction from the base of the flash, are, therefore, at differing potentials and can then act as a voltage source. A person standing with, say, two feet at differing potentials can have a current conducted through their lower extremities in proportion to the body impedance of this path. For the human this may not be of great significance as the current is usually small, and the pathway does not involve vital structures. For a four footed animal, however, whose forelimbs and hind limbs are widely spaced, and in whom such a pathway involves vital thoracic structures, the result may be less happy.

Figure 17.11 summarises these mechanisms.

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Zones	Physiological effects
Zone 1	Usually no reaction effects.
Zone 2	Usually no harmful physiological effects.
Zone 3	Usually no organic damage is to be expected. Increasing with current magnitude and time, reversible disturbances of formation and conduction of impulses in the heart are likely.
Zone 4	Ventricular fibrillation likely. Increasing with current magnitude and time other pathophysiological effects, for example heavy burns, are to be expected in addition to the effects of Zone 3.

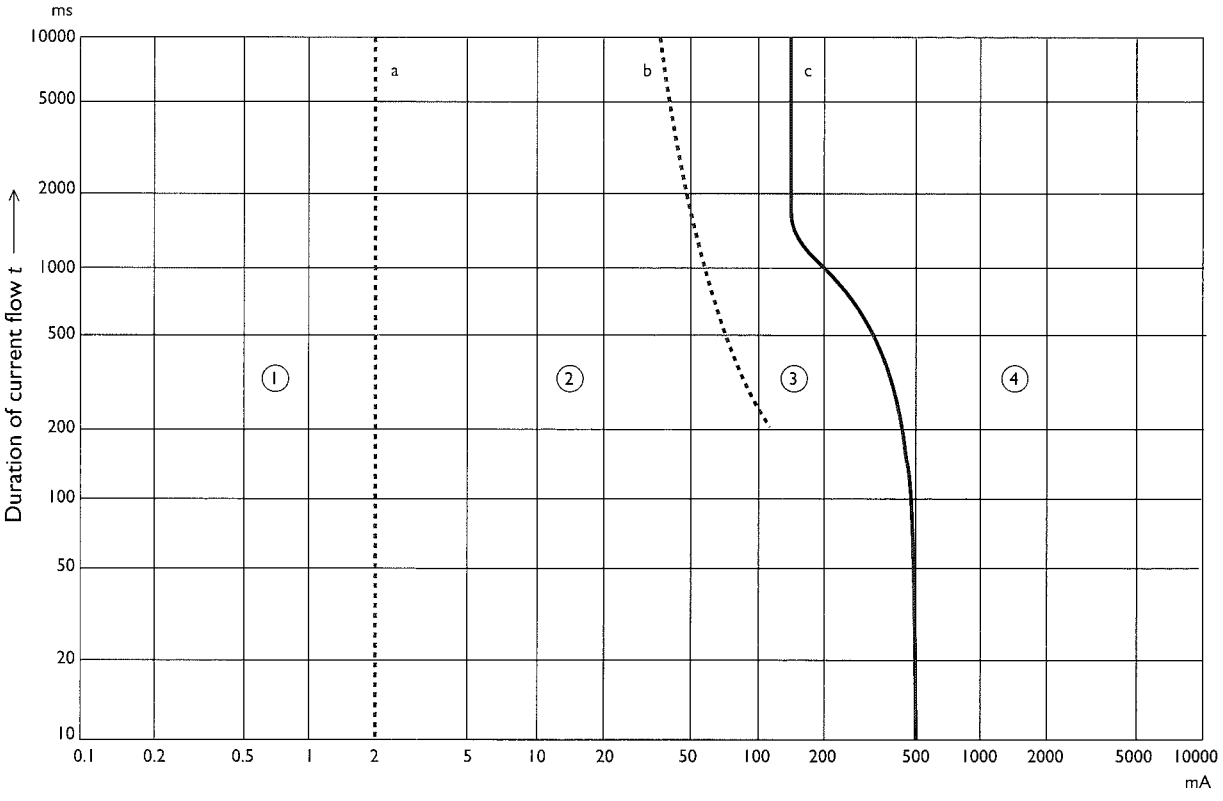


Figure 17.10 *Cardiac threshold for ventricular fibrillation for d.c. (By permission of Standards Association of Australia.)*

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Cardiorespiratory response

The most significant effect of lightning on the body is on the cardiorespiratory system, and it is only in recent times that the progression of this injury has been confirmed.

At the time of strike, the heart goes into immediate asystolic arrest, and the respiratory apparatus also goes into standstill. An initial spasm of respiratory musculature is seen, followed by relaxation and standstill in a neutral thoracic volume. After a small number of seconds the heart regains its intrinsic automaticity and restarts in a short period of bradycardia followed in quick succession by an intense tachycardia. This is catecholamine-mediated, but with the predominant amine being noradrenaline rather than adrenaline. Why this is so is as yet unexplained. Respiratory automaticity is not re-established, however, and without external ventilation the heart succumbs to a secondary hypoxic arrest and the victim dies.

The genesis of the cardiac arrest is thought to be a combination of both central brainstem vagal out-pouring and direct myocardial "stunning". The heart is fortunate however in having its own intrinsic automaticity and is able to restart "peripherally". Respiratory paralysis seems almost completely centrally mediated and as intrinsic respiratory automaticity is vested centrally, it is understandable that respiratory recovery does not occur spontaneously.

Current pathways

The pathways of lightning current through the body remain enigmatic. A search to explain the pathways is central to explaining the internal damage caused by lightning.

It has been demonstrated that a cranial lightning strike branches out over the cranium and current streamers enter the body through the cranial orifices – eyes, ears, nose, and mouth. It has been noted that each of these orifices lead in short order to the CSF (cerebrospinal fluid) – the eyes via the optic foramina, and also via the lachrymal apparatus to the nose and pharynx; the nose and mouth directly through the posterior pharyngeal wall to the brainstem; the ears through the auditory meati to the cerebellar angles and brainstem. It is hypothesised that these pathways explain the impingement of lightning current on the brainstem through the CSF, and histological evidence has been advanced to indicate that neural tissue in contact with CSF is at most risk. Further these pathways lead past lower brain centres – the cerebellum, basal ganglia, and brainstem – where the predominant damage is observed.

Also from cranial strike and entry as above, the pathways to major cervical and upper thoracic blood vessels are short and transmission of current directly to myocardium and thoracic musculature is facilitated, thus explaining the direct effects of lightning.

These observations lend support to the preferred passage of electric current via the ionic fluids of the body, and this is in accord with their various resistivities. CSF and blood are good conductors, with muscle being an intermediate conductor. Despite claims to the contrary, nerve is not a good electrical conductor being so lipid-rich and sheathed in lipid, and lipid and bone are poor conductors.

ELECTRIC SHOCK

Ventricular fibrillation – theories of electrical induction and maintenance

The most common arrhythmia generated by low to medium voltage electric shock is ventricular fibrillation (VF). It is an arrhythmia of incoordinate myocardial contraction with loss of mechanical pumping ability of the heart. The ECG finding is of random deflection at a frequency between 200 and 500 per minute. The circulatory arrest which follows can lead in 4 minutes to irreversible cerebral damage.

A theory of VF must explain both electrical induction and maintenance of the arrhythmia. It must explain the susceptibility of the heart in its vulnerable window – the first 20 per cent of the T wave, and also the related R on T phenomenon by which VF may be induced by an extrasystole occurring in the vulnerable window. Finally, it must explain the termination of VF either spontaneously or by electrical defibrillation.

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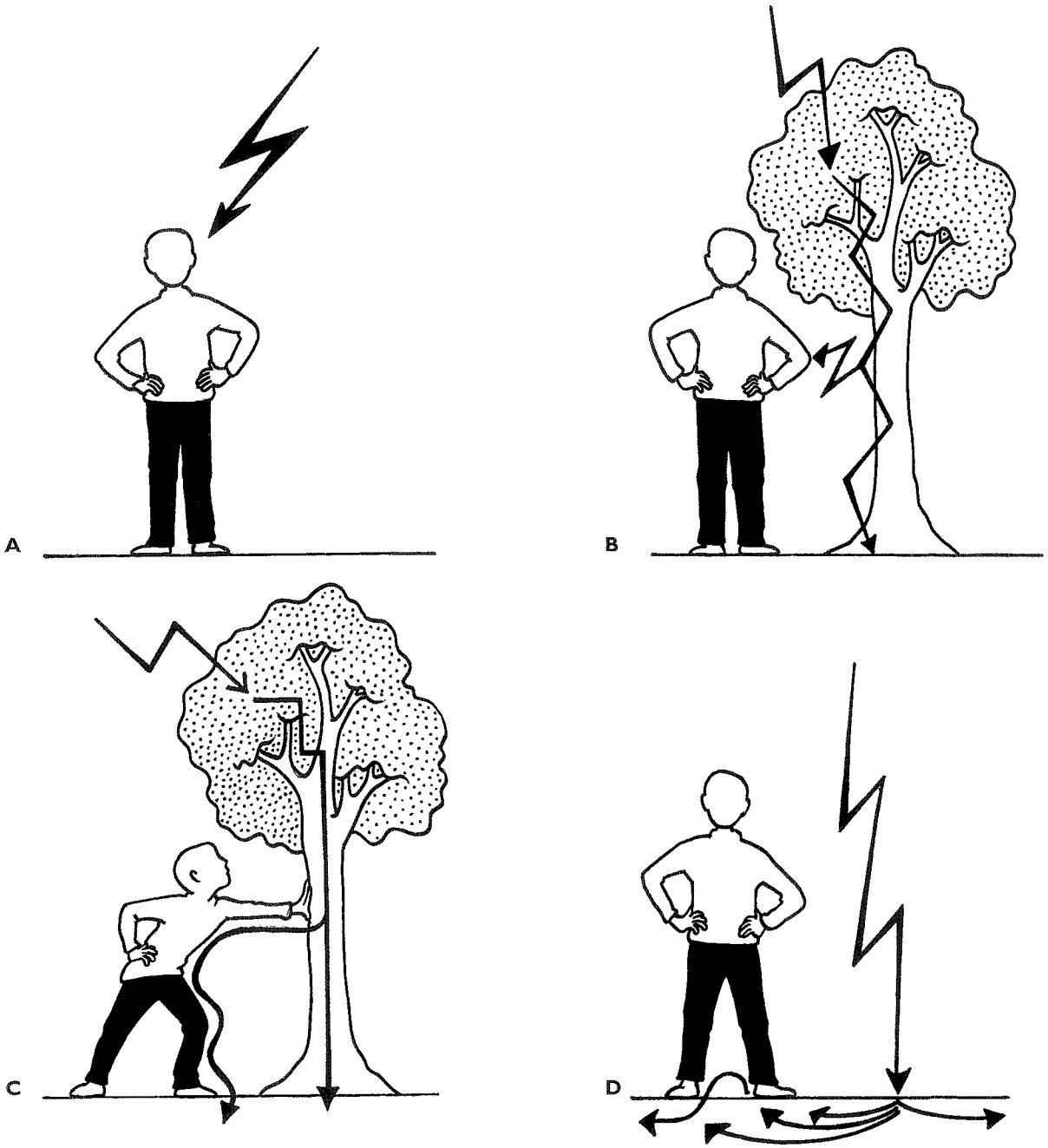


Figure 17.11 *The mechanisms of lightning shock:* ***A****, direct strike* ***B****, side flash* ***C****, contact potential* ***D****, earth potential rise.*

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Two theories have been advanced to explain the maintenance of the arrhythmia. The first is the theory of ectopic automaticity, and the second is the phenomenon of re-entry. The first theory proposes the establishment of several spontaneous ectopic foci all firing in random order and at high rate. This theory is now less accepted than re-entry and cannot, for example, explain the fact that a dilated heart, or an otherwise large heart, is more susceptible to VF. Experimental evidence does not support the establishment of ectopy even under strong electrical stimulus, and the spontaneous offset of ectopic activity is less accepted.

Re-entry is able to overcome these deficiencies. Re-entry requires the establishment of a circular path of conduction in myocardial muscle, which circular path is normally stimulated by entry of an excitatory impulse at one point in the pathway. Under normal circumstances the wave of excitation would spread in both directions around the circular path equally, meeting and becoming extinct opposite the entry point. This is shown in Figure 17.12a. Extinction occurs because the length of the wave of excitation, that is the state of refractoriness at any point, lasts longer than the spread of excitation, that is the wave of excitation is longer than the pathway.

Consider now what happens if one of two conditions is met. First, there is a unidirectional block in conduction in one part of the circle such that excitation normally does not spread in the forward direction down one limb. The wave of excitation travels down one limb of the circle and can then be transmitted back into the circle re-exciting it spontaneously. The pathway has effectively been lengthened so that now the wave of excitation is shorter than the pathway. This lengthening could also happen simply physically and explains the susceptibility of the dilated heart to VF. Further stimulation of the heart in the early phase of repolarisation (the vulnerable window) also effectively shortens the wave of excitation (see Figure 17.12b). So either physical lengthening of a circle of conduction, or electrical shortening of the wave of excitation, or a unidirectional block in a circle of conduction which achieves the former, can all predispose to VF.

Stimulation of a pathway by an exogenous stimulus (eg an electric shock) can produce an effective unidirectional block in a circle by inducing an extrasystole in a period of relative refractoriness. The premature stimulation necessarily introduces a wave of shortened excitation when occurring in the vulnerable window, and can spread only in one direction – the direction of maximal repolarisation. An alternative mechanism of producing a unidirectional block is by physical myocardial damage – perhaps direct myocardial contusion of thermal burn. This mechanism may in fact also explain the development of "late" arrhythmiae following electric shock, where the development of a pathological response to a damaging stimulus takes time to develop. The author has in fact seen late arrhythmiae (fatal) occurring two days after an initial shock.

In terms of VF induction, one must develop a coherent theory of the vulnerable window. It has been hinted at above, but is now amplified. As the normal wave of excitation spreads in both directions down a circle of conduction, both tails of the wave are followed by areas of absolute refractoriness, then relative refractoriness, and then repolarised tissue. Thus in one segment of the circle is a so-called "excitable gap" of myocardial muscle made up of fully repolarised tissue. Provided stimulation occurs when there is still some depolarised tissue in the circle, then a functional unidirectional block is produced, since the stimulated wave of depolarisation can only move "backwards" into excitable tissue. This situation is only possible in the early T wave of the ECG. It is shown in Figure 17.13.

In these terms, defibrillation can be seen as a means of reducing all myocardial tissue to the same level of depolarisation followed by relatively uniform repolarisation and the removal of unidirectional conducting paths of functional origin.

Neurological response

The neurological response to electric shock is difficult to separate from that due to cerebral hypoperfusion following induction of VF. However, some writers are convinced that electrical damage to the CSF induces unconsciousness when VF has not supervened.

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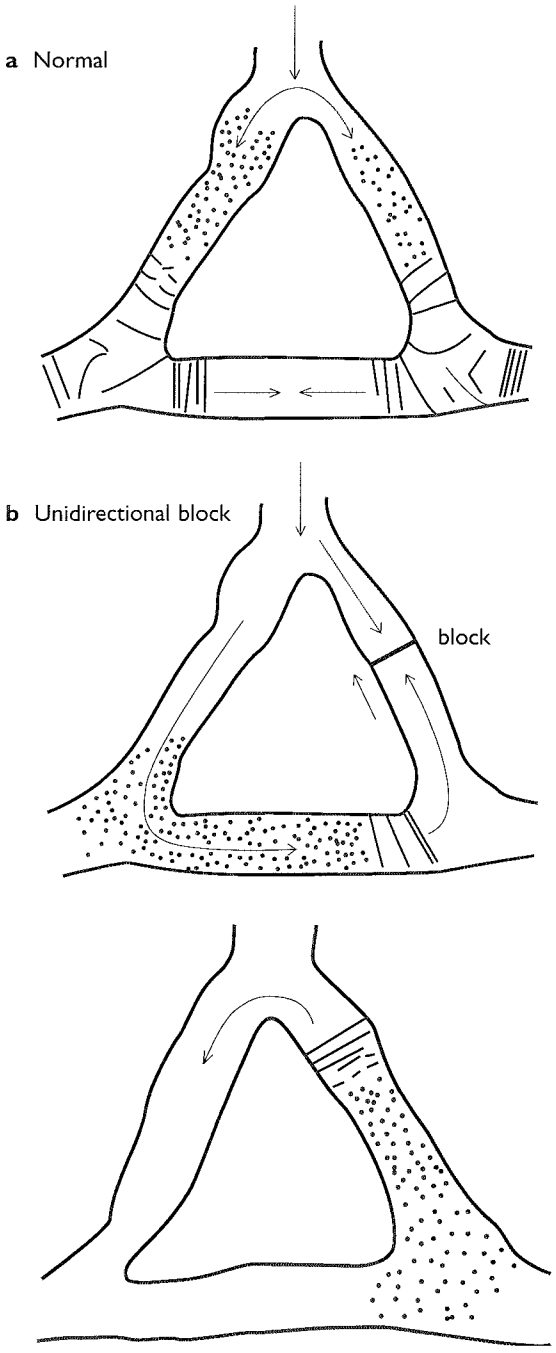


Figure 17.12 *The re-entry theory of ventricular fibrillation.* ***a*** *Under normal circumstances a wave of excitation enters a circular pathway and spreads in both directions, becoming extinct opposite the entry point.* ***b*** *Under pathological circumstances of either a unidirectional block in one limb, or where the wave of excitation is shorter than the pathway, restimulation of the pathway can occur. (Adapted from Bridges* et al. *with permission.)*

Particularly in high-voltage injury, central effects to paralyse respiratory function are regularly noted. Thus it is argued that all electrical victims, and particularly victims of high-voltage injury, should be regarded as potentially requiring respiratory support. In the author's experience such need, independent of support for VF, is very rare in low- to medium-voltage injury, and monitoring for cardiac arrhythmiac is a greater necessity. The possibility of associated cranial and cerebral trauma must always be considered. The brainstem is regarded by many as at particular risk, particularly with current involving the upper torso and arms. The presence of the reticular formation in the stem, and its role in consciousness, may provide an explanation for non-cardiac loss of consciousness.

Peripheral circulatory disturbance in the line of current traversal is seen especially in higher voltage injury, and may be akin to the phenomenon of keraunoparalysis *(vide infra)*. Peripheral and central autonomic disturbance leading to circulatory disturbance is seen, but must be distinguished from circulatory disturbance due to compartment syndromes following severe internal muscle damage.

In the short term following electrical injury (up to a small number of days) secondary neurological effects may be seen. These include temporary paralyses, pain of a neuritic kind, autonomic disturbance, and headache. Longer term effects are beyond our present scope.

Musculoskeletal response including burns

A significant part of electrical injury, as opposed to lightning injury, is the presence of internal muscle damage and skin contact burning.

The term "damage" rather than "muscle burning" is used deliberately. Until recently it was entirely accepted that internal muscle damage was due to thermal injury and was termed "internal burning". No doubt there is an element of internal thermal damage, but recent work sheds further light on the problem and has introduced the concept of *electroporation*. A more contemporary view recognises three forms of internal cellular damage. The first is electroporation, the second is electroconformational alteration of cellular proteins, and the third is

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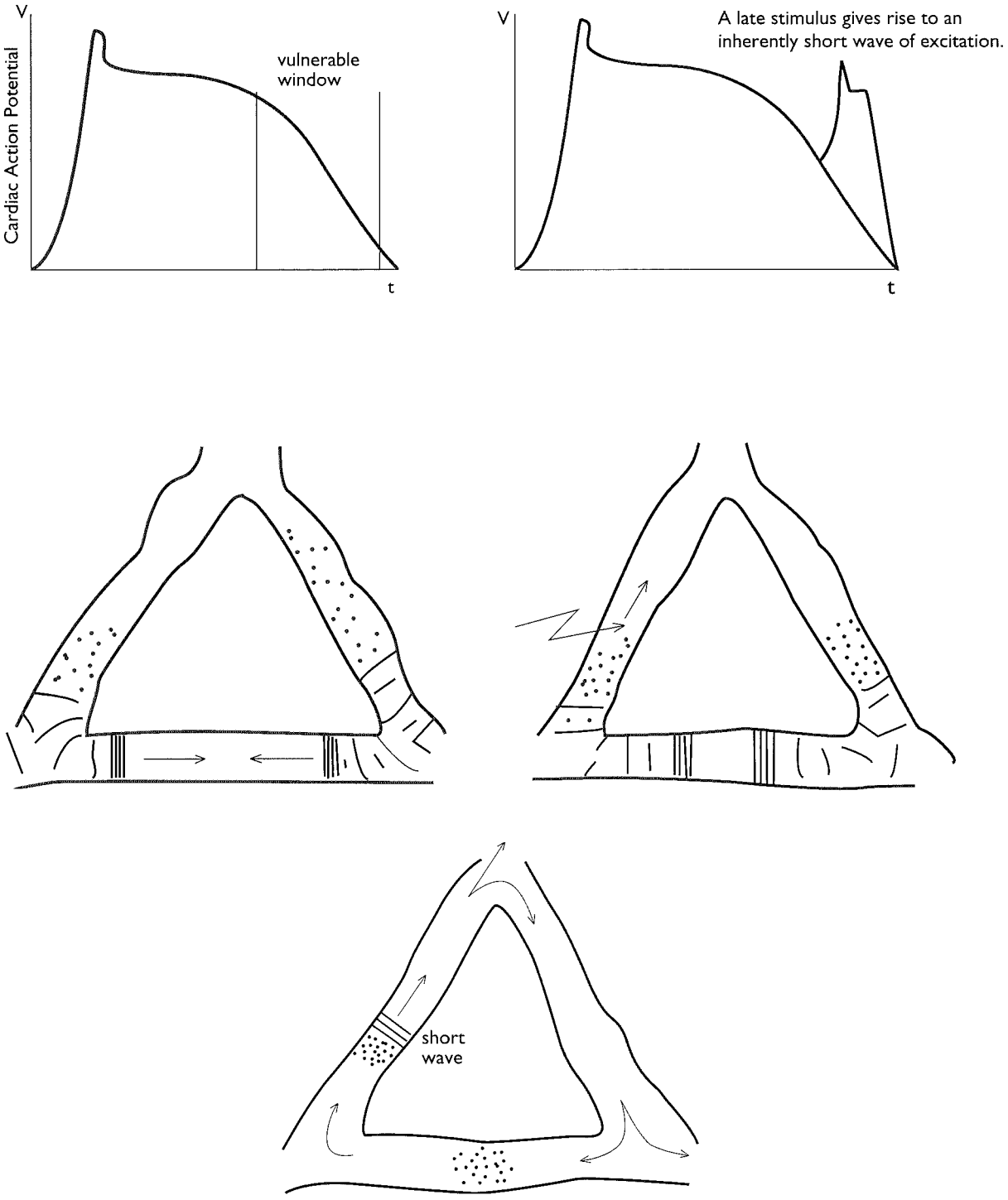


Figure 17.13 *The establishment of the vulnerable window. A normal wave of excitation spreads from superior towards extinction inferiorly. Stimulus occurring within a shortened wave of excitation can only travel retrogradely provided there is an area of depolarisation in the circle establishing a unidirectional block. This can occur in the early T wave of the ECG – the vulnerable window. (Adapted from Bridges* et al. *by permission.)*

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direct thermal heating. The net result of each of these mechanisms is cellular lysis and leakage of cellular contents due to membrane disruption. The central role of the cell membrane in these processes has only recently been highlighted.

Pursuing thermal damage first, small rises in temperature, even less than 10°C, can cause cell death. Disruption of cell membranes occurs at 42°C, protein bonding alters above 45°C, with proteins denaturing above 65°C. Protein changes are particularly significant for membrane proteins and, especially, for ion channel proteins. Before denaturation, conformational change is significant for ion channel function. Ultimately, cellular contents spill and cellular metabolism ceases. In the case of muscle cells, myoglobin enters the circulation with consequent toxicity for the kidneys.

Electroporation is a fascinating phenomenon where an applied electric field causes the formation of pores in cell membranes whose size relates to the magnitude of the applied field. This process has certain uses experimentally for the introduction of experimental substances into a cell. The pores, below a certain magnitude of applied field and, therefore, below a certain size, are self-sealing. Above a certain size, however, they are unstable, expand, and cause membrane lysis with consequent cell death. Theoretical biophysical analysis of the electroporation phenomenon have been undertaken, and the mathematically minded reader is left to pursue that aspect further.

An interesting consequence of both electroporation and membrane protein denaturation is the use of membrane sealing compounds in therapy of electrical injury. Initial success has been claimed using infused poloxomers – polymers of propylene and ethylene segments – which mimic biological surfactants.

A difficult aspect of traditional electric injury management has been the determination of the extent of internal damage, especially in muscle. In the past this information was necessary to determine the extent of any amputation for unsalvageable tissue, or simply for early debridement. Multiple and progressive debridement and amputation has been the norm as tissue damage demarcated. Many proposals for the use of dyes and tracers to determine levels of damaged tissue early in the injury course have been proposed with only partial success. Lee and colleagues, discoverers of electroporation, note that electroporated tissue appears macroscopically normal for a significant time after injury unless there has also been significant heating. This implicates electroporation rather than thermal effects in extensive tissue death. The possibility of membrane sealing therapy raises exciting prospects for the electrically injured.

In the past it has been noted that muscle damage can often follow a cone formation with the apex proximal and the long axis along a bone. It has been said that this is due to more significant thermal damage adjacent to high-resistance tissue which dissipated greater energy in the shock. This is an unsatisfying explanation as closer analysis (substituting Ohm's law into the power equation giving power = V^2/R) would indicate that for a given applied voltage power dissipation is inversely proportional to R and, thus, high-resistance tissues should dissipate less power than low-resistance tissues. Relative specific heat values need to be taken into account in addition, as does the time course of conduction of heat among the tissues. Electroporation may provide an alternative explanation in that the electric fields around high-resistance tissue may be higher than in low-resistance tissue and cause increased adjacent electroporation.

The above is a very simplistic qualitative introduction to electroporation, and the reading list at the end of the book provides access to more refined treatments.

In electrical injuries, the possibility of associated orthopaedic trauma due to prolonged muscle contraction must also be remembered.

LIGHTNING SHOCK

The cardiorespiratory response to lightning shock has been examined, as have theoretical explanations of its genesis. Of immediate concern in lightning injury are the neurological responses and aspects of trauma and burns.

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Immediate neurological concerns

Initially, the effects of lightning on the nervous system were thought to be thermal; however, recognising that lightning is such a brief phenomenon, internal heating is not thought now to be a significant factor, and indeed internal thermal damage of a type once postulated for electrical injury is simply not seen. Direct neural stimulation is now thought to be a major factor, and may well play a part in the asystolic cardiac arrest of lightning strike.

The syndrome of keraunoparalysis is enigmatic. The syndrome consists of cold pulseless cyanotic extremities presumably in the path of current conduction. A similar phenomenon is sometimes seen in electrical injury, particularly high-voltage injury, in the pathway of the current. Several factors, according to ten Duis, are thought to be involved in its genesis. First, direct stimulus of the neurovasorum peripherally, and with a possible central component, is thought to be involved. Secondly, a component of vascular spasm due to direct vascular smooth muscle contraction may be involved. Thirdly, it has been found experimentally that noradrenaline release in the injury is profound and this may contribute to vasospasm. Direct arterial wall endothelial injury may be involved with formation of thrombus. In any case the phenomenon is transitory and passes in anything from a few minutes to a few hours. If such a syndrome persists longer, then an alternative cause must be sought. No doubt a combination of causative factors may be involved.

Intracerebral pathology is seen in lightning injury with reports of both subdural and epidural haematomata. Vascular tears are seen at post-mortem and widespread petechial surface bleeding may be seen. CT findings indicate the lower regions of the brain to be most at risk, and the basal ganglia, cerebellum, and brainstem have already been mentioned. The postulated current pathways via CSF may account for this unusual distribution. Microscopically, neuronal abnormalities have been documented, such as chromatolysis of large cells, perineural and perivascular widening and separation, tears in cerebral substance and vessels, and, peripherally, "ballooning" of myelin sheaths.

The exact genesis of these changes remains to be elucidated, and gas liberation, electrolysis of water, electrostatic tissue separation, mechanical trauma, and fixation artefact have all been implicated.

Trauma and burns

Burns in lightning injury form only a small part of the injury and rarely require attention of great degree. That burns are a small part of the injury is often surprising to the layman, but it is probably a consequence of the very brief nature of the current passage, and therefore relatively low energy transfer to tissue bulk and skin. Burns are classified into six types.

1. *Full thickness entry and exit burns* – usually only small area, but may require debridement and grafting.
2. *Flash burns* – akin to welder's flash – from the intense ultraviolet emanation from the lightning channel.
3. *Contact burns* – burns due to contact with externally heated metal such as coins, buckles, necklaces, and so on – may require debridement and grafting.
4. *Keraunographic markings* – the tree-like fern markings seen transitorily on the skin, and which are said to be pathognomonic for lightning. They are connected with surface arcing of current and may also be seen in high-voltage shock when there is a significant component of arcing. Often termed "Lichtenberg figures", or "arborescent burns", they are thought to be generated by charge movement secondary to surface flashover.
5. *"Flower-like" burns* – thought to be similar to the keraunic marking, but due to surface charge of opposite polarity.
6. *Linear burns* – due to the passage of tongues of lightning arc across the skin surface thought to be along lines of low resistance (eg sweat lines). Thought to be thermal in origin and secondary to the formation of a superheated plasma by the arc. They usually require little treatment and form eschar which later sloughs. They are thought to indicate that surface flashover has occurred and are considered a good prognostic sign.

Examples of entry/exit and linear burns are shown in Plates 5 and 6.

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Trauma is not uncommon with lightning injury. Effects of blast on both abdominal and thoracic organs have been noted, and possibly are a factor in cerebral injury. Musculoskeletal injury must also be considered, with fractures and contusions both possible.

IMMEDIATE MANAGEMENT OF ELECTRICAL AND LIGHTNING INJURIES

The full management of electrical and lightning injuries has been outlined in detail elsewhere. Here, we briefly highlight aspects of primary management, and also aspects arising out of the previous discussion. A significant component of the immediate management of these injuries is the management of cardiac arrest, whether asystolic or fibrillatory. This management is covered elsewhere in this volume (q.v.) and will not be repeated here.

Electrical injuries

The initial on-site management of electrical injury requires an immediate and thorough assessment of the circumstances of the shock. It must be ascertained as a priority that a victim is safe to touch, for a victim in contact with electric current will form part of a circuit with a person contacting him or her and current will flow to the rescuer. One victim will quickly be transformed into two. If at all possible, power should be switched off at the most accessible main switch to the installation concerned. It is the business of on-site personnel to be aware where that switch is. It is the business of system designers to ensure that such switches are readily and quickly accessible from all parts of an installation. Removal of a victim from contact with live conductors may be extremely difficult due to the let-go phenomenon, and is not to be recommended without the exercise of the greatest caution if indeed at all, and the certainty that adequate insulation can be provided between rescuer and source of current. The higher the voltage involved, the greater the care that should be exercised. Unless it is immediately and easily possible, the switching off and/or disconnection of any source of current above household voltage level should be left for appropriate authorities. These principles may mean that a person is left in contact with electric current for a prolonged period because it is simply unsafe to effect removal. For rescuers this is a highly distressing circumstance and will mean loss of life. Unfortunate though this undoubtedly is, loss of a second life only compounds the distress, and this must be appreciated.

Once the victim is separated from the current, he or she is safe to touch. There is no residual charge or injuring agent "stored" in the body. This myth, emphasised as such by Cooper, has been responsible for loss of life. The need for CPR should be assessed immediately and instituted without delay if indicated. Whether unconsciousness has occurred or not, and whether cardiac arrest has occurred or not, a victim should be transported to medical aid, preferably the emergency department of a hospital. Related injuries, for example fractures or lacerations, should be given appropriate primary treatment prior to transport.

At the hospital, definitive management continues. Tetanus prophylaxis should be given if required and an ECG performed. Associated trauma should be treated according to its own priority. Ventilation may be necessary, as may inotropic support (medication to enhance cardiac muscular contraction). A victim should in general be monitored for a period.

It is controversial how long an otherwise asymptomatic person with a normal initial ECG should be monitored for cardiac arrhythmiae. The author recommends that at least 6 hours of cardiac monitoring should be undertaken on all patients with an electrical injury and a normal ECG on presentation. If at the end of a 6-hour period a repeat ECG is normal and patients are still asymptomatic, then they may be discharged into the care of a responsible adult for at least 48 hours with a graded return to normal activity over one week. In assessing the normality of the ECG, the more subtle signs must be sought, for example, changes in PR or QT intervals and QRS width, as well as more obvious signs. If the presenting ECG is abnormal, or the repeat ECG at 6 hours is abnormal, then the abnormalities should be treated on their own merits. It should be remembered

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that delayed arrhythmiae can occur. The author suggests that the period up to 72 hours is the most critical time for this occurrence. Some writers report delayed arrhythmiae up to 3 months following an accident, but this would seem to be excessive.

Therapy for burns should be commenced, and this is dealt with elsewhere in this volume.

Lightning injuries

By contrast, primary management of lightning injuries is relatively straightforward. There is no electrical reason why a lightning victim cannot be touched immediately, and they represent no danger to a rescuer. Artificial ventilation and/or external cardiac compression is paramount if cardiorespiratory arrest has occurred. This should be instituted without delay and kept up expectantly until transport to definitive medical help occurs and treatment as an inpatient is obtained. All lightning victims should be admitted to hospital and monitored on the same basis as electrically injured patients. Associated trauma should be dealt with.

There is a notion in the literature that all body metabolism ceases after lightning strike and, therefore, one has longer than usual before instituting CPR with the expectation of good outcome. This notion is specifically discounted by the author, and the institution of CPR in the lightning injured is just as urgent as with any other case of cardiac arrest. None the less, resuscitation of the lightning victim should be expectant, as mortality from the injury is low. Cooper has made the point that if cardiorespiratory arrest has not occurred in the initial strike, then survival is almost guaranteed. Priority attention should, therefore, be directed to "the dead" after a lightning strike as significant numbers of asystolic patients after lightning strike can be saved. This is not necessarily in accord with the usual rules of triage.

PROTECTION FROM LIGHTNING AND ELECTRICAL SHOCK

Electrical shock

A safe approach to dealing with electrical apparatus can prevent many injuries. The following guiding principles should be adopted.

Never interfere with, adjust or repair any electrical apparatus unless qualified to do so. Similarly, never install or manufacture electrical apparatus unless appropriately licensed. This particularly includes domestic installations, making of extension leads, and "running repairs" to electrical equipment. Leave all protective devices (fuses, circuit breakers) intact and never bypass them. If they malfunction, a reason must always be sought.

Never work on, or interfere with, live conductors. In dealing at more than operator level with electrical equipment, always disconnect it from the power source first. If tradesmen work on live apparatus, then established protocols must be followed. These are well known in the industry.

The use of RCDs (residual current devices) is strongly advised. These are devices which sense active and neutral current simultaneously; any difference implies current being diverted to earth, possibly through a person, and in this event power is automatically interrupted to the protected circuit.

If conductors are encountered in the course of daily activities, they should be considered live and dangerous until absolutely proven otherwise by qualified personnel. They should not be approached, and certainly not touched. Appropriate licensed inspection should be arranged, and in the intervening time a watch should be mounted to prevent accidental contact by others.

Lightning shock

When planning outdoor activities a weather forecast should be obtained first. At signs of an approaching storm shelter should be sought. A car or a substantial building makes a safe shelter. If unable to reach shelter, a person should crouch down in as low a profile as possible, separated from companions. The minimum ground contact should be achieved. To shelter under a tree is an unsafe practice.

To assess the imminence of a lightning strike, it is possible to time the interval between lightning being

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seen and its associated thunder being heard. Sound travels 1 km in 3 seconds. When the time to thunder is 30 seconds, the lightning is 10 km away and shelter should be sought. When the time to thunder is 15 seconds or less (5 km or less), shelter is an urgent priority.

During a storm, those indoors should not use a telephone or operate electrical apparatus.

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CHAPTER EIGHTEEN

EYE INJURIES

Lawrence W. Hirst

Eye injuries occur either as isolated events to a small localised area of the body, or as part of a more complicated form of trauma in which the eye is a "passenger" and injured along with many other sites, particularly the face and the brain.

In either situation, injury of the eye is usually obvious on careful inspection, although the overall extent of the injury may become masked by damage and clouding in the front of the eye preventing adequate examination of the back of the eye. The more difficult situation is where the eye injury is associated with lid and facial injury, and where even simple examination of the eye may become difficult because of lid swelling, and mucus and blood discharge. Finally, in the face of significant eye injury, the usual neurological signs derived from the eye, such as pupil reactions, vision, and eye movements, may be misleading or difficult to interpret, masking the picture of significant brain damage. First aiders and paramedics have a major preventive role in limiting further damage following eye trauma.

Trauma to the eye takes many forms. Penetrating sharp objects, contusion from blunt objects, foreign bodies, chemical burns and acute radiation exposure from sun, snow or welding equipment – all may involve the first aider, paramedic or doctor before they come to the ophthalmologist.

In this chapter, many of the processes described, for examination and initial care, go well beyond the role of the first aider. (For example, it is not part of the first aider's role to apply lignocaine to the eye, or to use a 21-gauge needle to remove a foreign body.) They are included here because an understanding by the first aider of the broad nature and purpose of these paramedical, medical and ophthalmological processes will help to convey an appreciation of the rationale for the standard first aid procedure; this in the main amounts to observance of the principle "protect from further harm".

One of the most important aspects of pre-hospital care is to be able to examine the eyes, and measure visual function in a simple yet accurate way.

EXAMINATION

Visual acuity

Vision is by far the most sensitive monitor of dysfunction of the eye. With this in mind, it is vital to be able to assess this in a systematic, reproducible and accurate fashion. It is important also to record the results of vision testing to avoid the possibility of subsequent claims that the examiner was the cause of the reduced vision when it was actually the result of the injury. This is particularly relevant when the examiner also undertakes treatment, such as foreign body removal, which may be wrongly blamed for subsequently discovered reduced vision.

The testing of visual acuity should be performed with a standardised Snellen Chart at 6 metres, with each eye tested separately, preferably with the appropriate distance spectacles (those worn for TV or driving) (Figure 18.1). If the vision is not close to 6/6, then a pinhole should be used in front of the eye being tested while the other eye is occluded using a card or similar object (Figure 18.2). The pinhole will generally improve

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Figure 18.1 *Recording vision in right eye with left eye occluded (without a pinhole).*



Figure 18.2 *Recording vision with left eye occluded and a pinhole in front of right eye.*



Figure 18.3 *Recording vision with finger counting.*

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the visual acuity in the presence of any refractive errors, such as astigmatism or myopia. The vision should then be recorded. The recording of vision utilises a ratio of 6/? where the denominator is the number of the smallest line on the Snellen Chart which the patient is able to read at 6 metres. The numerator reflects the distance at which the patient is being tested, which is usually 6 metres.

If the patient is unable to see any of the letters on the chart at 6 metres, then, rendering assistance, walk the patient towards the chart, while occluding the eye not being tested. Note when the patient can first identify the largest letter on the chart; if, for example, at 2 metres, the vision will then be recorded as 2/60. If the patient is unable to identify the 6/60 letter at 1 metre, then sit the patient down and see if he/she is able to count the number of the examiner's fingers held 1 metre from their face (Figure 18.3). If so, "count fingers at one metre" should be recorded in the chart. If they are unable to identify the number of fingers, then a hand should be moved slowly in front of their face at 1 metre, still occluding one eye, and a positive response should be recorded in the chart as "hand motions at one metre". If this is still negative, carefully bunch up some tissues over the uninjured eye and hold them firmly against the nose and around the bony rim of the orbit to prevent any stray light entering the eye, then darken the room entirely and shine the brightest light available into the injured eye requesting the patient to respond when they see the light and when it goes away (Figure 18.4). Accurate responses to this should be noted down as "light perception" on the chart. The inability to identify even the strongest light in the eye should be noted down as "no light perception" and carries with it an ominous prognosis for any recovery of useful vision.



Figure 18.4 *Recording vision in right eye with light, while occluding left eye.*

In the first aid situation, where there is of course no Snellen Chart, the first aider may be able to record some indication of the casualty's visual acuity by asking them whether they can see fingers held in front of each eye separately; if so, how many, and if not, whether they can see movement of the fingers. Failing that, see whether they can detect the light from a torch shone into the eye.

Pupil reflexes

The pupil response is also of vital interest, although it is frequently impossible to test in the face of significant intraocular damage. The importance of the pupil response with respect to unilateral eye injury is the observation of an *afferent pupil defect* using the swinging flashlight test. The light from a flashlight is swung rhythmically with both eyes held open, from one pupil to the other leaving only a second or so between the presentation of the light to each eye (Figure 18.5A and 18.5B). The pupil of the eye, on which the light is being shone, is carefully observed for the initial reaction of the pupil. In normal situations where the front part of the visual pathway is intact in both eyes, there should be a

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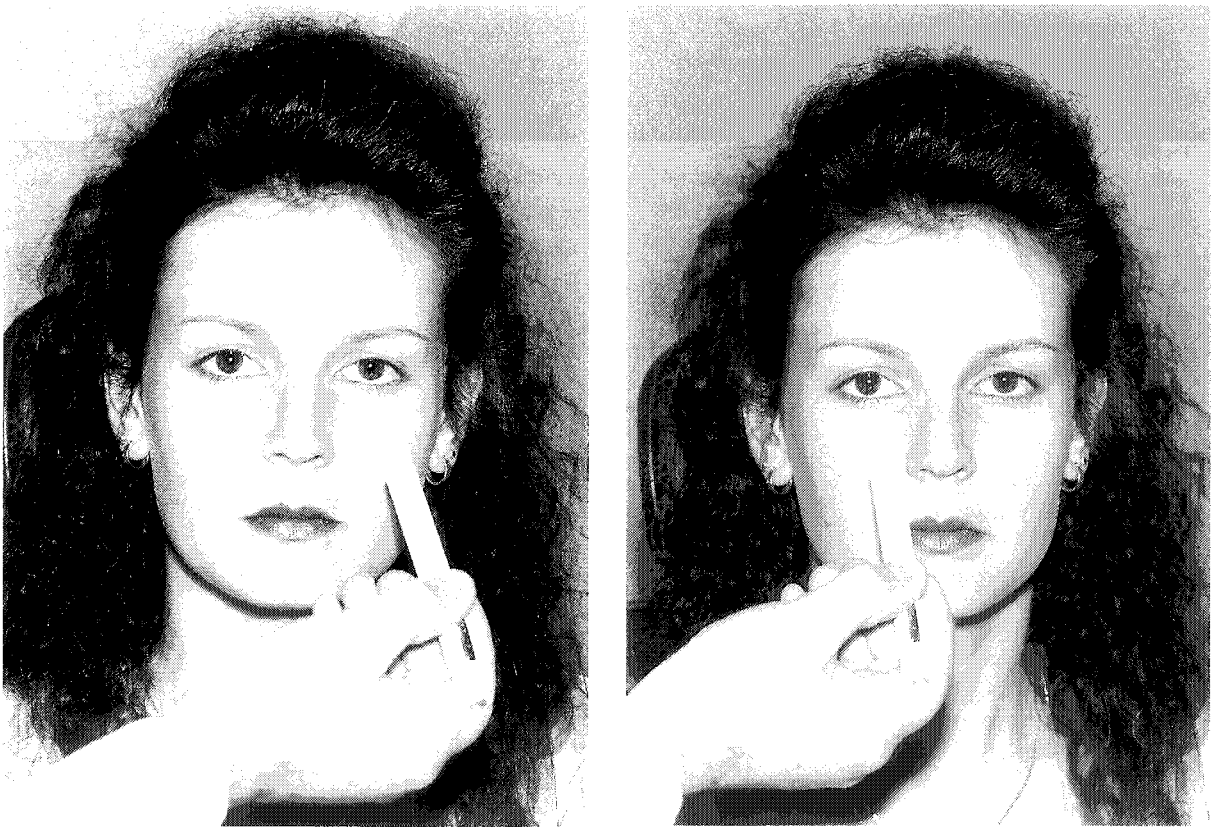
small constriction. *Any* initial dilatation is a sign that this eye has a reduced afferent or visual pathway with respect to the other or normal eye. This is a reliable and objective sign that there has been significant damage either to the retina, the optic nerve or optic chiasm. This is particularly useful when all external examination and even internal examination of the eye suggests there has been no damage.

Eye movements

Eye movements should be checked by asking the patient to follow an object, a light, etc. in all nine principal directions of gaze: up, down, left, right, up and to the right, up and to the left, down and to the left, down and to the right and straight ahead. An asymmetry of movement in the injured eye must be explained by swelling of the orbital contents, a weakness or paralysis of one or more of the muscles, or an entrapment of one of the muscles in a break in one of the bony orbital surfaces.

External examination of the eye

More injuries are missed by incomplete external examination of the eye than for any other reason. It is desirable to examine the eyes with some form of magnification, preferably an operating loupe or, if necessary, a magnifying glass, together with a strong focused beam of light close to the injured eye (Figure 18.6). Although this can be done by one observer, it is frequently helpful to have one person hold the lids apart



A B

Figures 18.5 A and B *Swinging flashlight test.*

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and shine the light on the eye, while the other person examines with the magnification source. In holding the lids apart, particularly where injury to the eye is suspected, it is of critical concern that no undue pressure is exerted against the globe itself. In the situation where the globe has been seriously injured with a through and through perforation or a foreign body inside the eye, undue pressure on the eye can cause expulsion of intraocular contents, such as vitreous humour, retina or the lens, and result in an irreparable situation. Because of this, the lids are held firmly against bone, well away from the eye. The upper lid is held against the frontal bone, or the bone of the forehead, and the lower lid is held against the maxilla, or the cheekbone. One can then say definitively that there is no direct pressure being applied to the eye, when these bones are felt beneath the fingers. If the patient is then asked to look in all principal directions of gaze, a complete examination of the front part of the eye can be undertaken. This should disclose any damage to the front of the eye. Any disruption of the superficial epithelial lining on the surface of the eye will be seen as a brilliant green-staining area, after adding a drop of 1 per cent fluorescein and 0.25 per cent lignocaine (available as sterile single-dose minim), and using a cobalt filter over the light source.

Ophthalmoscopy

The use of a direct ophthalmoscope to examine the inside of the eye is vital, but requires someone trained in



Figure 18.6 *Examination of eye with a focused beam and a magnifying lens.*

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examining the *normal* eye with a similar instrument. It will permit observation of opacities inside the eye, even if the external eye appears entirely normal.

Visual fields

On occasion, examinations of the visual fields to confrontation may be useful. This requires seating yourself approximately one metre from the patient with eyes on the same level and having the patient occlude one eye with one hand and using opposite eyes (ie your left eye for their right eye) for the confrontation test. The patient should look at your eye and you should look at their eye, carefully watching them for any loss of fixation, present your two hands in the extreme of the field as you see them nasally and temporally. They may count fingers or just watch for the presence of the hands. This can be repeated in the mid-field at the horizontal level, and above and below, presenting the fingers simultaneously in both temporal and nasal fields each time (Figure 18.7).

Use of drops

For those medically qualified, local anaesthetic drops can be used as a diagnostic aid. These may make it possible to separate the lids and examine the eye more fully in a patient with significant pain as a result of eye damage. Where there is concern of combined eye/head injuries, it is ill-advised to dilate the pupils to examine the back of the eye unless coexistent head injury has been ruled out.



Figure 18.7 *The testing of visual fields to confrontation.*

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Eversion of upper lid

Eversion of the upper lid is essential in patients complaining of foreign body sensation or in whom scratches can be seen by blue (cobalt) illumination of the fluorescein-stained ocular surface. Foreign bodies, especially sawdust, frequently lodge on the conjunctiva of the upper tarsal plate. This test is best performed by grasping the upper lashes firmly while the patient looks down, using a fulcrum at the upper tarsal plate, such as a cotton-tipped applicator, and pulling the lid forward and up over this fulcrum and holding the lid carefully by the lashes without touching the conjunctiva. The area under the upper lid then can be examined for any foreign bodies or occult injury. Any smaller foreign bodies that may have accumulated mucus will fluoresce brightly if fluorescein had been instilled prior to eversion.



Figure 18.8 *Removing a hard contact lens with a suction cup.*

INITIAL CARE

Contact lens removal

The removal of a contact lens is relatively straightforward but should not be undertaken if the surface of the eye is obviously badly injured with a through and through laceration. In this case, the contact lens is best left in place until an ophthalmologist is able to see the patient. If one is medically qualified, removal of a soft contact lens is accomplished after the instillation of a drop of local anaesthetic. The contact lens is moved over onto the sclera with a finger tip, and then gently pinched off the surface of the eye between thumb and index finger, and placed in some balanced salt or normal saline solution. A hard contact lens is most easily and best removed using a small vacuum cup (Figure 18.8).

Taping of lids

The lids may require taping as an emergency procedure in unconscious or comatose patients to avoid exposure of the eye, which will lead to drying and may result in infection and perforation of the eye within a 24-hour period. This is best performed using a transparent sticky tape found in most offices. One to one and a half inches of the tape is cut, and placed on the upper lid skin vertically at the mid-point of the upper lid across the upper lashes; the lids are then closed and the remainder of the tape is carefully applied to the lower lid over the cheek. Examination of the lid aperture with a light should confirm that no part of the eye is visible.

Foreign body removal

Many foreign bodies do not penetrate the eye and simply sit on the conjunctival membrane. If practical, these should be simple to remove, using extreme gentleness and care. If they are adherent, and if one is medically qualified, removal of a superficial conjunctival foreign body should be undertaken under topical anaesthesia, with the head well supported and the patient in a supine position. With the patient staring straight up at the ceiling and the lids held by an assistant, a cotton-tipped applicator, moistened with an anaesthetic drop, is wiped across the conjunctiva with the foreign body being removed as it adheres to the cotton tip (Figure 18.9). A superficial corneal foreign body can be removed similarly. On occasion careful use of a 21-gauge needle, (Figure 18.10) with the bevel

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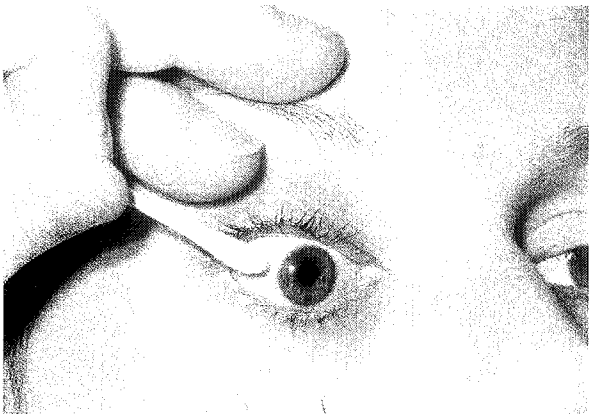


Figure 18.9 *Removing a conjunctival or corneal foreign body with a cotton-tipped applicator.*

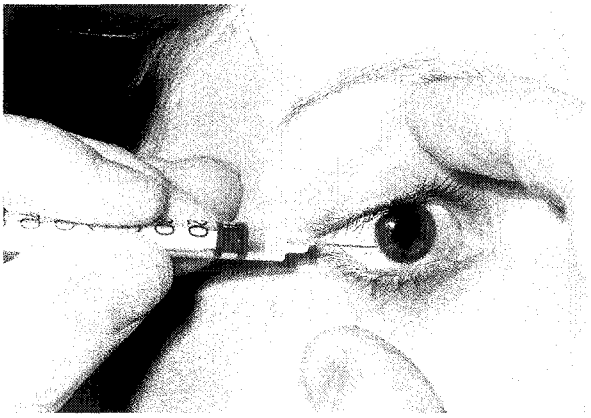


Figure 18.10 *Removing a superficial foreign body with a needle.*

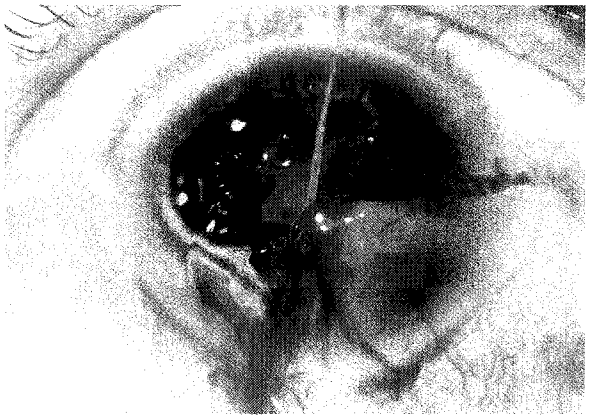


Figure 18.11 *A large perforating injury with prolapsed iris.*

uppermost and held tangential to the corneal surface, can be used to flip out a superficial foreign body. The third possible method is the use of a sterilised dental burr, which can be held perpendicular to the surface of the cornea. Rotation of the burr will usually dislodge a superficially embedded foreign body. If there is any doubt about the depth of the foreign body, or possibility of occult perforation of the eye, no attempt should be made to remove the superficial foreign body.

Pressure patch

A pressure patch is designed to minimise movement of the eye under the closed lids and therefore to aid in the healing of epithelial abrasions, or after superficial foreign body removal, and to significantly reduce pain. A pressure patch should *never* be applied when there is a suspected perforating injury of the eye, significant blunt injury or chemical injury to the eye. The application of a pressure patch requires some practice and consists of the following steps.

An antibiotic ointment is placed in the lower cul-de-sac of the eye and the skin above the eye and over the cheek is carefully cleaned with an alcohol pad. The patient's head is supported against a wall or on a bed and the patient is requested to keep both eyes closed during the entire procedure. A single pad is folded in half and placed with the crease underneath the superior orbital rim and a single pad placed horizontally across this folded pad. A piece of 1-inch Micropore tape, approximately four inches in length, is stretched across the pad and applied firmly to the skin above the eye and the skin over the cheek. Overlapping strips of tape should be applied, usually five or six, to complete the pressure patch.

The patch should be removed at 24 hours for review. If the eye is not entirely healed at this stage and the vision has not returned to normal, the patient requires immediate referral.

First aid for sharp injury

After obtaining a history, and taking the vision, the eye should be carefully examined as previously described. Even where there is concomitant facial injury or head

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injury it should be possible to examine the eye and determine if there is significant damage.

Examination of the lids and the eye may reveal lacerations of the skin or less obvious lacerations of the surface of the eye. Ominous signs are the protrusion of iris through a laceration site (Figure 18.11), blood inside the eye, or a soft eye. Foreign bodies may be seen on the surface of the eye, protruding into the eye or within the eye itself. Instead of having a regular, black, round, pupil, a traumatic cataract may be present, seen as a white pupil. Even in the absence of these external signs of injury, occult perforating injury may have occurred in the back of the eye which can be recognised only as a reduction in visual acuity or by ophthalmoscopy.

After assessment of the eye, it should be protected with a plastic or metal shield, taped so that the shield is resting on the nasal and frontal bones and the maxilla, vaulting over the eye (Figure 18.12). This is to protect the eye from any external force including the patient's own fingers. If the patient has an isolated eye injury and can be transferred to a location for definitive treatment by an ophthalmologist within 2–6 hours, road transport is satisfactory. During this time, no pads should be applied to the eye, and the patient should avoid straining if at all possible to prevent increased intraocular pressure, secondary to vascular engorgement of the eye and possible expulsion of intraocular contents through a laceration in the surface of the globe. Definitive surgical treatment may need to be undertaken within a few hours of examination so the patient should not be allowed to eat or drink. Most significant injuries of the eye require operative repair under general anaesthesia.

Although tetanus has rarely been described as a result of isolated perforating injuries to the eye, tetanus toxoid should be administered if the patient's vaccination status is unknown.

The patient is reassured and no drops are put in the eye, apart from the one drop of topical anaesthesia, if necessary, to examine the eye. If an intraocular foreign body is suspected, samples of the implement or object responsible may be sent with the patient, so that the metallic nature of the possible intraocular foreign body and its magnetic properties can be tested. This is not of vital importance and should only be undertaken if the material is small and readily to hand. Protruding intraocular foreign bodies which penetrate to the inside of the eye should generally be left in place and covered with a shield, or if this is not large enough, or in situations where a shield is not available, even a polystyrene coffee cup can be inverted over the foreign body and the globe and foreign body protected with a shield. The foreign body should be sent with the patient.

Where transport by road of the patient is likely to take more than 6 or 8 hours consideration may have to be given to evacuation of the patient by air. Because of



Figure 18.12 *Correct position for protective plastic shield.*

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the possibility of gas bubbles within the eye after perforating injuries, it is important that the evacuation take place in a pressurised aircraft or, if not, below an altitude of 6000 feet.

Definitive therapy may be given to corneal abrasions, corneal and conjunctival foreign bodies and subtarsal foreign bodies. As described earlier, fluorescein will demonstrate vividly any area of abrasion on the surface of the eye as a well defined, brilliant green-staining fluorescent area. If no penetrating or severe injury of the eye is suspected and the eye has been scratched by a fingernail, a piece of paper etc., or by removal of a superficial foreign body, then a pressure patch may be applied to the eye. This is the only circumstance in which a pressure patch should be applied to the eye, and this form of treatment is to be avoided if there is a suspicion of any more significant injury to the eye. The application of a pressure patch to an eye which has had a severe blunt injury, or a perforation of the eye, may lead directly to loss of the eye through expulsion of intraocular contents.

Corneal and conjunctival foreign bodies are treated as previously described, a pressure patch may be applied, and the patient reviewed at 24 hours. If the defect has not healed entirely or the vision is not back to normal, then the patient should be referred.

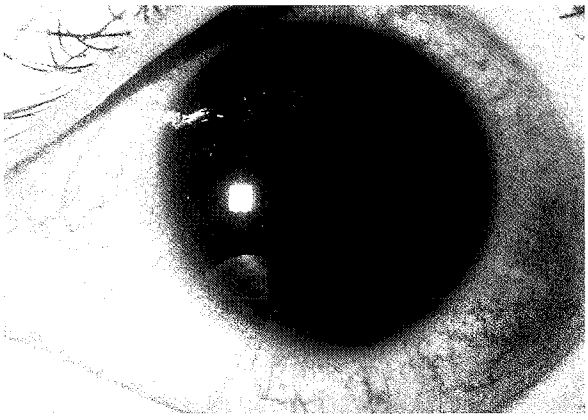


Figure 18.13 *Total "eight-ball" hyphaemia concealing all view of underlying iris details.*

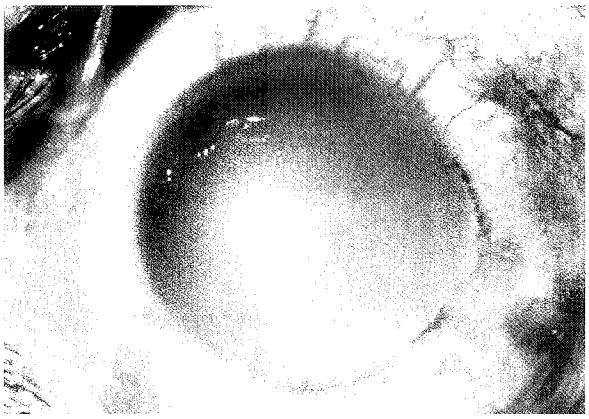


Figure 18.14 *Severe chemical burn showing opacification of cornea and blanching of conjunctiva.*

First aid for blunt injuries

Once again, a history should be taken and the vision tested prior to any examination or treatment. The appearance of intraocular blood (hyphaemia) (Figure 18.13), a soft eye or an irregular pupil are certain signs of a severe blunt injury. Even in the absence of these signs, severe trauma to the back of the eye may only be noted by the reduction in visual acuity, or examination with an ophthalmoscope. Confirmation that significant blunt injury has occurred should result in protection of the eye with a shield and immediate referral.

First aid for chemical injuries

When the patient is seen within minutes of a chemical injury the testing of visual acuity may be delayed. It is imperative that the eye is flushed out vigorously as soon as possible.

The sign of a very severe burn to the eye is a relatively white eye where no vessels in the conjunctiva can be seen and the cornea is so opacified that details inside the eye are obscured (Figure 18.14). This generally is an eye with an extremely poor prognosis as a result of severe chemical burns, usually alkaline in nature.

A drop of topical anaesthesia is instilled into the eye or, if this is not available, irrigation is undertaken

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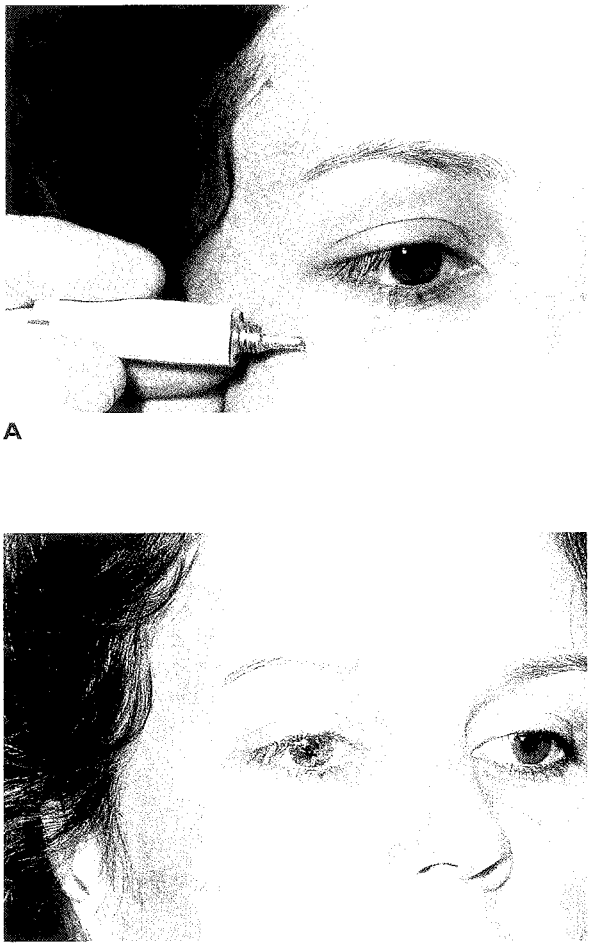
without topical anaesthesia. It is preferable to use sterile normal saline solution, but the critical issue is to use whatever fluid is available for copious irrigation, without delay. The surface of the eye is flushed for at least 10–15 minutes and the upper lid is everted and particulate matter wiped off with a cotton-tipped applicator and also irrigated off. It is important also to pull the lower lid well away from the eye and irrigate the inferior cul-de-sac between the lid and the eye (Figure 18.15).

If litmus paper is available, it may be used to test the tear fluid on the surface of the conjunctiva after irrigation to see that the pH of the surface of the eye is neutral.

Any particulate matter that has been removed, or containers which describe the chemical which has contaminated the surface of the eye, should be sent with the patient and the patient should be referred immediately. The damage from most chemicals, but



Figure 18.15 *Irrigation of the eye with an intravenous giving set.*



A

B

Figures 18.16 A and B *Ointment applied to skin around eye* ***A****, and then plastic wrap sealed to the skin over the eye* ***B*** *to create a hermetic seal.*

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particularly from alkalis, to the surface to the eye, is virtually instantaneous, but certainly occurs within the first seconds to minutes. Because of this, *immediate* irrigation under a tap, or with whatever fluid is readily available, is imperative.

First aid for exposure keratitis

Any patient who is unable to blink their eyelids because of local facial damage, facial nerve damage, or because of their conscious state, should have the surface of the eye protected from drying out by adequate taping of the lids together. If the eyelids are held apart on a normal eye, the surface of the eye will dry out within 20–40 seconds. There will be a defect in the surface of the eye within 10–15 minutes and an ulcer and possible infection of the eye within a few hours, followed by perforation of the eye within 24 hours. This may be avoided by early taping of the lids closed, as described before. The tape may be taken down at any time to observe the pupils, but if the patient is in a situation where pupils need to be monitored constantly it is possible to protect the exposed eyes from drying without closing the lids. Sterile eye ointment is placed on the skin surrounding the bony orbit (Figure 18.16A) and then a piece of clinging plastic wrap is placed over the eye and fixed to the skin using the ointment (Figure 18.16B). It should be observed that tears from the surface of the eye will condense on the inside of this membrane creating a humid environment which will prevent drying out of the eye and yet permit observation of the pupils. This is a second-best option only, compared to the use of tape to close the lids.

CONCLUDING COMMENT

The foregoing is a brief account of the key elements in the examination of, and the early care for, eye injuries. It includes some aspects, such as the copious irrigation of the chemically injured eye, or the use of plastic wrap to protect the unblinking eye from drying out, which are well within the scope of the first aider. Some of the other procedures described above require a higher level of expertise or equipment, but all carry important implications for the first aider: the need to reassure the casualty, in what for them may be a particularly frightening situation; the need to record as far as possible the defects in their vision when first encountered; and above all the need to protect their injuries from further damage until such time as they are in expert care.

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CHAPTER NINETEEN

FRACTURES

Villis Marshall

The pre-hospital management of fractures is a common and very important part of first aid. The diagnosis, stabilisation and transportation of a patient with a fracture can be one of the most rewarding aspects in the first aider's professional life. The first aid drills and skills to manage the victim with a fracture are the same whether the care giver is a volunteer first aider, a salaried paramedic or a doctor who has stopped at the scene of an accident. This chapter provides some of the underlying biomedical detail and pathophysiology which explains why fractures occur, and gives a basis for the current doctrine of fracture management.

Fractures challenge the first aider. The challenges are to diagnose them quickly and to prevent broken bones further damaging surrounding soft tissues, and to treat the whole patient whose fracture is usually just one aspect of his or her plight.

It is important to recognise that long bone fractures in particular (Figure 19.1) result in significant blood loss, often as much as 1–2 litres, and by carefully immobilising these fractures, the patient will be more stable when they reach a hospital emergency department. Thus, good fracture care is a very important part of good first aid care of the whole patient.

CAUSE OF FRACTURES

What is a fracture? A fracture is simply a break in the continuity of a bone.

What produces a fracture? A fracture can be produced either by direct or indirect force, or by muscular action.

How a fracture can be produced by direct force is obvious, but the role of indirect force in this process is sometimes forgotten (Figure 19.2). The recognition that indirect force can play a role is particularly important when one is assessing a patient. For example, if a casualty has fallen from a height and landed on their feet, a fracture around the ankle may be obvious, but what is not so obvious immediately is that they have also suffered a crush fracture of their lower lumbar vertebrae.

Biomedical properties of bone

Bone is a two phase material that consists of bone mineral (hydroxyapatite) and matrix (mostly collagen). The tensile strength of bone, 140 meganewtons per square metre (MN/m^2), is largely due to the matrix component and the compression strength of 200 MN/m^2 is due to the mineral component. The hydroxyapatite crystals are arranged in discrete units. This means that a crack traversing a crystal will meet an interface resulting in a T-shaped crack. As a result of this arrangement, energy will be dissipated and to some extent this arrangement will prevent the further extension of the crack. The importance of the mineral component is further supported by the observation that a bone which is 70 per cent mineralised is three times stronger than a bone which is only 63 per cent mineralised (Harkess *et al.* 1984).

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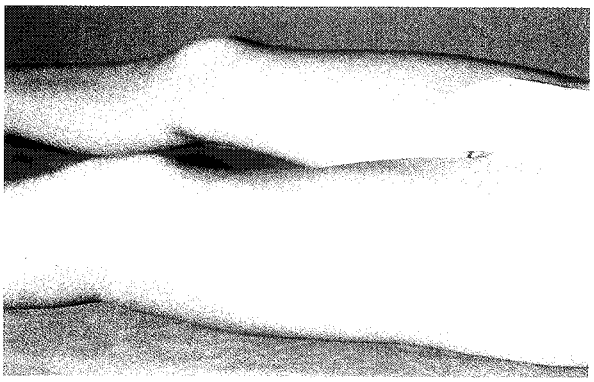


Figure 19.1 *Closed fracture of the femur, with swelling due to the significant loss of blood into the surrounding tissues. (Unless otherwise stated, all photographs in this chapter courtesy of Flinders Medical Centre.)*

Biomechanics of fractures

Whether a bone will break will usually depend upon both extrinsic and intrinsic factors. The extrinsic factors that are important causative agents are the magnitude of the force as well as the duration and the direction of the force applied to a bone. The intrinsic factors that determine whether a bone will stand the extrinsic factors were detailed many years ago by Evans (1961). These include the bone's energy-absorbing capacity, modulus of elasticity, fatigue strength and density. A bone can absorb energy by changing shape; thus, the more rapidly a bone is loaded, the greater the amount of energy absorbed prior to fracture. Consequently, if a



Figure 19.2 *Fracture of the body of the fourth cervical vertebra. This type of injury is due to flexion type injury and results in an unstable fracture.*



Figure 19.3 *Comminuted fracture of the proximal phalanx of the thumb, showing a number of fragments*

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force is applied slowly, the fracture will tend to be linear, whereas if the bone is loaded rapidly, an enormous amount of energy is infused into the bone resulting in an "explosion" in the bone with a comminuted fracture resulting (Figure 19.3). Frankel and Burstein (1970) found that the energy required to fracture a femoral neck in an experimental situation was 60 kg.cm. Undoubtedly, kinetic energy well in excess of this is produced in falls, but this can be dissipated by musculature and other soft tissues which prevent a fracture; the loss of these tissues in older people, as well as other factors such as osteoporosis, tend to make them more susceptible to fractures from relatively minor falls.

Fractures as a result of muscular action are relatively uncommon and, when they do happen, frequently involve bones such as the patella. Fractures produced by this mechanism tend to be more frequent in elderly people whose bones may be weakened by osteoporosis (a loss of the normal mineral content of the bones).

What happens when bone is broken?

The same process occurs irrespective of the site of the fracture; however, if the major long bones are involved, the magnitude of the response is much greater. As a result of the fracture, small and often large blood vessels can be disrupted, and bleeding occurs at the fracture site with the formation of a large haematoma (blood clot). The damaged ends of the bone will eventually die because of disruption of the blood supply. Normally, if it is a relatively simple fracture then only a small amount of bone on either side of the fracture will be involved but, if the bone is broken into a number of pieces and there are large fragments, some of these fragments may be devitalised and die. The response that occurs after a bone has been fractured can be divided into the inflammatory, reparative and remodelling phases. While the time taken for a bone to heal varies from fracture to fracture, in general about 10 per cent of the healing process is occupied by the inflammatory phase, 30–40 per cent by the reparative phase, and 60–70 per cent by the remodelling phase. Obviously, there is overlap between these phases, which is why the summation of the percentages exceeds 100 per cent.



A



B

Figures 19.4 A and B *This 14-year-old boy was kicked on the chin by a horse.* **A,** *taken under anaesthesia, shows the point of impact of the horse's hoof. However, as* **B** *shows, the mandible has sustained several fractures, with the middle segments "floating free" – a comminuted fracture. (Photographs courtesy of Dr Caroline Acton.)*

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The inflammatory phase is characterised by the migration of white blood cells, or inflammatory cells, from the surrounding capillaries into the blood clot. As well as the inflammatory cells, which come from the small blood vessels, cells will also migrate in from the sheath of tissue (periosteum) that covers the bone. The inflammatory cells, in particular cells called "macrophages", have the ability to break down and remove both blood clot and damaged cells, particularly muscle cells that may also have had their blood supply impaired at the time the bone was fractured. Concomitantly, new capillaries start to grow into the area as this is a very important step to ensure that adequate oxygen and nutrients are readily available. If this does not occur, then the repair process will either be delayed or not take place at all.

This process will be adversely affected if the bone ends are not adequately stabilised. Movement will cause disruption of the new vessels that are formed as part of the healing process, and this will in turn result in a delay in this process.

Following the ingrowth of the blood vessels, a callous starts to form. This callous is formed from specialised cells that are derived from the bone and have the ability both to form cartilage and to lay down new bone. As well as the cells that form these new tissues, other cells called "osteoclasts" appear and these cells begin to break down any dead bone. This cellular mass, which has both new bone being formed and old bone being removed, is called the "callous". The callous is usually obvious about two to three weeks after the fracture has occurred. Over the next 4–12 weeks, depending on the site of the fracture, the callous is slowly converted into the new bone. Initially, the mass of new bone laid down exceeds the margin of the original bone and over a period of time, this excess bone is removed, a process called "remodelling", until finally the damaged bone takes on a reasonably normal appearance. Bone is usually able to carry normal loads between 6 and 16 weeks after the original injury, assuming that the process has not been slowed by infection or delayed healing due to poor stabilisation of the bone ends.

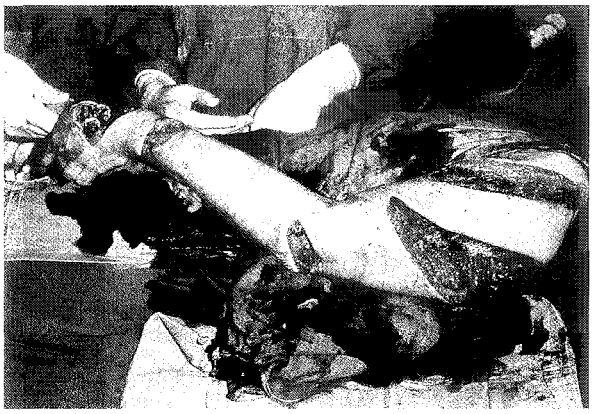


Figure 19.5 *Multiple open wounds with compound and complicated fractures.*

CLASSIFICATION OF FRACTURES

Fractures may be divided into two broad groups: open and closed. Both categories can be associated with injuries to structures such as vessels and nerves, and, if significant damage occurs to the surrounding structures, then the fracture is referred to as a "complicated" fracture (Figure 19.5). An open fracture differs from a closed one as, in the open fracture, there is a communication between the fracture site and the exterior. This is obvious if there is a skin laceration over the fracture, but in a fracture of the nose or base of the skull, the breach in the mucosal lining over the bone is not obvious; nevertheless they too are considered to be open fractures because of the communication with the exterior. Open fractures are obviously more prone to subsequent infection.

Disposition of fractures

The appearance of fractures is obviously extremely variable, but there are a number of patterns that can be recognised.

1 *Complete fracture* Here the bone is broken into at least two parts. The fracture may be transverse or spiral, or oblique in appearance. The last group is

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often the most difficult to maintain in good alignment. Occasionally, the fracture is impacted. This occurs when a bone is broken and then the shattered ends driven together again. A comminuted fracture is one where there are more than two fragments. This often occurs in long bone fractures or finger fractures where there may be a number of fragments of bone at the fracture site. Where there are multiple fragments of bone, the fractures often heal poorly.

2 *Incomplete fracture* In this situation, the bone is not completely divided and the periosteum, the tough fibrous sheet that covers the bone, is still in continuity. A common example is the so-called "greenstick" fracture that occurs in children. In children, the bones appear to be less brittle, rather like a green branch, and will tend to bend rather than fracture. The other common incomplete fracture is where compression forces occur and a compression or crush fracture results. These fractures are seen commonly in the vertebrae and in the ankle, particularly the calcaneum. In this type of fracture it is rarely possible to achieve reduction, the fractures are stable and almost always a degree of residual deformity will persist.

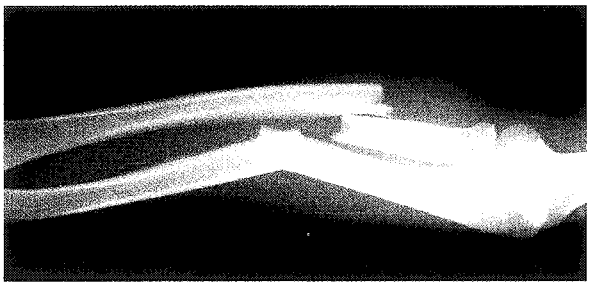


Figure 19.6A *Fracture of the radius and ulna with significant displacement and shortening.*

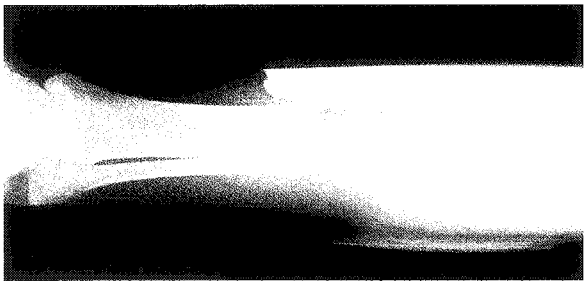


Figure 19.6B *Fracture of the tibia and fibula with significant displacement and some shortening due to muscle action.*

Displacement of bone fragments

In most fractures, there will be displacement of the bone fragments. An important factor in the degree of displacement and final position of the bones will be the nature and direction of the force that has produced the injury. However, once the continuity of the bone has been disrupted, then gravity and, more importantly, the now unopposed action of muscles will bring about tilting or overlap of the fragments. Thus, it is not uncommon for a fracture to result in shortening of the limb due largely to this unopposed muscle action (Figures 19.6A, 19.6B and 19.6C).

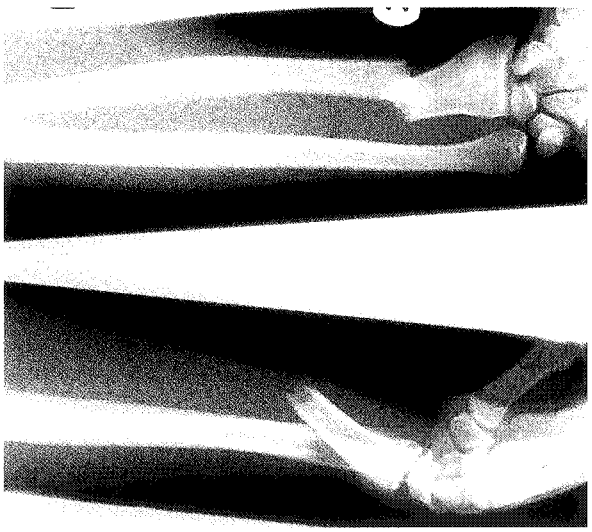


Figure 19.6C *Fracture of the radius with considerable angulation – a transverse fracture.*

Assessment of a patient with a fracture

The initial assessment by the first aider is the same as for any casualty – the St John Ambulance DRABC action plan (Danger, Response, Airway, Breathing, Circulation).

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Once that has been completed, the next step is to assess the patient fully for other injuries in the routine way.

DIAGNOSIS OF A FRACTURE

In general, in conscious patients, the presence of pain is the most important indicator of possible bony injury. The other cardinal signs are swelling, deformity and loss of function.

Loss of function is an extremely valuable sign and must never be overlooked. This applies particularly to casualties who may have sustained a fracture of the pelvis. Often it is difficult to elucidate any of the common signs and symptoms but, as soon as the casualty tries to bear weight, they get pain or the limb will give away. Whenever this occurs, always suspect a fracture.

It is important to remember that in the early stages there may not be a great deal of swelling, particularly in the thigh and upper arm, as a unit of blood spread throughout the thigh will not produce much more than a centimetre increase in circumference, which is very difficult to detect. If there are pelvic or rib fractures, it is important to recognise that there is a high probability of coexisting soft tissue injury. For example, in the case of fractured ribs there may be associated damage to lung, liver or spleen. Because of this common association, it is always important to maintain a careful check on the general condition of any casualty with suspected rib fractures or other fractures that are likely to be associated with major organ damage. Sometimes on initial assessment it is not possible to be certain that there is a fracture present. However, if there is any doubt, it is always preferable to over-treat, rather than under-treat, a casualty.

COMPLICATIONS OF A FRACTURE

It is important that all persons rendering first aid are aware of the possible complications.

Shock – neurogenic and oligaemic

While the pain of a fracture may produce neurogenic shock, the loss of blood associated with a major long bone fracture will frequently cause oligaemic shock. Because there are no obvious wounds or external blood loss, the potential importance of the effect of a fracture on the circulatory system may not be immediately obvious. However, all casualties with pelvic and limb fractures need to be treated for shock and require rapid transfer to specialised medical care.

The principles of care are to assist with venous return, if possible by elevation of the limbs and the administration of oxygen.

Infection

In the case of open fractures, it is important to be aware of the very real risk of infection in such a situation. Post-traumatic wound infection has become the most common cause of osteomyelitis (bone infection – see Figure 19.7) which can greatly impede the satisfactory healing of a fracture. The incidence of infection will be increased if a wound is severely contaminated and also where there is death of soft tissue and bone. Fortunately, infection rates with good management are low with the reported figures indicating that about 2–3 per cent of people sustaining open fracture will end with an infection and, in over 80 per cent of these infections, *Staphylococcus aureus* will be the causative agent. While almost all organisms can cause infection, the two that have the most life-threatening potential are tetanus and gas gangrene. Tetanus is produced by *Clostridium tetani* which flourishes in dead or dying tissue. It produces an exotoxin (a chemical substance) which passes through the blood and lymphatic systems to the central nervous system. There this toxin becomes bound to particular nerve cells and, as a result of damage to these cells, there is interruption to the normal transmission of nerve impulses; this then affects, in particular, the tone of muscles. This disruption of the normal controlling mechanisms causes the muscle to go into spasm. It is important to remember that tetanus is a preventable disease and everyone should be immunised. It is always helpful to ask if casualties have been immunised and to record this. It is also worth noting that over 70 per cent of tetanus cases result from injuries that occur in the home.

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Gas gangrene is produced by an organism called *Clostridium welchii* which is an unusual germ in that, unlike most bacteria, it survives best if there is a low level of oxygen in the tissue. If the conditions are appropriate, this bacterium can produce a toxin which has the ability to bring about destruction of the walls of the cells, particularly of the muscle tissue. This causes extensive death of these tissues.

COMPLICATIONS INVOLVING SOFT TISSUES

Vascular injuries

Blood vessels are most commonly injured when there are fractures around the knee, the shaft of the femur and the elbow. The important signs and symptoms that indicate there has been significant damage to the arterial supply of a limb are that the limb will become very painful and, on examination, will be found to be pale and pulseless, and normally cannot be moved by the casualty without very severe pain. Early medical intervention is mandatory; however, if for some reason transfer of the casualty cannot occur within one hour, it may be appropriate to try very carefully to reposition the limb to try to re-establish some blood flow. Wherever possible, this should be attempted only on medical advice. Equally important when trying to stabilise a fracture, the vulnerability of the blood supply must always be borne in mind. If, in trying to stabilise the fracture, it becomes evident that this is impairing the blood supply, then that approach should be abandoned and another method of splinting or stabilising the fracture used. It is also important to remember that the swelling that may occur, even after the fracture is stabilised, may also impair the circulation to the limb if the bandages are too tight and, therefore, it is imperative to check constantly that the distal part of the limbs have adequate blood flow.



Figure 19.7 *Bone destruction of the head of the fifth metatarsal bone due to osteomyelitis.*

Nerve supply

It is also important to be aware that nerves, as well as blood vessels, may be damaged in the presence of a fracture (Figure 19.8). It is important, if possible, to establish if there has been nerve damage prior to splinting a fracture. Injury to a nerve will produce numbness which may be confused with the numbness produced by damage to a blood vessel and the resultant anoxia due to poor blood flow. In the case of poor blood supply, one would expect no pulse and very poor perfusion of the tissue; this usually makes the cause of the numbness easy to establish by careful examination.

TREATMENT OF FRACTURES

The aim of treatment of any fracture is to stabilise the ends of the bone. If this can be achieved, it should minimise pain, reduce bleeding and reduce the likelihood of further injury to the soft tissues surrounding the fracture site. Having diagnosed a fracture, splintage can be achieved by using the body or external appliances to maintain a stable position. In all instances of bleeding, open wounds will need to be treated first.

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The most convenient way to stabilise a fracture is to use the uninjured part of the body as a splint. Such an approach is appropriate, particularly if the fracture is stable and there is minimal deformity. In some upper limb fractures, for example, all that may be required is a sling and minimal bandaging. However, it is important to remember that if the body is used to stabilise the fracture, any of the natural hollows of the body must be padded so that the fracture is stabilised against a flat base.

In general, the greater the deformity and the longer the time interval before the casualty will receive medical care, the greater the case for some form of external splintage. Rigid splintage systems have been used to stabilise fractures since ancient Egyptian times; thus they are by no means novel. In all instances, the splinting material needs to be padded and bandages applied in such a way that the fracture site is not deformed. The advantage of this type of splintage system is that appropriate splints can be readily improvised from materials available at most accident sites. The other method of splintage, used particularly by ambulance services, is the air splint system where an inflatable device specially designed to suit either the upper or lower limb can be quickly applied. The advantage of such a system is that it produces uniform pressure over the limb and provides effective stabilisation. However, they are not suitable for all types of fracture and, in particular, may not be easily or safely applied if there is significant deformity.

TWO SPECIAL FRACTURES

In this chapter, no attempt has been made to cover the specific management of all fractures. However, comment is appropriate regarding some special fractures.

Pathological fracture

In this instance, the bone breaks not because of abnormal stresses, but because the bone has been weakened due to some pre-existing bone disease. In particular, if cancer has spread to the bone, it may damage the bone and bring about a weakening, or the bone may become abnormally brittle because of some bone diseases such as Paget's Disease.



Figure 19.8 *Fracture of the humerus. This type of fracture is often associated with damage to the radial nerve, as the nerve runs in close proximity to the shaft of the humerus.*

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Stress fracture

A stress fracture is one that occurs in apparently normal bone. It seems to occur most often if there has been repetitive minor trauma to a bone rather than one major incident. Sites that are most commonly affected are the metatarsal bones (bones of the forefoot) the fibula and, much less commonly, the humerus or femoral shaft. There is frequently a history of unaccustomed repeated activity, such as running or walking long distances. In most instances, the casualty will experience pain either during the activity or shortly after the activity has stopped. The findings are similar to those associated with other fractures, with pain and swelling being the most common findings. Usually there is no deformity.

The first aid treatment for both pathological and stress fractures is the same as for any other fracture.

SPINAL INJURIES

Because of the potentially serious and irreversible nature of damage to the spinal cord, special mention is made of these fractures. Fractures involving the spine can be classified as either stable or unstable. As far as casualty management is concerned, the most worrying are the unstable fractures. In this group, the cord may have already been damaged, or more significant inappropriate movement may bring about damage. Fortunately, the majority (90 per cent) of spinal fractures are stable, and in only 5 per cent is the cord damaged. The stability of the vertebrae depends primarily on the integrity of the strong ligaments that run between them, and the strongest component of this system is the posterior ligament.

If a person is involved in a major accident, a spinal injury must always be considered. This applies particularly to high-speed motor vehicle accidents and falls from significant heights. When assessing such a casualty, always enquire about numbness or weakness in the limbs if the casualty is conscious. Beware of the casualty who complains of neck stiffness or pain, even if they are moving. It is also important to establish the nature of the accident, as a fall from a height or an event likely to produce a whiplash injury, such as a rear-end motor accident, are well established mechanisms by which vertebrae may be fractured.

In assessing such a casualty, look to see if there is any external damage such as an abrasion to the head, which may suggest a hyperextension injury; test for altered sensation, particularly in the limbs; and carefully feel along the backbone, checking the alignment of the spinus processes and testing for tenderness. Do not move the patient. If, however, movement is necessary, try to ensure that the patient is always moved in one piece. By this means it is less likely that the position of the spine will be altered. This is extremely important, particularly in the presence of an unstable fracture.

Debate exists as to the best way of stabilising fractures of the cervical (neck) vertebrae. Most agree that a collar is required, and recently there has been strong support for the use of rigid collars in the belief that they will allow the least mobility. However, whilst these collars may produce the best stability, they are not readily available at the site of many accidents. The next option is to use a soft collar which can often be improvised using pieces of plastic foam or newspaper. These softer collars will not achieve the same degree of stability, but the most valuable aspect of a collar is that its presence immediately alerts people looking after such a casualty that the casualty may have a cervical fracture. This in itself is enormously valuable. It is also important to remember that a collar should be applied before a casualty with a suspected spinal injury is shifted from the site of an accident. In the case of all fractures, it is very important to ensure that there is no undue pressure on the skin by splints or items that may be in the casualty's pockets or inadvertently left underneath the casualty when he or she is placed on a stretcher. Attention to skin care is particularly important in casualties who may have spinal injuries, as one of the major long-term problems in the management of these casualties can be skin ulcers.

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CHAPTER TWENTY

HEAT STRESS, DEHYDRATION AND THE HEAT DISORDERS

Michael Flynn

The first aider and the paramedic are often confronted with dehydrated patients. Heat exhaustion and heat stress are not rare, and contribute disproportionately to morbidity and mortality in otherwise fit young adults. These two themes are thus important topics for those who work in the pre-hospital setting. The science of temperature regulation and fluid balance control is an inescapable theme for those who work with victims in the field.

Along with other highly developed mammals and birds, the human organism can operate efficiently only with its internal body temperature maintained within a narrow range, and this despite extremes of environmental conditions and physical activity. Complex temperature regulating mechanisms adjust heat production and heat loss. These have evolved to enable the internal or core temperature to be maintained in a narrow 1.4°C range (usually 35.8–37.2°C oral, 36.1–37.5°C rectal).

Mammals are known as homoeothermic or warm blooded, whereas species with less developed temperature regulating mechanisms, such as reptiles, amphibia and fish are poikilothermic (cold blooded). For humans, failure to maintain body core temperature within this narrow 36–37.5°C range can lead to physiological and pathological changes. If prolonged, abnormal body temperature can lead on to morbidity and mortality.

Victims of heat disorders may present as single cases, or they may come in clusters. Historically, heat disorders have led to well documented mass casualty episodes such as those occurring during the hajj (the annual pilgrimage to Mecca), the Indian Mutiny and the 1967 Six Day War. Despite the fact that most of the basic physio-pathological principles underlying heat disorders have long been known, serious injuries and fatalities still occur in what is an eminently preventable condition. A basic knowledge of heat disorders, the conditions liable to produce such disorders, and the mechanisms of prevention and treatment outside the hospital setting is required by many. In particular, the managers of such diverse entities as heavy industry, mining, fire fighting, the military and mass participation fun run sporting organisations must be aware of heat and dehydration problems. In this chapter, heat stress is taken to mean the sum of metabolic and environmental heat loads; heat strain is the effect on the body of heat stress. Heat stroke is the life-threatening clinical syndrome which develops as the end result of the effects of severe, unchecked heat stress.

TEMPERATURE REGULATION

The maintenance of core temperature is determined by the interaction between heat production and heat loss, and is controlled primarily by temperature-regulating centres in the hypothalamus. The spinal cord can also play a critical role. Other highly thermosensitive sites which provide important inputs to the integrating centres in the hypothalamus and spinal cord occur in the midbrain, medulla oblongata, abdomen, skeletal muscle and skin. Thus it appears that temperature-sensing sites are distributed at different densities

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throughout the body core; together with an extremely strong cutaneous thermoreceptor input, body temperature, as the regulated variable, is the end result of these signals being integrated in the hypothalamus and spinal cord.

HEAT STRESS

The heat stress imposed upon the body comes from two sources: intrinsic metabolic heat production, and externally imposed environmental heat.

Metabolic heat load

The major source of the body's heat is the contraction of skeletal muscle. For example, shivering may cause a 5-fold or exercise a 10–20-fold increase in metabolic rate. During exercise, the biochemical processes leading to muscle contraction are relatively inefficient. The excess energy is released as heat which, if not dissipated, accumulates in the body and causes a rise in core temperature. Heat is also produced by basal metabolic processes which are influenced by catecholamines (the body's stress hormones) and thyroxine, and by the specific dynamic action associated with the ingestion of food.

Environmental heat load

External heat input may be radiant, conductive, convective or some combination thereof. Environmental heat load comprises ambient temperature load and radiant (usually solar) heat load modified by humidity and wind velocity. The sun's rays and working place sources, such as furnaces, factory machinery and engine room spaces, are the principle sources of radiant heat. Hot winds (heated, flowing air) transfer heat energy to the body by convection.

HEAT LOSS

There are a number of mechanisms by which the body dissipates heat. In temperate conditions (say 21°C) in air at rest, small amounts of heat are lost through respiration and via the excretory processes. However, the major heat loss is through convection, conduction and radiation (70 per cent), and the vaporisation of sweat (27 per cent). The principal method of regulating heat loss is by varying the volume of blood flowing to the surface of the body and by sweating. This process is mediated by the sympathetic nervous system involving both increased activity (active neurogenic vaso-dilatation) and also withdrawal of vasoconstrictor tone acting on arterioles, venules and arterio-venous anastomoses. This increase of blood flow to the skin is further enhanced by increasing cardiac output and redirecting blood flow from splanchnic, renal, fat and muscle beds. Sweating is triggered primarily by sympathetic cholinergic stimulation of the eccrine sweat glands. Sweat glands are widely distributed, but are particularly well represented in the face and scalp.

As the environmental temperature increases and approaches body temperature, radiation losses decline and vaporisation losses increase. Vaporisation of 1 gram of water removes about 0.6 kilocalories of heat. Sweat secretion may reach values as high as 1600 millilitres per hour. The effectiveness of this as a means of removing heat is reduced as the humidity of the air increases. When environmental temperatures exceed body temperature, radiation and convection/conduction processes no longer dissipate heat and, instead, act to add to the heat load of the body. In these conditions, evaporation of sweat is the *only* operative mechanism for heat removal.

The wearing of loose-fitting non-restrictive porous protective clothing can assist in the heat dissipation process by promoting convective transfer of heat through the air spaces between and beneath the layers and protect against high radiant heat loads. However, this does not apply for heavy protective clothing such as that worn by fire fighters, or the impervious Nuclear, Biological and Chemical (NBC) suits worn by military personnel. Use of such clothing, particularly where the face, scalp and neck are covered, will substantially increase the likelihood of heat strain casualties. Children, adults with impaired circulation (diabetics,

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the elderly), and those with nervous system abnormalities (alcoholics, para/quadriplegics) and skin abnormalities (ectodermal hypoplasia/scarring from extensive burns) have an impaired response to heat stress, and are more vulnerable to heat strain disorders. From an occupational perspective, foundry workers and miners are at special risk. Patients taking drugs such as atropine/scopolamine, ephedrine (cough mixtures), anti-histamines, B-blockers and psychotrophic drugs (monoamine oxidase inhibitor antidepressants, tricyclics, lithium and cocaine) are also more likely to succumb to heat stress. These drugs can be extremely detrimental by pharmacologically inhibiting the sweating process. A diagrammatic representation of the factors involved in the production and loss of heat from the body is shown in Figure 20.1.

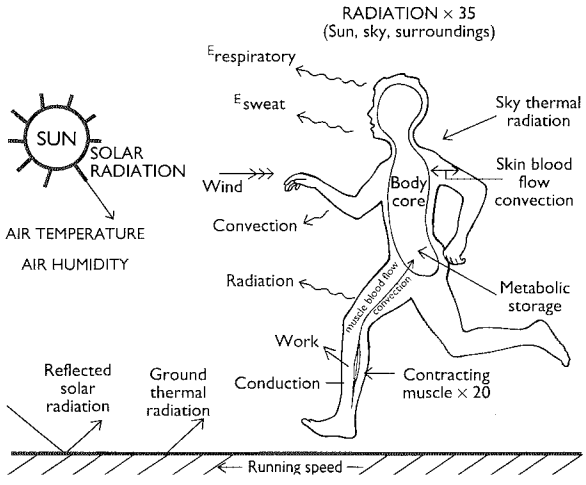


Figure 20.1 *Schematic diagram portraying production and transfer of heat within the body and heat exchange with the environment. E = Evaporation. Environmental heat load may increase by a factor of up to 35. Intense muscular activity can cause a 20-fold increase in metabolic heat load. (Modified from Gisolfi and Wenger (1984). By permission, Elsevier Science Publishers BV-Amsterdam.)*

HEAT INDICES

Various indices designed to quantify environmental heat load have been developed; however, all have their limitations. One index commonly used is the Wet Bulb Globe Temperature (WBGT), devised for monitoring the outdoor environment in sunlight for the purpose of reducing heat casualties in the United States Marine Corps training operations.

For outdoor, sunlight conditions, the WBGT Index is computed from readings of

a a stationary wet bulb thermometer exposed to the sun and to the prevailing wind, (natural wet bulb – nwb),

b a 6-inch black globe thermometer similarly exposed (globe temperature, T_g),
and

c a dry bulb thermometer shielded from the direct rays of the sun (air temperature T_a).

The WBGT Index is computed as follows:

$$\text{WBGT} = 0.7 \times \text{nwb} + 0.2 \times T_g + 0.1 \times T_a.$$

FLUID HOMEOSTASIS AND DEHYDRATION

Because of the pivotal role of sweating and changes in regional blood volumes and flows in the dissipation of heat, particularly with exercise, an understanding of the mechanisms by which the body maintains its fluid balance is needed when considering the prevention and treatment of heat disorders. To maintain a fluid balance to meet minimal obligatory (insensible) respiratory loss (600 millilitres per day (ml/day) in adults) and a urine volume of 800 ml/day necessary for the excretion of metabolic end-products in a non-sweating adult male, a minimum fluid intake of about 1700 ml/day is required. Given that eccrine sweating can be sustained at over 1 litre per hour in certain environmental/exercise conditions, corresponding volumes of water are required in order to maintain an adequate circulating blood volume. A limiting factor is the ability of the stomach to empty ingested fluid. At a maximal emptying rate of 900–1200 ml/hour, such a sustained loss of fluid in sweat is feasible in all but extreme cases, provided

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adequate water is available and that this water is actually ingested. In conditions where replacement requirements for the loss of sweat exceed the maximal emptying rate of the stomach, personnel will become increasingly dehydrated regardless of the amount imbibed. Water will be lost from all three fluid compartments of the body – intravascular, interstitial and intracellular. As an example, a hydrated 70 kg non-heat-acclimatised man exercising in the heat over 4 hours who loses 6 per cent of his body weight (4.2 litres) as sweat, will have lost 20 per cent (700 ml) of his plasma volume if he does not drink. Excessive alcohol consumption may cause dehydration and predispose to heat-related illness.

SALT BALANCE

Sweat is a hypotonic fluid comprising predominantly water and sodium chloride. In the unacclimatised, the sodium concentration in sweat is of the order of 75 milliequivalents per litre (mEq/l). Replacement of fluid without a concomitant replenishment of salt can lead to heat cramps. Supplemental salt may be required for personnel undergoing acclimatisation. However, the salt in normal food will usually suffice after acclimatisation has occurred.

THIRST AND FLUID REPLACEMENT

A number of factors trigger thirst. These include hypokalaemia, hypoglycaemia, hypernatraemia and hyperosmolality. At the cellular level, it is believed that the primary mechanism regulating thirst, and therefore fluid intake, is a Na^+-sensitive receptor, possibly in the preoptic region of the brain.

The term "voluntary dehydration" is used to describe the tendency for humans, working in heat, not to replace all of the water losses incurred due to sweating.

In this context, thirst may be "inadequate", or its onset is delayed. This has led military commanders to enforce the concept of "water discipline" or "water doctrine", whereby troops ingest water on a regular basis, under orders if necessary, irrespective of the presence or absence of thirst. The same concept has been extended to long-distance fun runs where participants are educated and encouraged to drink before, during and after the run, utilising drink stations situated at regular intervals along the route. In practical terms, fluid intake should be sufficient to provide a plentiful amount of pale (dilute) urine. Water requirement tables necessary to support planned work/rest cycles in varying environmental conditions have been developed for the guidance of military supervisors. Separate tables have been developed as guides for work cycles during both day and night, and for jogging. In using such tables, the qualifying conditions must be duly recognised and allowances made for lack of acclimatisation, the wearing of protective clothing, pre-existing illness and age.

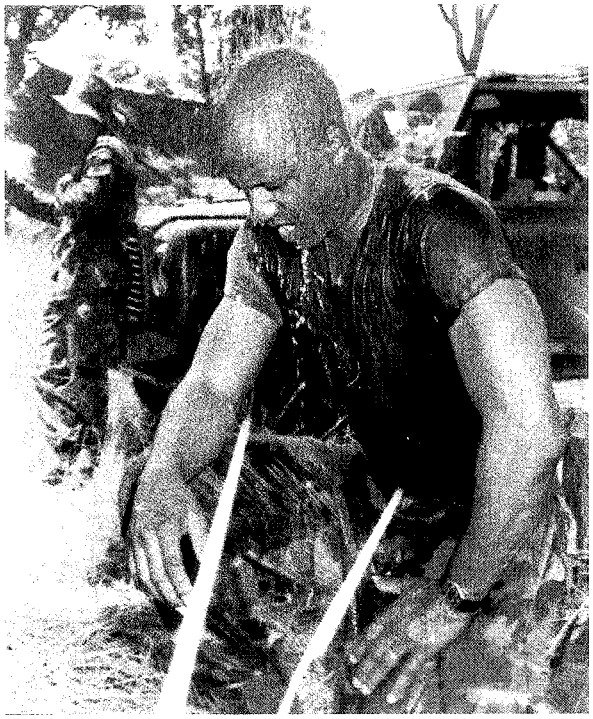


Figure 20.2 *A United States soldier on combined exercises in tropical northern Australia, suffering from heat stress. (Photograph courtesy of* Army Magazine.*)*

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ACCLIMATISATION

The phenomenon of acclimatisation refers to the physiological changes experienced by humans when they move to a warm geographical area or are artificially exposed to a warm environment or significant daily exercise. Over a period of 8–14 days, the body adapts to the change in environment by increasing the capacity to increase skin blood flow and to sweat; both mechanisms are triggered at lower body temperatures and increase more rapidly with increasing heat stress. The amount of sodium chloride potentially lost in the sweat is reduced by 60 per cent with the sodium concentration falling from 75 mEq/l to 30 mEq/l. This changes sweat from the equivalent of 0.5 isotonic saline to 0.2 isotonic saline.



Figure 20.3 *Rehydration at the finishing point. (Photograph courtesy Defence Public Information Queensland.)*

By contrast to the previous example, the hydrated 70 kg heat-acclimatised man exercising in the heat who loses 6 per cent of his body weight (4.2 litres) as sweat over 4 hours without drinking will only have lost 14 per cent (490 ml) of his plasma volume. Physiological indices in response to exercise show an increase in circulating plasma volume, progressive improvement (lowering) in the final heart rate, final rectal temperature and skin temperatures, reflecting the observation that daily exposure to exercise under environmental conditions of heat stress leads to improved tolerance to extreme heat. The adaptive processes of acclimatisation are lost progressively over as little as two weeks on return to temperate climates.

CLINICAL FEATURES OF HEAT DISORDERS

Increase in body core temperature is manifest in a range of clinical disorders including the feverish patient with influenza responding to microbiological challenge, the sunburned, through to the critically ill patient with heat-damaged organs and life-threatening pathology. Five specific heat related disorders are described:

- heat oedema
- heat syncope
- heat cramps
- heat exhaustion
- heat stroke

Heat oedema

Heat oedema presents as swelling of the feet and hands in warm weather. It is presumed to be a result of vasodilatation with vascular leak exacerbated by limb dependency leading to an increase in interstitial fluid. No active treatment is necessary other than gentle exercise and elevation of the legs, and keeping the patient cool. Diuretic therapy is not appropriate and may increase the risk of a more serious heat disorder.

Heat syncope

Heat stress leading to the combination of mild dehydration, decreased vasomotor tone and lower limb

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and pelvic venous pooling when in an upright posture progressively leads to decreased central venous pressure. In time, cardiac filling pressure may become inadequate to sustain stroke volume. The reduced cardiac output and lowered systolic blood pressure then precipitate classic vasovagal syncopal fainting. Prompt recovery usually follows the assumption of a horizontal posture.

Oral rehydration is advisable. Heat syncope is probably not a discrete clinical entity, rather an aggravation of the normal neurally-mediated syncopal episode.

Heat cramps

Painful, skeletal muscle spasms develop following strenuous exercise, or following several hours profuse sweating at rest in a hot environment. The most likely cause is salt depletion. Treatment involves cessation of activity, rest in a cool environment and replacement of fluids and electrolytes either orally or parenterally.

Heat exhaustion

Heat exhaustion is the most common clinical disorder resulting from work or physical activity in a hot environment. The term is used to describe a number of syndromes leading to collapse. Heat exhaustion must always be suspected in a person who collapses physically in a hot environment or during exercise in a warm environment; rectal temperature is commonly elevated but may be only 38.0–38.5°C, and mental confusion is common. The predominant feature is cardiovascular insufficiency brought about by insufficient water intake, salt depletion, or a mixture of both when great losses of sweat have occurred. It is not associated with evidence of organ damage. Two major forms of heat exhaustion have been described: water depletion and salt depletion heat exhaustion. A further functional differentiation has been made between Exercise Induced Heat Exhaustion (EIHE) and "Classic" Heat Exhaustion (CHE). Both forms may progress to heat stroke.

Water depletion heat exhaustion is seen in healthy persons and results from physical exertion in a hot environment, for example, mining, construction work, training of military recruits, or long distance running especially if unacclimatised. Large losses of (hypotonic) sweat combined with inadequate fluid intake result in dehydration and hypernatraemia. Water depletion heat exhaustion features prominently in EIHE.

Symptoms and signs include thirst, fatigue, giddiness, anxiety, headache, nausea, vomiting, oliguria and pyrexia, while in the advanced stages delirium develops. Rectal temperature is usually at least 39°C. Patients should be actively treated when the core temperature rises above 38°C.

The diagnosis of water depletion heat exhaustion is usually straightforward. Incipient forms may be difficult to recognise, and it is incumbent upon personnel in supervisory roles to be familiar with the conditions that predispose to heat exhaustion, and to know the early symptoms of heat exhaustion such as apathy and loss of concentration. Rapid recovery usually follows cessation of activities, removal of the patient to a cooler environment, loosening of clothing and oral rehydration. The main danger is that of an early resumption of activity which may lead to heat stroke. The appearance of symptoms of heat exhaustion in one member of a group should serve as a warning that other members may be similarly affected.

Salt depletion heat exhaustion is typically seen in sedentary elderly subjects exposed to environmental heat stress who replace their sweat loss with large volumes of water only and develop salt depletion. Salt depletion heat exhaustion features prominently in CHE. In this form, symptoms may be more subtle with non-specific fatigue and weakness accompanied by high body core temperature.

In practice, elements of both contribute to the clinical picture. Common presenting features are high body temperature (greater than 38.1°C), followed by weakness, giddiness, fatigue, headache, and dry mouth and tongue. Uncomplicated heat exhaustion is now commonly regarded as part of a continuum of events leading to heat stroke and it may indeed be regarded as incipient heat stroke.

Heat stroke

This is a serious condition in which tissue cell damage follows failure of the normal thermoregulatory mechanism after exposure to hot environments and/or

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strenuous exercise. Without prompt treatment, mortality can be up to 70 per cent. Clinical features include:

- core temperature greater than 40°C,
- disturbances of the central nervous system,
- anhydrosis (dry pale hot skin), and
- disseminated intravascular coagulation (DIC), organ, tissue and cell damage, and disturbances of metabolism.

Classic heat stroke typically appears in the elderly during heat waves. Exertion-induced heat stroke occurs in young healthy individuals. Heat stroke has been observed to occur at lower core temperatures in the exercise-induced type than in the classic heat stroke group. A high index of suspicion is required by both medical and paramedical personnel for cases presenting with confusional states in conditions predisposing to heat disorders. Measurement of rectal temperature should be included as part of the systematic examination. Absence of sweating is not a consistent feature (the skin may be moist or flushed), and the differentiation between heat exhaustion and heat stroke is not straightforward. Complications may not develop till late and, in many cases, the diagnosis of heat stroke can be made only retrospectively. In practice, it is safer to assume that heat exhaustion casualties may develop complications and, therefore, to manage them as incipient heat stroke victims.

PATHOPHYSIOLOGY OF HEAT STROKE

The mechanism leading to cessation of sweating in heat stroke is thought to be due to sweat gland fatigue and/or vasoconstriction of the cutaneous blood vessels. This is the outcome of the breakdown in the balance between competing demands for the regulation of blood pressure and regulation of body temperature. During exercise and heat stress, initially there is sweating and increased blood flow to the limb muscles, myocardium, fat and skin. The increased blood flow is provided by an increased cardiac output and redistribution of blood flow away from abdominal viscera. With fluid loss in sweat and an increase in cutaneous (peripheral) blood volume, the central venous volume and central venous pressure eventually begin to fall. The blood flow to skin is reduced, supposedly owing to cardiopulmonary baroreceptor activity, and sweating is reduced and then ceases, aggravating the rate of rise in core temperature. The thermoregulatory capability of the body is lost. Collapse occurs due to central nervous system (CNS) or other effects of severe hyperthermia, such as erythrocyte sphering, disseminated intravascular coagulation with microthrombosis and coagulative necrosis. Without prompt treatment, diffuse neurological and other damage leads to death. A summary of the underlying physiological processes involved in the development of heat exhaustion and heat stroke as shown in Figure 20.4.

Central nervous system dysfunction is so common in severe heat strain that it is regarded as a key diagnostic

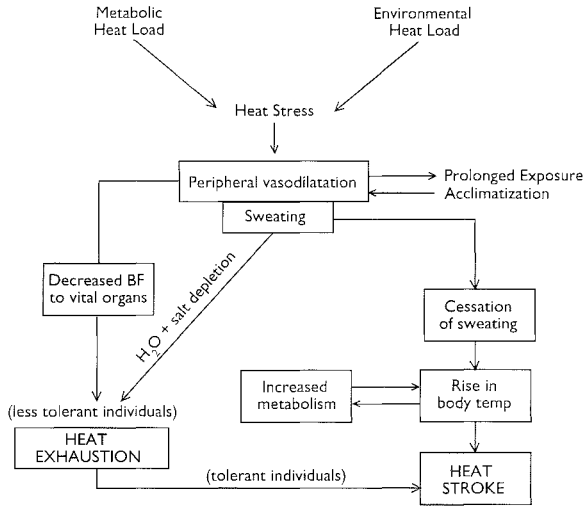


Figure 20.4 *Physiological processes involved in the development of heat exhaustion and heat stroke. (As adapted from Hales and Khogali (1987). Courtesy Elsevier Science Publishers BV-Amsterdam.)*

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feature for heat stroke. In comparison to some animals (camels, sheep and dogs), humans have a much restricted ability to cool the brain selectively by maintaining a differential between the core temperature and that of the head. CNS dysfunction is caused by metabolic disturbances including acidosis, hypoxia and direct thermal damage. Rhabdomyolysis ("muscle rot") is a frequent acute complication of exertional heat stroke. Large quantities of potassium, myoglobin and other metabolic products are released. These may contribute to renal failure in addition to the direct effects of hypoperfusion and acute tubular necrosis. Hepatic damage is reflected in elevated transaminase levels, clotting factor deficiencies and jaundice. Inhalation of vomitus may produce diffuse pulmonary damage, and contribute further to the general hypoxic cellular insult.

Severe endotoxaemia is increasingly being recognised as a serious complication, due to breakdown of the intestinal tissue barrier to lipopolysaccharides (LPS) in intestinal contents brought about by the hyperthermia and reduction in intestinal blood flow and to the inability of the reticulo-endothelial system to cope.

Post-mortem findings in fit young heat stroke casualties have revealed widespread evidence of a generalised bleeding tendency (DIC) with cutaneous ecchymoses, pulmonary, gastro-intestinal tract, cardiac, renal, adrenal and cerebral haemorrhage.

MANAGEMENT AND FIRST AID

The clinical outcome of patients with heat exhaustion and heat stroke is primarily a function of magnitude and duration of temperature elevation before the onset of definitive treatment. Treatment based upon an understanding of the basic patho-physiological changes can reduce morbidity and prevent mortality.

Although rapid cooling is advised, evidence of a correlation between rate of cooling with improved outcome in heat stroke is inconclusive. A variety of techniques have been used, and no particular technique or rate of cooling has been unequivocally demonstrated to be superior with the exception of the highly specialised Mecca Body Cooling Unit. What is clear is that cooling should be initiated as soon as possible after recognition of heat illness. Consideration needs to be given not only to the management of one casualty who may need advanced intensive care, but also to the management of multiple casualties in scenarios bereft of sophisticated facilities.

If heat stroke is suspected and the temperature is elevated, cooling should not be delayed to accomplish a diagnostic evaluation. Cooling and evaluation should proceed simultaneously.

Basic principles of first aid (such as the St John Ambulance DRABC action plan – Danger, Response, Airway, Breathing, Circulation) including cardio-pulmonary resuscitation take priority at all times. Airway control is essential. Nausea and vomiting are common. Oxygen should be provided if available. Heat casualties should be removed to a shaded, cool environment. Clothing should be loosened or removed, and the patient cooled by applying cold packs to the neck, axillae and groins or wetting and massaging the body surface with tepid/warm water and accelerating evaporation by fanning. Immersion of a casualty in a bath of cool water or, if in field conditions, improvised canvas tubs in elevated frames in ventilated shade are effective. Immersion techniques significantly increase the difficulty of nursing casualties. Cutaneous cooling with ice or forced evaporation may cause counter-productive peripheral vasoconstriction and initiate shivering which will act to oppose the cooling process. Elevation of the legs promotes venous return.

Notwithstanding the role of dehydration in the pathogenesis of heat disorders, the parenteral fluid volume requirements of most heat exhaustion (and most heat stroke) patients can be met without large volumes of intravenous fluid. In adults in the absence of vomiting, 1–1.5 litres of saline or dextrose/saline are needed in the first 1–2 hours and will usually be sufficient to allow subsequent oral replacement to restore the balance of the fluid deficit. More vigorous fluid replacement carries with it the risk of congestive heart failure, and cerebral and pulmonary oedema. No

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more than 2 litres of fluid should be administered without laboratory surveillance. Rehydration and cooling should continue until the temperature is 38°C, the casualty is lucid, is tolerating oral fluids and does not experience orthostatic hypotension on standing. Electrolyte, metabolic and acid–base disturbances in heat stroke are complex. They may include hypokalaemia and hyperkalaemia, hypoglycaemia and lactic acidosis. They require appropriate management in hospital.

With neurological disturbance, patients are frequently agitated, combative or fitting. Small doses of a benzodiazepene can be administered for control of fits.

Advanced management for heat stroke requires sophisticated facilities. Early recognition, prompt first aid measures based upon an understanding of the underlying patho-physiological changes, and expeditious transfer to an appropriately equipped intensive care centre will give heat stroke victims the best chance of recovery. Active cooling should be continued during evacuation.

PREVENTION

Prevention of heat casualties can be facilitated by adherence to established Occupational Health and Safety (OH&S) guidelines for industrial, military and recreational pursuits. Emphasis needs to be directed to principles of acclimatisation, provision of adequate water and protective clothing and, if conditions dictate, an enforced drinking policy or "water discipline". Physical activity should be restricted. Work/rest/exercise cycles must take due cognisance of physical fitness, age, clothing and ambient environmental conditions. Special instructions including basic survival techniques are needed for those who operate in desert environments.

A variety of heat stress indices have been developed to assist managers and supervisors to minimise heat stress casualties; however, all have their limitations, and no index provides sharp boundaries between acceptable and unacceptable activities.

It is incumbent upon managers and supervisors to understand the potential for subtle incapacitation in workers due to heat exhaustion. Errors of judgement may expose such workers (eg fire fighters) to further and perhaps critical heat strain, or to accidents in the case of those who are operating machinery.

Within a group, the appearance of one heat casualty may mean that other members are also at risk. A high index of clinical suspicion is required for the recognition of symptoms of heat exhaustion and incipient heat stroke. It is essential to suspect heat exhaustion in cases of collapse after exposure to heat.

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CHAPTER TWENTY-ONE

COLD INJURIES – HYPOTHERMIA AND FROSTBITE

Peter Sullivan

Warmth, warmth, more warmth! for we are dying of cold and not of darkness. It is not the night that kills, but the frost.
(Miguel de Unamuno (1864–1936), Spanish philosopher)

The term "cold injuries" tends to conjure up images of icy landscapes, unexpected disasters and extremely low temperatures. However, hypothermia and frostbite can occur in a wide range of settings and to a wide range of people – from the polar explorer to the shipwreck victim floating about in temperate seas, from the intrepid mountain climber to the deep-sea diver, from the solo bushwalker to the thousands of Napoleon's troops who perished while retreating through the harsh Russian winter, and from the premature infant with poor temperature control to the old-age pensioner in an under-heated home.

With an increasing number of people engaging in water sports and various outdoor or "wilderness" activities, there is an ever greater potential for hypothermia and frostbite to be a threat, quite literally, to life and limb. However, with proper understanding of the cause of these injuries they can largely be prevented, or at least successfully treated in the field if detected at an early stage. If ignored until well established, the first aid management of cold injuries can be extremely difficult.

To adequately comprehend the cause, initial management and prevention of such cold injuries it is essential to understand something about human thermoregulation.

REGULATION OF BODY TEMPERATURE

In order to function effectively, human beings must maintain an internal body temperature within a few degrees of the normal 37°C. This preservation of a stable temperature is achieved by a balance between heat production and heat loss and is regulated through a complex set of detectors (in the brain, especially the hypothalamus, in the skin, and in a variety of deep body sites) and effectors (in smooth muscle, in the blood vessels, in sweat glands, and in shivering or exercising muscles).

In cold conditions, the body attempts to maintain its internal (ie "core") temperature by increasing heat production and decreasing heat loss.

Heat production

Heat is constantly produced by normal body functioning such as respiration and digestion, that is to say metabolic heat production. Even more heat is produced by muscular activity during exercise – up to 10–20 times above resting levels. Shivering is an involuntary form of exercise designed to increase heat production (by up to about 5-fold) when the core temperature starts to drop.

Mechanisms of heat loss

Radiation Heat can be lost from a warm body to the surrounding colder environment by infrared radiation. Temperature difference and available surface area are the major factors in determining how much heat is radiated to the environment.

Conduction Direct contact with surfaces colder than the body results in conductive heat loss. When standing in dry conditions, the human body loses little heat by conduction – only what is conducted through the soles of the feet. Consequently, conduction is often not incorporated in laboratory-based formulae for thermal

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balance (see example below). However, conductive heat losses are markedly increased if clothing becomes wet, or if an individual becomes immersed in cold water or has to lie down on a cold surface.

Convection With convection, heat is transferred within a liquid or gas by the movement of heated particles or molecules (eg water at the bottom of a saucepan rising as it is heated). Individuals will lose heat by convection even in still conditions; heating of the layer of air next to the skin will cause it to rise, with cooler air moving in to take its place. This cool layer will in turn draw heat away from the body as it warms up to skin temperature. Convective heat losses are much greater in windy conditions because this layer of warmed air is constantly being swept away.

Evaporation Heat is lost from the body through the latent heat of evaporation, that is the energy required to convert a liquid (eg water) to a gas. This can occur through sweating, insensible perspiration, respiration or wet clothing. Like convective losses, evaporative losses are much higher in windy conditions.

Reduction of heat loss

The body reduces heat loss by constricting the blood vessels that would otherwise carry warm blood to the skin. This is called "vasoconstriction". Compared to other animals, humans are not very efficient at such physiological heat loss reduction mechanisms, but make up for this by the ability to insulate themselves with clothing and shelter.

Thermal balance

As long as the body stays in thermal balance, that is with neither heat gain nor loss, the core temperature will remain in steady state at around 37°C.

Thermal balance can be considered in terms of mathematical formulae. For example:

Heat balance =

$M - W = Q_{sk} + Q_{res} = (C + R + E_{sk}) + (C_{res} + E_{res})$

where all terms have units of watts per square metre (Wm^{-2}), and

M = rate of metabolic heat production
W = rate of mechanical work
Q_{sk} = total rate of heat loss from the skin
Q_{res} = total rate of heat loss through respiration
C_{res} = rate of convective heat loss from respiration
E_{res} = rate of evaporative heat loss from respiration
C = rate of convective heat loss from the skin
R = rate of radiative heat loss from the skin
E_{sk} = rate of total evaporative heat loss from the skin

However, thermal balance can perhaps be more easily understood in terms of Figure 21.1.

However, there are limits to both heat production and insulation, and when these prove insufficient to maintain the body's core temperature, hypothermia occurs. In addition to this generalised body cooling,

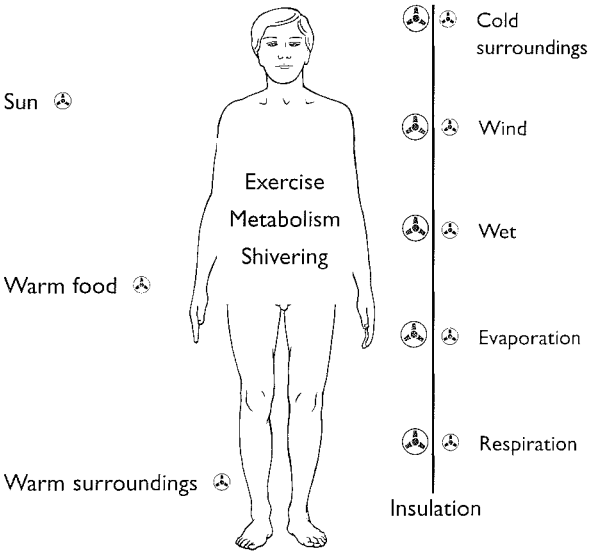


Figure 21.1 *Thermal balance. As long as the amount of heat lost is equal to that produced by the body (plus any heat gained from food and radiation sources such as the sun) the body is in a state of thermal balance and the core temperature will remain at approximately 37°C.*

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attempts to shunt the warm blood to the core can mean that the extremities chill even faster, leading to local cold injuries.

Wind-chill

The phenomenon of feeling colder in conditions of even low wind has been recognised since earliest times. However, it was not adequately quantified until the middle of the twentieth century when Paul Siple and his colleagues conducted a series of experiments into the heat loss of uninsulated cans of water. Subsequently they developed the concept of a Wind-chill Index (WCI) and formulated an equation to estimate the rate of cooling of exposed skin:

$$WCI = (10\sqrt{v} + 10.45 - v)(33 - t_a)$$

where v is wind velocity in metres per second and t_a is ambient temperature in degrees Celsius.

A variant, known as the wind-chill equivalent temperature chart, has been widely used to help predict the likelihood of hypothermia and frostbite. However, it is recognised that there are significant flaws in Siple's model and its application to clothed humans. Therefore, an improved version has recently been developed (Figure 21.2).

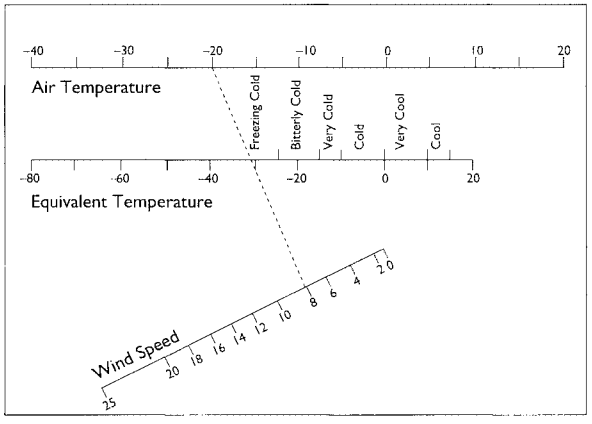


Figure 21.2 *Wind-chill nomogram. The improved wind-chill nomogram based on the work of Steadman and Dixon. The dashed line shows an example of its use. With an air temperature of –20°C and a wind speed of 8 metres per second the effect on exposed flesh is equivalent to a temperature of –32°C.*

Effect of alcohol in thermoregulation

Elevated blood alcohol levels are very commonly found in association with hypothermia. However, the exact role of alcohol in the development of hypothermia is a complex subject – certainly more complex than the often stated comment that alcohol hastens the development of hypothermia because it causes the surface blood vessels to open up, thus allowing more heat to be taken away from the body surface by radiation, conduction and convection.

The main mechanisms by which alcohol intoxication can lead to the development of hypothermia include

- decreased ability to assess risks or make properly considered judgements;
- decreased perception of cold and hence a decreased incentive to seek adequate clothing or shelter;
- the sedative effect of alcohol, leading to sleep – which is known to cause an increase in heat loss.
- impaired shivering when the core temperature does start to drop; and
- impaired glucose metabolism resulting, in severe cases, in decreased heat production.

THE NATURE OF HYPOTHERMIA

Hypothermia can be better understood, and hence managed, if it is recognised that it is not a single condition but rather the same end result of a number of different processes. Hypothermia can come about in various ways – cold water immersion, exposure to cold-wet conditions (such as in the field) or to cold-dry conditions (such as in an unheated home), induced hypothermia for surgical reasons, or hypothermia that is secondary to a variety of medical conditions such as drug intoxication, hypothyroidism or malnutrition. The physiological changes and results of treatment that are noted in one process need not necessarily apply to the other forms of hypothermia. In addition, assessment in the laboratory of the effectiveness of the various

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re-warming methods is limited by the fact that human studies are restricted by ethical considerations to cases of fit individuals with only mild hypothermia (core temperature above 34–35°C) where rapid spontaneous re-warming will occur anyway. Animal studies are not always applicable and case reports of accidental hypothermia, by the very nature of the conditions under which they occur, are often not fully documented.

Classification

Many authorities divide accidental hypothermia into categories of mild, moderate and severe on the basis of core temperature. Unfortunately, there is little agreement as to the boundaries for each category. For the purposes of this discussion, the following limits will be used – mild (34.0–34.9°C), moderate (30.0–33.9°C), and severe (less than 30°C). While useful in a hospital situation where an accurate core temperature can be obtained (see section on diagnosis below for a more detailed discussion), such core readings are often not possible in the first aid situation. None the less a general estimate can be made based on the observable signs (see Table 21.1).

Other authorities use duration as the criterion, and employ the term "acute hypothermia" for those cases where victims have been hypothermic for less than 6 hours, "subacute hypothermia" for cases between 6 and 24 hours, and "chronic hypothermia" for cases of more than 24 hours. This has a certain advantage as far as treatment is involved, for while there may be a case for rapid re-warming (eg in a hot bath) in certain acute cases, it is generally inappropriate for chronic hypothermia. In chronic cases significant fluid and electrolyte changes usually occur and actions such as rapid re-warming are far more likely to result in cardiovascular complications.

Table 21.1 Signs and symptoms of different levels of hypothermia

Level	Core temperature (°C)	Signs and symptoms
Normal	37.6	"Normal" rectal temperature
	37.0	"Normal" oral temperature
Cold	36.0	Subjectively cold
	35.0	Shivering
Mild hypothermia		Slurred speech
	34.0	Slowed thinking and movement
Moderate hypothermia	33.0	Hallucinations and amnesia
	32.0	Shivering ceases
	31.0	Decreasing consciousness
	30.0	Unconscious
Severe hypothermia	29.0	Slow pulse and respiration
	28.0	Heart arrythmias develop
	27.0	Reflexes lost
	26.0	Appears dead
	25.0	Spontaneous ventricular fibrillation may develop

These are typical signs and symptoms at different core temperatures – individuals may vary considerably. "Normal" rectal and oral temperatures may show a diurnal variation of about 1°C. It is worth noting that there is a zone between normal and hypothermia for which there is no medical terminology other than "being cold", that is, not everyone who is cold is hypothermic. (Based on Harnett et al. *(1983))*

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Another useful method is to categorise accidental hypothermia according to cause: "exposure hypothermia", "urban hypothermia" and "immersion hypothermia". This method has the advantage of ease of presentation and will be adopted for this chapter. Most of the discussion will focus on exposure hypothermia, but many points discussed in this context are applicable to the other two forms.

EXPOSURE HYPOTHERMIA

Exposure hypothermia is most likely to be encountered in the situation of the lost, exhausted or injured walker or cross-country skier. However, it can also creep up more subtly on groups or individuals – especially if they are ill-equipped for cold, wet or windy conditions.

The signs and symptoms of exposure hypothermia are often mistaken for fatigue, and even the casualty may not realise the true nature of his problem. The onset of hypothermia is often accompanied by shivering, apathy, tiredness and impaired judgement. Frequently, the behaviour of the hypothermic person becomes abnormal, with aggression or lack of co-operation, and a slowing in the rate of walking. Speech usually becomes infrequent and slurred, and visual abnormalities may occur. Muscle weakness, cramps, and clumsiness give way to repeated stumbling and falling until the victim eventually collapses with exhaustion and impaired consciousness, incoherence or stupor. Companions should keep alert for the "umbles" – the mumbles, fumbles, stumbles and grumbles.

After the casualty collapses, observers may note that the victim is cold to touch and displays generalised muscle rigidity, that the heart rate is slow, and that the peripheral pulses are difficult or impossible to find. Death can occur within a few hours of the development of these symptoms.

Management of exposure hypothermia

There is still considerable dispute as to the optimum treatment of the hypothermic victim. The following discussion does not claim to address all aspects of every case. Rather, it sets out a framework which provides the first aider with a clear set of priorities that can be implemented from the time the hypothermic victim is first discovered.

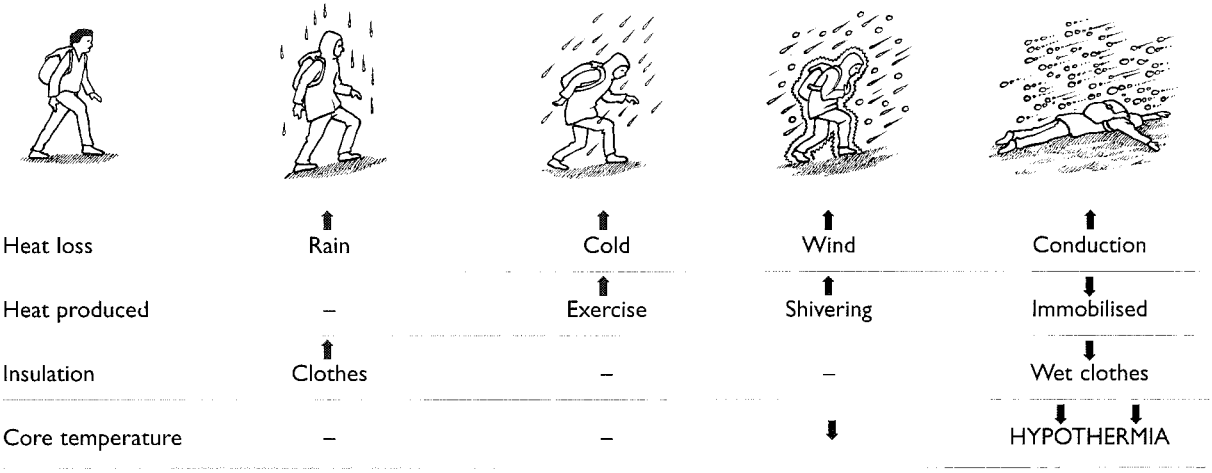


Figure 21.3 *Development of exposure hypothermia. As environmental conditions of rain, wind and snow worsen, the opportunities for heat loss increase. These losses can be countered by insulation (clothing, both windproof and/or rainproof). In addition, mechanisms for increased heat production can be called into play, namely increased exercise and shivering. However, both exercise and shivering can be exhausting and when the victim is finally forced to lie down heat production drops dramatically and heat loss via conduction to the cold ground increases. Life-threatening hypothermia can now develop rapidly. (Based on a diagram by A.G. Nicol (1974), modified by D. Maclean and D. Emslie-Smith (1977). Illustration courtesy Dr T. Cochran.)*

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This six point plan for the management of accidental hypothermia includes:

1 Assessment of airway, breathing and circulation
2 Diagnosis
3 Gentle handling of the casualty
4 Prevention of further heat loss
5 Provision of additional heat to the victim
6 Evacuation

This list is not meant as a flow chart or checklist in which, once dealt with, each item can be forgotten and the rescuers can move onto the next point. Rather, each priority must be anticipated, re-checked and re-considered throughout the rescue.

Priority 1 Assessment of airway, breathing and circulation

This is the highest priority in first aid for all emergencies. However, hypothermia is one of the few conditions in which the absence of a detectable pulse need *not* automatically mean that cardiopulmonary resuscitation (CPR) is required.

In severe hypothermia, respiration may be extremely shallow, and the heart beat can be very slow and weak. The pupils may be fixed and dilated. It may be virtually impossible to detect signs of life without an electrocardiograph. Because the chilled victim's need for oxygen is greatly diminished (by approximately 7 per cent per degree Celsius) this very low level of cardiorespiratory activity may be sufficient to maintain an adequate oxygen supply to the brain. This concept has been described as the "metabolic icebox". Attempts at CPR on such a patient may well trigger ventricular fibrillation and result in death.

The decision whether or not to initiate CPR on a hypothermic victim found in such an apparently pulseless state is still open to debate. Some authorities, such as Evan Lloyd of Scotland, have cautioned that CPR should not be commenced if a person is found apparently dead unless "there is a reasonable expectation that effective cardiopulmonary resuscitation can be continued, with only brief periods of interruption for movement, until the casualty can be transported to hospital where full advanced life support can be provided". However, other protocols, especially those from the United States, recommend that CPR should *not* be withheld unless the victim has other lethal injuries or a frozen chest that is impossible to compress, or the situation is too hazardous for rescuers.

Similarly, there is some dispute over the rate at which CPR should be performed. In some European nations it is advocated that, in order to avoid the dangers associated with hypocapnia (low carbon dioxide levels), CPR should be performed at only half the normal rate. This recommendation has only rarely been adopted in the English-speaking world.

Whatever choice is made, one thing is essential – that a central pulse (carotid or femoral) be checked for very carefully (preferably for a full minute), before any decision is made on whether or not the victim is indeed pulseless and hence requires resuscitation.

During prolonged CPR, the re-warming process should be continued. The definition of death is failure to revive on rewarming above 32°C.

Priority 2 Diagnosis

The correct diagnosis of hypothermia is important, because, as has just been discussed, the first aid management of this condition differs from that of other conditions for which cardiopulmonary resuscitation may be required.

1 *The first clue to diagnosis is a high index of suspicion* Always consider hypothermia in the following situations.
   - Adverse environmental conditions – low temperatures, high winds, wet conditions.
   - At-risk individuals – those who have been lost, injured, exhausted or inadequately clothed.
2 *Clinical assessment* The victim's signs and symptoms may also point to the correct diagnosis. Continual shivering, poor co-ordination, confusion, slurred speech, hallucinations and dilated pupils are all indications of mild to moderate hypothermia. Behaviour and the level of consciousness are the most important indicators of the victim's status – consciousness may be lost at core temperatures around 30°C. Profound hypothermia (under approximtely 25°C) may mimic death with

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Plate 9 *Severe frostbite in the toes of a Himalayan climber. The outcome was the loss of all toes. (Photograph courtesy of Mr D. Capps.)*



Plate 10 *Frostbitten fingers before thawing. The affected tissue is white, hard and ice cold. (Photograph courtesy G. Merril, Antarctic Division.)*

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Plate 11 *In this case of frostbite only the little finger on each hand has been affected. Three days after the freezing incident, blisters filled with clear serous fluid extend along the back of the fingers. (Photograph courtesy R. Kiernan, Antarctic Division.)*



Plate 12 *When, as in this case, the frostbite blisters are blood-filled, it is usually a sign of more extensive underlying tissue damage. This case was sustained near the summit of Mt Everest and eventually required amputation at the level of the first knuckle. (Photograph courtesy of Lincoln Hall.)*

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undetectable pulse, blood pressure and respiration, pupils lacking response to light stimulus, and muscle rigidity simulating rigor mortis. Remember, never assume the victim is dead until he/she has been re-warmed.

3 *Measurement of body temperature* By definition, hypothermia is a condition in which the core body temperature is less than 35°C. Therefore, to confirm the diagnosis and determine the level of hypothermia, it is important to obtain an accurate reading of the victim's core temperature. But at what site should the temperature be taken, and with what sort of thermometer?

Site of temperature measurement

- *Oral* Placing a thermometer under the tongue is a time-honoured method of assessing a fever. However, it is very inaccurate at assessing the core temperature in a cold field setting. Localised cooling of the mouth, especially if the victim has been mouth-breathing, can lead to falsely low readings.
- *Axillary* Temperatures recorded at the armpit are less reliable than rectal ones but more so than oral. Studies with induced hypothermia in a hospital indicate that axillary temperatures are consistently from 1.0°C to 1.5°C below core temperature. Whether this finding is transferable to the hypothermic victim in the field has not yet been established.
- *Rectal* A deep (12–15 cm) rectal probe gives a reasonably accurate initial assessment of a victim's core temperature. However, during re-warming it will usually lag behind true core temperature, resulting in the much-debated "after-drop" effect. In addition, it requires the loosening or removal of the victim's clothing – allowing further heat loss. Also, it is regarded as an intrusion by even semi-conscious casualties.
- *Oesophageal* There is evidence that of all the non-invasive means of assessing core temperature, a probe passed into the oesophagus through either the mouth or nose is the single most accurate and quickly responding method. Unfortunately, most conscious patients find it uncomfortable and practical difficulties (including the possibility of inducing vomiting) generally make it unsuitable for use in the first aid situation.
- *Tympanic* The ear drum or tympanic membrane shares its blood supply with the hypothalamus and, hence, is considered a good indicator of core temperature. The earlier sensors which had to be placed in contact with the eardrum (often painfully so) have been superseded in recent years by non-contact, infrared sensors. However, there is some evidence to suggest that after the subject has been exposed to cold and windy conditions false readings may occur because of local chilling effects – even after some time in sheltered surroundings. Whether any such inaccuracy is clinically significant has not yet been established.

Choice of thermometer

- *Standard clinical thermometer* The standard clinical thermometer as usually used in hospitals and homes is designed for assessing fevers, and records only down to 35°C and hence is of no assistance in assessing the level of hypothermia.
- *Low reading glass thermometer* Because of the above problem, special mercury–glass thermometers are available that record down to 25°C. However, they share the other problems common to the standard clinical thermometer – they are fragile, need to be very thoroughly shaken down for a hypothermic case, are slow to equilibrate and are difficult to read in poor light conditions.
- *Digital electronic thermometer* These are small, sturdy, easy to read and accurate. As opposed to the standard mercury–glass variety that takes at least 3 minutes to equilibrate, the electronic model gives a reading in 45–60 seconds. Disadvantages include the fact that they record only down to 32°C, can be inserted only 5–6 cm into the rectum, and have an ambient temperature working range of 10–40°C and, hence, in cold conditions must be kept warm (eg inside the rescuer's clothing).

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- *Thermistor thermometer* In most cases this is still the preferred method of monitoring core temperature. The probe can be inserted as far as required into the rectum (usually 12–15 cm) and does not need to be removed to be read. Until recently these were fairly bulky and expensive, but now, small, hand-held versions can be purchased for only a few hundred dollars.
- *Infrared tympanic thermometers* The advantage of such devices is that they are easy to use, give an almost instantaneous reading, and involve no undressing or discomfort of the victim.
- *Hands* Even if no equipment is available, the victim's companions can make a rough assessment of thermal status by slipping a hand under the patient's clothing into the armpit. If the patient feels as warm as usual, hypothermia can be excluded as a cause of unconsciousness.

In practice, because of all the difficulties listed above, rescuers rarely get to measure body temperature in the field. In such cases the best and most reliable indicator is the patient's mental state.

Priority 3 Gentle handling of the victim

The direct effect of the cold, poor oxygenation due to slow respiration and circulation, and accompanying acidosis of blood returning from the periphery, all result in the heart being in a state of increased irritability. Consequently, any rough handling of the victim can trigger ventricular fibrillation and subsequent death.

The need for gentle handling must be kept in mind at every stage of the rescue. The victim must be carried to shelter with great care. Wet clothes may need to be cut off. Wherever possible, evacuation should avoid transport across rough terrain.

Priority 4 Prevention of further heat loss

This is essential and can be achieved in the following ways.

- *Provide shelter urgently* Numerous lives have been lost when groups push on, attempting to get an obviously hypothermic victim to the comfort of a distant hut. At the earliest warnings of impending hypothermia the party should "go to ground". Even simple shelters such as snow holes and small tents have been known to save lives.
- *Insulate from the ground* The closed-cell foam mats commonly used by bushwalkers have been found to be very effective at decreasing conductive losses to cold ground, metal, snow or ice.
- *Replace wet clothing with dry* Wherever possible, wet clothing should be replaced – but only after the casualty is inside some form of shelter.
- *Cover the head* A variety of figures are often quoted, but two of the more reliable studies suggest that at –4°C up to 50 per cent of metabolic heat production can be lost through the uncovered head, and at –15°C this figure can go up to as much as 70 per cent.
- *Wrap in sleeping bag, space blanket or thermal recovery capsule*

  Space blankets. These sheets of metallised plastic were initially designed for use in outer space where most of the body's heat loss is by way of radiation. On earth the hypothermic victim can lose far more heat through conduction and convection. If space blankets are used, they must be carefully wrapped around the patient to minimise any air movement and resultant convective or evaporative heat loss. Similarly, because of the high conductive heat loss through the metallised material, it is essential that the victim wrapped in such material be insulated from the cold ground. Despite these shortcomings space blankets are often used because their small size lends itself to their inclusion in first aid or emergency supplies.

  Thermal recovery capsules. A number of these devices are currently available commercially. Some are made from various forms of space blanket (ie high infrared reflection) material, others from high insulation synthetics. Anecdotal evidence of their effectiveness is plentiful but there are few convincing studies to demonstrate that they are any more effective than a high-quality sleeping bag.

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- *Minimise respiratory heat loss* Heat loss from the respiratory tract can be minimised by wrapping a scarf around the nose and mouth, but only in such a fashion as to avoid obstructing respiration. Do not give oxygen which may have been chilled in transit to the field site unless it has been re-warmed (and preferably humidified); otherwise, it can be a considerable source of further heat loss.

Priority 5 Provision of additional heat

This is another area of controversy with regard to the management of hypothermia, some experts believing that active heating should not be performed outside a hospital. However, it must be acknowledged that, in a remote setting, provision of additional heat in the field may be essential – if only with the intention of stabilising the core temperature rather than actually re-warming the victim. If it is necessary to provide additional heat, a number of options are available.

- *Generalised warming of a shelter, hut or tent* This is a not a very efficient method but is often all that is required in mild cases. Avoid overheating the shelter – approximately 18–22°C is sufficient. The main danger to be avoided is placing the victim in front of a raging fire or radiant heater – this may result in a too rapid opening up of constricted vessels in the periphery and a subsequent drop in blood pressure.
- *Warm drinks and high-energy food* These have both physiological and psychological value, but such drinks and food must be given only to a conscious victim. Alcohol and strong caffeine drinks are best avoided.
- *Body-to-body contact* A warm volunteer can strip off the clothing covering the torso (leaving the trousers in place – studies indicate that extremely little heat is transferred via the legs) and share a sleeping bag with a similarly stripped victim. Again, this is not a very efficient method but in many circumstances it may be all that is available.
- *Heat packs* There are a variety of these available on the market. Most rely on a slow exothermic heat reaction which is initiated by exposure to the oxygen in the air. Some packs can even be re-charged by boiling in hot water. They must be used cautiously, as some are capable of burning the skin unless covered. They should be packed around areas such as the neck, sides of the chest, armpits and groin.
- *Airway re-warming* There is some dispute about the effectiveness of attempting to re-warm a hypothermic casualty through the provision of warmed breathing gas. What is not often recognised is that the real advantage of such a system is not the heat gain that it imparts so much as the respiratory heat loss which it prevents. There are several devices on the market which can deliver such heated and humidified gas in both the field and hospital setting.

Priority 6 Evacuation

Any evacuation plan must take all of the above listed priorities into consideration.

- It should allow for frequent assessment of the airway, breathing and circulation.
- It must ensure that there is no further heat loss.
- It should ensure that any rough handling or movement of the patient is kept to a minimum.

If these conditions cannot be met, it may well be better to take any required personnel and equipment to the victim and treat on-site.

After recovery, cases of even moderate severity should usually not be moved for approximately 24 hours – by which time the victim's energy reserves are likely to be replenished.

Prevention of exposure hypothermia

- Education and training are essential – for both the general public and field party leaders.
- Adequate clothing should be worn or carried – remember the layer principle.
- Adequate shelter – bivvy bags, tents etc. should be carried at all times.
- Ensure adequate hydration and high-energy nutrition.
- Never ignore shivering.
- Get to shelter early – do not force the party on when any member is apparently hypothermic.

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- Take special note of anyone who is injured, shivering, lagging behind or acting strangely.
- Learn to assess weather conditions, especially rain and wind.

URBAN HYPOTHERMIA

The term "urban hypothermia" is useful to cover a wide variety of conditions. As the name implies it commonly occurs in the home or in a moderately sheltered urban setting. Unlike immersion or exposure hypothermia, where a person with normal heat production and conservation is overwhelmed by very cold temperatures, urban hypothermia normally occurs in situations where the victim has either impaired heat production or decreased ability to conserve heat, and in only moderately cold settings. The type of scenario often encountered includes that of elderly people in chronically under-heated housing, neonates and infants with poor thermoregulation (Figure 21.4), sudden disablement (eg fractured hip with a long interval before discovery), hypothyroidism and other endocrine disorders, head trauma, spinal cord transection, drug-induced coma, a variety of extensive skin conditions, burns, patients on various therapeutic drugs such as phenothiazines, and derelicts sleeping out. On a world-wide basis, far more cases of hypothermia are attributable to these causes than to exposure or immersion.

In all these cases the onset is usually slow, sometimes over days. This allows time for a number of physiological changes to develop, such as a fall in circulating blood volume, sodium and potassium depletion, and a variety of other biochemical changes, all of which can complicate treatment. Consequently, this group often has a much higher mortality rate – in spite of the fact that access to ambulance and hospital facilities is usually much easier than in immersion or exposure hypothermia.

Mild levels of urban hypothermia are probably much more common than is generally recognised. A high index of suspicion needs to be maintained if this condition is to be detected and, hence, adequately treated. It should especially be considered in cases that involve the very young and the very old; in the colder months; in those living in under-heated homes or shelters; in conditions of poor nutrition, poverty and social isolation; in those affected by drugs and alcohol; and finally, in any patient where the mental state is lower than might be expected from the more obvious medical problem.

Treatment of urban hypothermia

As in the case of exposure hypothermia, the priorities are to maintain airway, breathing and circulation; to handle the victim gently; and to prevent further heat loss. However, in the first aid situation there is rarely a place for providing additional heat to cases of urban hypothermia, and certainly not for attempting rapid re-warming. All cases need to be transported, as gently as possible, to a hospital where careful monitoring, slow re-warming and the anticipation of complications are the keys to a successful outcome.

IMMERSION HYPOTHERMIA

Immersion hypothermia can occur not only as a result of water sports, such as swimming, diving, and kayaking, but also following accidental immersion, such as shipwreck or falling into cold water. The signs and symptoms of immersion hypothermia are much the same as for exposure hypothermia, but come on far more rapidly because of the much greater thermal conductivity of water.

Immersion hypothermia can be complicated by the likelihood of drowning or near-drowning. The muscle weakness and rigidity which accompany hypothermia can severely interfere with the victim's ability to swim or stay afloat – the victim may drown long before hypothermia reaches a level which would be lethal in itself.

The question of how long a person can survive in water at a particular temperature is one that is frequently posed but has no easy answer, as so many factors are involved. Among the more important considerations that determine an individual's core body temperature following immersion are

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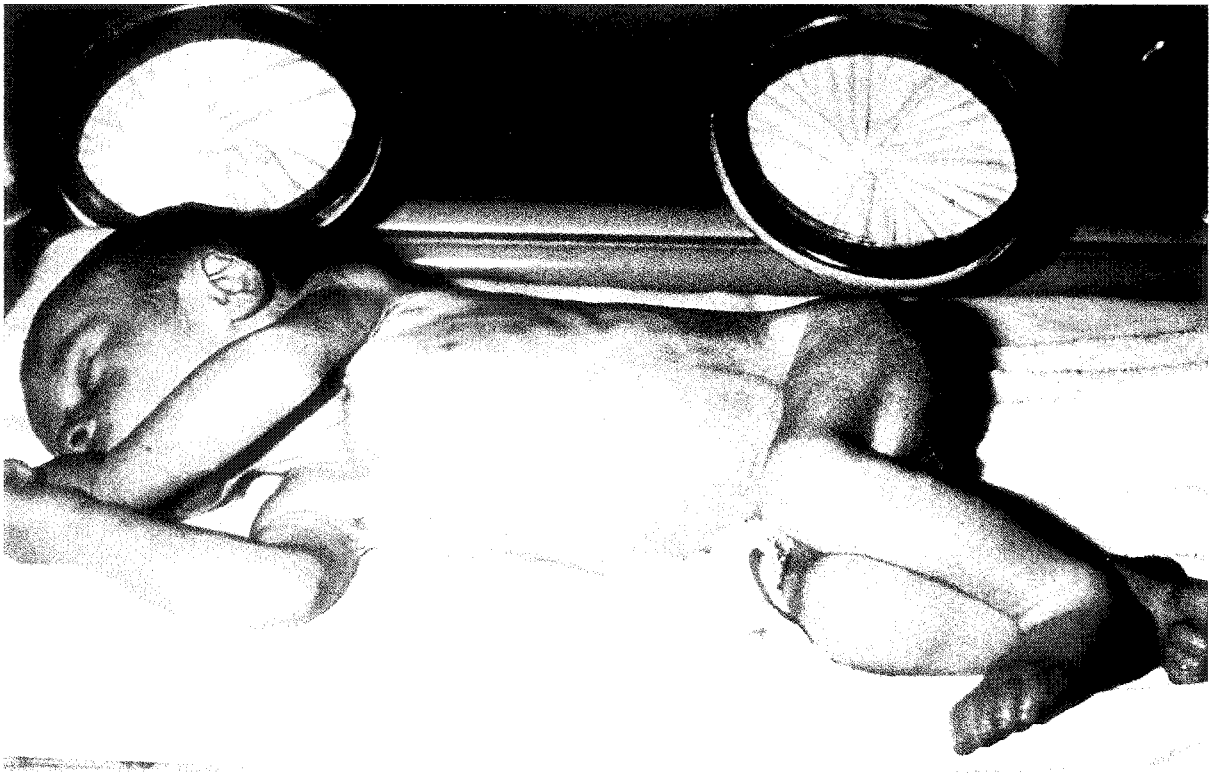


Figure 21.4 *Hypothermia in a subtropical, urban environment. The very young are particularly susceptible. Here the feet and hands are cyanosed and blotchy, and the skin of the abdomen is white and waxy. This newborn infant, found abandoned in a rubbish bin in Brisbane, did not survive. (Photograph courtesy of J. Pearn.)*

- water temperature,
- duration,
- insulation (fat and clothing),
- level of activity, and
- weather conditions (especially wind and wave action).

Life jackets, wet suits and immersion suits can prolong survival time many fold (Table 21.2).

The amount of voluntary movement made in the water also substantially affects the rate at which hypothermia develops. This is because swimming dissipates the layer of warmed water which might otherwise form around a person. Exercise will reliably raise body temperature only if the water temperature is above about 24°C. Hence, it is generally better to curl up into the heat escape lessening posture (HELP) than to attempt to swim any significant distance to safety (Figures 21.5–21.7).

Treatment of immersion hypothermia

The usual ABC of resuscitation applies – airway, breathing, circulation. A low reading thermometer can be helpful in distinguishing between immersion hypothermia and near-drowning.

Prevention of immersion hypothermia

- Always wear life jackets when there is a risk of immersion.
- Wear as much clothing as possible, even in the water, especially headgear.
- Do not exercise in an attempt to stay warm or attempt to swim more than a very short distance.

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- Adopt the HELP or huddle position (Figures 21.5–21.7).
- Survivors of boating mishaps should attempt to get as much of their bodies out of water as possible (eg by climbing onto a capsized boat). The convective and evaporative heat loss brought about by even strong winds will be less than the conductive loss to cold water.
- The victim should be recovered from the water as quickly as possible. Ideally, this should be done with the casualty in a horizontal position so as to reduce any shock or hypotension caused when "the hydrostatic squeeze" is removed as the victim is plucked from the water. This is a counsel of perfection and not always possible. However, there are both commercial and home-made devices available (known as "Jason's cradles") which are designed to achieve this goal.

NOTES ON ADVANCED LIFE SUPPORT FOR ALL FORMS OF HYPOTHERMIA

- Despite oft-repeated warnings that endotracheal intubation may precipitate ventricular fibrillation in the hypothermic victim, there appears to be little evidence to support this claim.
- Intravenous fluids should be warmed to about 42°C. Central lines are preferable to peripheral, as considerable cooling of I.V. fluid can occur in the links before reaching the core. In addition, peripheral veins are often hard to cannulate in the severely hypothermic victim.
- Avoid Lactated Ringer's Solution (Hartmann's) as the cooled liver may not be able to metabolise lactate.
- At core temperatures of under 30°C the heart is usually unresponsive to cardioactive drugs. Not only will such drugs be ineffective in a severely hypothermic victim but, as re-warming proceeds, may become pharmacologically active – with undesirable consequences.
- Defibrillation is unlikely to be successful until the core temperature has been raised above 30°C.
- The high electrical impedance of cold skin may make it impossible to obtain a decent ECG trace. Needle electrodes through the skin may be required.
- Shivering artefact can also make ECGs difficult to interpret.
- As well as bradycardia, J waves (or Osborn waves) may be found on the ECG. These are small positive deflections following the QRS complex. While not diagnostic they may act as an indicator or reminder of hypothermia.

Table 21.2 Estimated survival time in hours (with a 95 per cent confidence range) for lean subjects in rough seas at 6.1°C. (Based on Steinman and Kublis (1986).)

Clothing type	Time to incapacity (34°C)	Time to unconsciousness (30°C)	Time to cardiac arrest (25°C)
Light clothing	0.4–13	0.8–2.6	1.3–4.3
4.8 mm wet-suit	1.6–4.7	3.1–9.9	4.9–16.2
Insulated dry coveralls	2.9–8.8	5.7–18.2	9.1–30.0

It should be noted that incapacity, and hence death from drowning in an individual not wearing some form of flotation, would occur in less than one-third the time it would take for the individual to die from a hypothermia-induced cardiac arrest. An immersion suit, worn on top of high insulation underclothing, provides both flotation and a much longer survival time.

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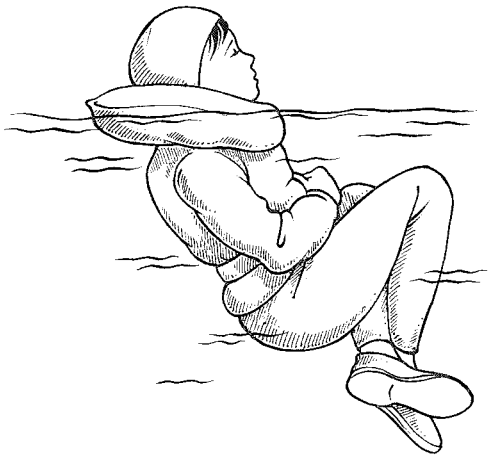


Figure 21.5 *The heat escape lessening position (HELP). By decreasing the body surface area and protecting the groin, armpits and torso, the victim can lessen heat loss. (Illustrations for Figures 21.5–21.7 courtesy of Dr T. Cochran.)*



Figure 21.6 *The modified HELP position. In rough sea conditions adopting the standard HELP position can result in the individual rolling sufficiently to frequently submerge the head in the cold water. In such cases it is better to uncurl the legs, though keeping them crossed at the ankle. This will provide greater stability as well as some degree of heat conservation.*

FROSTBITE

In conditions of subzero temperatures, heat loss from peripheral tissue may be sufficient to allow ice formation within those tissues. This freezing of the extremities or exposed areas is called "frostbite". As it involves a living organism, the processes involved are much more complex than in simple freezing, such as occurs when a piece of meat is placed in a freezer. Ice crystals can form either within cells, rupturing cell walls, or between cells. Growth of intercellular ice crystals draws out water from the adjacent cells, changing the osmotic pressure and causing tissue death (Figure 21.8). In addition the cold itself damages the walls of the capillaries in the affected area, allowing plasma to leak out. Consequently, the blood flow (which has already been decreased to the body's periphery by vasoconstriction) is further

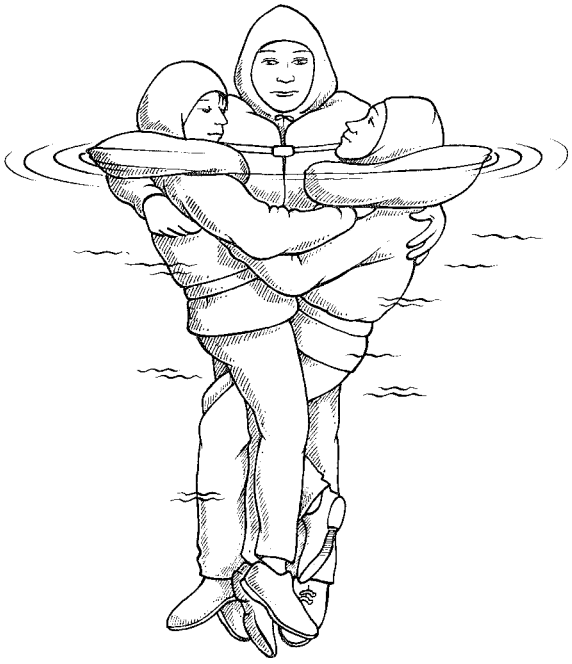


Figure 21.7 *The huddle position. Heat loss can be decreased when members of a group huddle together, providing some reduction of convective heat loss from the torso.*

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impaired. As a result, blood cells and platelets sludge and stick together causing microemboli (small blockages) thus further decreasing oxygenation to the already damaged tissues.

In comparable conditions of cold, frostbite is more common at high altitude because of the decreased oxygen levels of tissues. Other contributing factors include wind-chill, exposure to cold metals or fuel (accelerating conductive and evaporative losses respectively), constrictive clothing (eg tight socks) which can impede the circulation to the extremities, wet clothing, injuries (eg a broken limb), dehydration, and general hypothermia (the body may need to "sacrifice" areas of the shell to try to preserve heat in the core). There is also some evidence to suggest that there is a higher risk in those who smoke or have had a previous episode of frostbite.

Theoretically, frostbite can be classified into a number of different grades of severity depending on the depth to which the tissue is frozen. Frostnip is an early, reversible stage of frostbite in which the affected tissue blanches and loses sensation; there may be minor frosting of the skin surface but it remains pliable. Superficial frostbite is said to occur when freezing involves only the skin and subcutaneous tissues. Deep frostbite involves the underlying muscle, nerve, blood vessels and even bone. At the initial presentation, it is often impossible to assess accurately the extent of the damage. However, Table 21.3 may be used as a general guide.

Signs and symptoms

Frostbite usually appears on the exposed areas of the body, such as the nose, cheeks and ears, and on the extremities, especially the fingers and toes. It presents as firm, cold, white areas. One of the first signs is that the affected area *stops* feeling cold and painful. Remember – if an area ceases to feel painfully cold – investigate!

In frostnip, ice crystals form only in the already dead, outermost layer of the skin. On re-warming, the underlying live tissue may show an inflammatory response with redness and swelling which may last for several hours. Peeling of the skin, rather like sunburn, may occur 24–72 hours after re-warming; sometimes mild hypersensitivity to cold persists for months or even years.

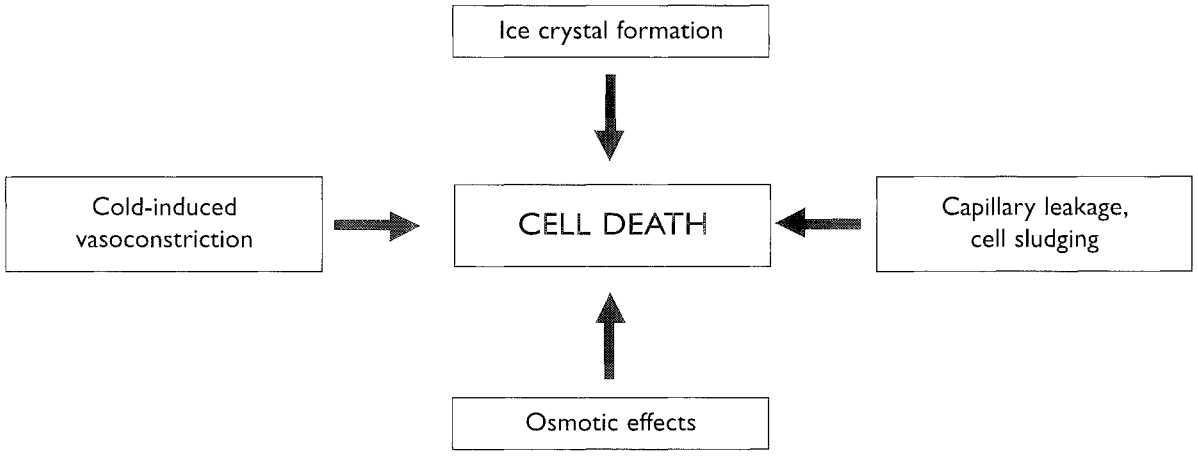


Figure 21.8 *Mechanisms by which frostbite brings about tissue destruction.*

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In true frostbite the affected area is cold, hard, and anaesthetic. The skin is either waxy-white or a mottled blue-grey colour. On warming it becomes blotchy red, swollen and painful. Blisters commonly develop after some hours of re-warming and may cover entire fingers or toes – especially on the dorsal side. If the blisters contain clear fluid, the underlying tissues may still be viable and, in general, tissue loss is less than if the blisters are blood-filled. After a week or so the frostbitten tissues develop a thick black covering called an "eschar". Over several weeks or months this dead tissue will shrink and slough off. With superficial frostbite, the eschar may be only a few millimetres thick and, when it peels off, fresh pink skin will be found underneath. With deep frostbite, loss of far more tissue occurs – when the eschar finally separates, skin, tissue and even bone may peel away.

Treatment of frostnip and frostbite

Treatment can be divided into three sections: self-treatment for frostnip and small areas of superficial frostbite, hospital treatment for more serious cases, and field treatment for victims isolated from hospital facilities.

Self-treatment

Frostnip and small areas of superficial frostbite should be treated immediately by gently placing the affected fingers in the opposite armpit, or by placing a warm hand over a frostnipped cheek or ear, but remember *never rub*. Feet can be re-heated on the warm abdomen (under a parka) of a companion. Re-warming will be accompanied by the return of sensation – often painfully so. Once the colour and consistency of the skin have been restored the person can safely resume normal activity but must be especially vigilant against recurrence.

Hospital treatment

Frostbite which involves entire digits, a foot or a hand, is a serious condition and the first aider should endeavour to get the victim to hospital as quickly as possible, attempting to avoid both thawing and further freezing en route. At hospital the affected area will be treated by rapid re-warming in a water bath at 38–42°C (unless already thawed).

Field treatment

The decision to treat frostbite in the field can be an extremely difficult one, and a number of factors need to

Table 21.3 The various stages of frostbite with accompanying signs and symptoms. (Based on "Frostbite" Chapter 10-9 *First on the Scene, Complete Guide to First Aid and CPR*, St John Ambulance, Canada, 1995.)

Stage	Description	Signs and symptoms
Frostnip	Only the top layer of the skin is frozen. Usually affects the ears, cheeks, nose, fingers and toes.	White, waxy-looking skin. Casualty feels no pain.
Superficial frostbite	The full thickness of the skin is frozen. This follows frostnip when further heat loss is not prevented and freezing continues.	White, waxy-looking skin. Skin is firm to touch, but tissue underneath is soft. May feel pain at first, followed by numbness.
Deep frostbite	The skin and the tissues underneath are frozen, sometimes to the bone. May involve an entire hand or foot.	White, waxy-looking skin that turns greyish blue as frostbite progresses. Skin feels cold and hard. Complete absence of sensation inc. pin-prick.

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be considered: the severity of the case, the expected time before evacuation is possible, the facilities available, and the experience and state of health of other members of the party. The following points also should be borne in mind.

- Never thaw a part if there is any likelihood of its being refrozen. Thawing and refreezing causes far more tissue damage than continuous freezing.
- An individual with a frozen foot is not necessarily a stretcher case – but will become so once the foot is thawed, swollen and painful.
- In general, field re-warming should not be attempted if more definitive medical treatment can be reached within two hours.

If, after taking all of the above into account, it is determined that it is necessary to treat a frostbitten patient in the field, the following procedures should be observed.

- Set up camp or some sort of shelter so that further heat loss from the patient is minimised.
- Check to see whether the patient has a degree of hypothermia as well as frostbite. Re-warm if necessary.
- As with hypothermia, rehydration with warm drinks is important.
- Remove any constriction to circulation (eg rings).
- Removal of boots or gloves must be effected gently, to avoid further trauma to the affected area.
- Avoid using radiant heat (fire or radiator) or rubbing of the affected area.
- Immerse the affected part in a tub of water at 38–40°C. Use as large a volume of water as possible – a small pan will very quickly cool below the effective temperature once a frozen extremity is put in it. Agitate the water to ensure even distribution of heat. Continue until the thawed part flushes – this may take 20–30 minutes.
- Thawing will be extremely painful – give analgesics if available.
- After thawing, gently dry the affected part, cover with a sterile dressing and elevate.
- Do not burst the blisters unless sterile conditions are possible. There is some evidence to suggest that there are advantages in draining blisters that contain clear fluid – but such advantages are lost if infection occurs.
- Give aspirin if available – not only for its pain-killing properties but also for its ability to inhibit prostaglandin release. Prostaglandins are chemicals produced by the body that are involved in promoting inflammation and have been found in large quantities in frostbite blisters. Although aspirin has not yet been proven to be effective, it is also unlikely to do any harm, and it is one of the few remedies likely to be available to an isolated party.

Prevention of frostbite

- Proper clothing and well fitting boots are especially important. Mittens are better than gloves and in windy conditions, should be secured by a harness. Adequate headgear is important for both hypothermia and frostbite. Avoid tight clothing that can restrict the circulation. Clothes need to be kept clean; otherwise the insulating layer of trapped air is diminished.
- Avoid generalised body cooling, exhaustion and fatigue.
- Avoid sweating into high insulating clothes – use the layer principle to adjust clothing assemblage to the task.
- Maintenance of good hydration and nutrition are important.
- A wind-chill chart (see nomogram, Figure 21.2) should be consulted to estimate the equivalent temperature and then individuals should dress and act accordingly.
- Employ a buddy system in which people watch each other for the early tell-tale white areas on cheeks, nose, and ears.

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CHAPTER TWENTY-TWO

POISONING

John Pearn

Attending a case of poisoning can be one of the most stressful challenges for the first aider. After the accidental or suicidal ingestion of harmful substances there is usually a "window" of time before symptoms develop, and relatives or bystanders always expect the first aider, paramedic or doctor to "do something". If it is the first aider's own child or grandchild who has swallowed something nasty, the situation can be doubly stressful. Perhaps in no other area of first aid is there greater potential for doing harm by some types of misguided intervention.

Accidental and suicidal poisonings are common and together comprise an important public health issue. In 1990 the American Association of Poison Control Centres (AAPCC) documented 1.7 million consultations which followed human ingestion of poisons. This annual survey did not include fatalities or suicidal drug overdoses, 70 per cent of which are not reported to Poisons Centres.

The first aider may be confronted with any one of five types of poisoning.

1 Children, almost always in the one- to three-year-old age group, who have swallowed tablets or chemical fluids.
2 Adults, accidentally poisoned in industrial, farming or household incidents.
3 Accidental drug overdoses, usually involving teenagers or younger adults, involving alcohol, narcotics or other substances of abuse.
4 Adults who have attempted suicide.
5 Homicidal attempts to poison adults or children.

The principles of first aid and of pre-hospital care remain the same in each of these scenarios. The D for Danger in the St John Ambulance DRABC mnemonic (Danger, Response, Airway, Breathing, Circulation) is particularly apposite in the initial management of a victim who has been poisoned. Some substances, such as cyanide and pesticides of the organophosphate group of chemicals, may still be on the victim's skin, face or clothes, and have the potential to injure those who administer care. Other victims intoxicated by gases may still be in dangerous environments which are a potential risk to rescuers and first aiders. Those poisoned by alcohol or drugs may be belligerent or combative, as also may be their colleagues or fellow victims in group intoxications. These threats of secondary poisoning or injury to the first aider are potential rather than realised risks and, although there are occasional case-reports of secondary symptoms experienced by contaminated paramedics, in practice the risk is very small indeed. Much greater is the risk of secondary injury – such as drowning, suffocation, inhalation of vomitus or injuries from falls – to the primary victims themselves. Here, the first aider's role is paramount.

After a quick assessment of the accident scene and attention to airway, breathing and circulation, if necessary, several specific actions are essential. A sample of the tablets, drugs, fluids or powder must be collected, as well as the containers, packets, bottles or tins involved. Of all accidents and incidents which involve first aiders, poisoning incidents lead to the greatest likelihood of help being needed by police and coroners – in addition to that required downstream by emergency care

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physicians and intensivists. Thus suicide letters must be noted and kept, and discarded wrappers or packets, or needles and syringes, observed and recorded.

In all parts of the world specialised help now exists for those attending the victims of poisoning – help in the form of specialised information from national, state or local Poisons Information Centres and Poisons Hotlines. Such consultation, initiated by the first aider or paramedic, is part of the pre-hospital management of each case. This is because victims ingest or are poisoned by any one of many thousands of agents, many of which are obscure; detailed knowledge of all of them cannot be expected to be the province of any single individual.

NAMES OF CHEMICALS AND DRUGS

There are more than five million known and described chemicals. Of these, some 35 000 are used in industry, manufacturing and commerce. More than 3000 are used as drugs in medicine. All are potentially involved in accidental or suicidal poisoning.

Every drug used in medicine has at least four names. As an example, in the case of aspirin, these names are as follows.

1 The chemical formulation – 2-(acetyloxy) benzoic acid.
2 The chemical name – acetylsalicylic acid.
3 The Generic or Approved Name – aspirin.
4 Trade names such as "Disprin" or "Aspro Clear".

The Generic or Approved Name is given by bodies such as the British Pharmaceutical Council in the case of new drugs developed in the United Kingdom, and by the United States Adoption Names Council (the USAN Council, established in 1964) in Washington. The Generic Name is the common name usually used internationally. The first aider should be aware that a single drug or chemical may have up to thirty trade or brand names. All Poisons Information Centres have computerised cross-referencing systems which make order out of this potential chaos, and save the first aider, the paramedic and the doctor from wallowing in a communications nightmare.

A *poison* is defined as "any substance – liquid, solid or gaseous – which impairs cell function, impairs health or destroys life". Poisons are absorbed by ingestion, by inhalation into the lungs, and by direct absorption from the skin or delicate mucous membranes of the body, such as the conjunctivae.

A *toxin* is a poison made by a living organism. A *venom* is a toxin which is made in a specialised gland and injected into the victim by a specialised device such as a snake's or spider's fang.

HOW DO POISONS ACT?

Poisons, whether they be inorganic chemicals or toxins or venoms, act in many different ways. Some natural substances, such as common salt, become poisons simply if they are ingested or injected in an excessive dose. Some substances depend for their toxicity on their concentration. Thus some corrosives, such as caustic soda, are mildly irritating in very weak solutions but can cause frightful burns in higher concentrations. Time of contact is also very important – snake venom which is quickly neutralised by antivenene (antivenom) may produce no damage at the cellular level but, if the antidote is not given for several days, permanent damage to cells will have already occurred and the late use of antivenom may be of no help.

Some poisons act by direct contact, destroying cells with which they come into contact. Acids and alkalis on the skin, in the eyes and in the stomach are examples. The body's cells are damaged by extremes of pH which cause the cell membranes to break down and the cell contents to be destroyed.

Some poisons act by being absorbed into cells, where they stop cell metabolism. Cyanide acts in this way – it binds itself very quickly to the atoms of ferric (Fe) iron in molecules of the enzyme cytochrome oxidase which is essential for oxygen metabolism ("cell respiration") in every body cell. The poisoned cell switches instantly to its emergency bypass anaerobic metabolism but, if rescue is not achieved, the lactic acid debt becomes too great and the cell stops working. As this affects all body

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cells, death quickly results, as attested by witnesses of victims in United States judicial gas chambers.

Many chemicals act in this way, turning off vital metabolic processes in cells. Other chemicals are poisonous because they stimulate cells too much. The anticholinesterases – used widely as pesticides in agriculture and animal husbandry – act in that way.

Some dangerous and common poisons are not toxic in themselves, but are changed in the body to harmful substances which then do the damage. Methanol (methyl alcohol) is one such example. After ingestion it is absorbed and circulates in the blood stream. As the methanol passes through the liver it is converted to formic acid which is highly toxic and it is this which can permanently damage or kill the body. In this case, the antidote, ironically, is ordinary ethanol, which occupies the liver cells in its own metabolism, thus partly keeping away the potentially deadly methanol. This type of action is called "competitive inhibition".

The body's organs and cells react to these poisonous insults in many ways. Mammals have evolved a wondrous array of reflexes which protect them automatically from death or injury if poisons are absorbed.

If corrosives or acids or too-concentrated solutions are swallowed, gagging occurs. Vomiting often occurs to expel the offending poison. The gastro-intestinal tract may increase its peristaltic rate dramatically, to reduce the time available for absorption of an ingested toxin or chemical. In the case of inhaled toxins, often the larynx goes into spasm in a short-term attempt to protect the airway or lungs.

If acids or alkalis have been swallowed, buffer systems come into play with body chemicals combining with the poison to reduce its corrosive or burning effects.

The body has a wonderful capacity to reject, expel, dilute and detoxify poisons. The whole aim of first aid and pre-hospital care under these circumstances is to keep the patient alive – a clear airway, a beating heart and circulating oxygen-carrying blood – whilst these compensatory mechanisms are occurring naturally. In practice antidotes are only rarely used, and then almost always only by doctors in the controlled and monitored environment of the emergency department or intensive care ward. Occasionally, in specific circumstances, the first aider will help expulsion of swallowed poisons by inducing vomiting. In some circumstances this action is very important.

POISONINGS IN CHILDHOOD

All young children experiment by eating plants, berries, tablets, powders and dirt, and fluids of all types. Children are admitted every day to every major hospital having swallowed liquid fertiliser, dog repellant, mother's oral contraceptive pills and grandfather's heart tablets. The Top 10 swallowed substances causing parents and health professionals to ring Poisons Information Centres are:

- tablets and liquids used to treat the symptoms of colds and respiratory tract infections
- household and garden plants and berries
- simple analgesic tablets (aspirin and paracetamol)
- detergents (including toilet bowl cleaners)
- liquid fuels (paraffin, kerosene, petrol)
- household disinfectants
- cosmetics (eg perfumes, lipstick, toothpaste, deodorants)
- antihistamines
- oral contraceptives
- household bleach

Often the first aider or paramedic is confronted with a smirking toddler reeking of petrol or kerosene (paraffin) and a hysterical mother. Sometimes a child will be maddened or shocked with pain from mouth and oesophageal burns from eating dishwashing powder. Occasionally, a child will be comatose from eating sedative tablets. In each case the initial first aid drills – danger assessment, airway maintenance, artificial ventilation and external cardiac compression if needed – are the same. Children can deteriorate rapidly in a poisoning situation. It is also the exception rather than the rule to know just how much fluid or how many berries or tablets a child may have swallowed. For these reasons, admission to hospital for assessment is the rule in every case. If more than one first aider is present, it

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should be the most experienced first aider who remains with the child until handover to full medical assessment.

In 1994, the Department of Human Services and Health in Australia conducted a large survey which showed that the general public does not appreciate that the following chemicals in particular are potentially very dangerous to children:

- camphor
- iron tablets
- eucalyptus oil
- quinine tablets
- alcohol (in children)
- mothballs (naphthalene)

Studies by the author have shown that the admission: fatality ratio following the ingestion of poisons by children is some 2000:1. Thus death is now rare. Those children who do die, do so as a result of swallowing:

- tablets for heart disease (eg digoxin)
- drugs used to treat asthma
- poisonous plants (especially oleander and mushrooms)
- drugs used to treat psychiatric depression (eg tricyclic antidepressants)
- caustic soda and similar corrosives

Almost all children who are admitted alive to hospital, following the ingestion of poisons, not only survive but also have no after-effects. Some who have ingested corrosives and acids are left with damage to the face and gastro-intestinal tract; a very small proportion are brain damaged from lack of oxygen induced by coma or by the direct effect of the poison on body cells. A common cause of admission to hospital is the swallowing of button batteries used to power hand-held toys or computer games. In a small proportion of such cases these disintegrate before being expelled in faeces and can cause problems of stricture, or absorption of toxic metals.

In any poisoning incident involving a child, there is the parent to care for also. Parents are understandably always distressed, often sick with guilt, sometimes angry with partners or other relatives, and sometimes hysterical. Experienced paramedics handle the whole situation knowing that statistically it is highly likely that all will turn out very well if correct procedures are followed.

ADULT ATTEMPTED SUICIDE

In completed suicides the first aider will attempt resuscitation, but it is ordinarily the coroner who has the major role. Much more challenging is the situation where the victim has swallowed drugs or poisons and is alive, and often not only alive but in that window of time before any symptoms of ingestion have yet appeared. Most deaths from suicidal overdose occur outside hospital but, if a patient can be kept alive after ingesting drugs or poisons and escorted to hospital alive, the survival rate varies from 94 to 99 per cent. The main causes of death relate to the toxicity and dose of substance swallowed, to delay in being found by paramedics or first aiders, and to the aspiration of vomitus.

A large Australian study of 1000 consecutive overdose patients admitted to a busy emergency department over a 6-year period shows (Table 22.1) the rank order of most common usage of drugs and chemicals used by would-be suicides who reached the emergency department alive.

Table 22.1 Incidence of various drugs in 1000 overdose cases

Benzodizepines (eg Valium)	44%
Alcohol (ethanol)	35%
Tricyclic antidepressants	19%
Paracetamol	15%
Phenothiazines (eg Largactil)	11%
Antihistamines	8%
Opioids (heroin, morphine, pethidine)	8%
Anticonvulsant drugs	4%

These figures total to more than 100 because almost half (48 per cent) of those who attempt suicide by ingesting drugs take more than one preparation.

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Those victims who suicide successfully tend to swallow fatal overdoses or a large dose of a single drug or a toxic chemical such as the pesticide paraquat, hydrochloric acid, or overdoses of antidepressants or heart drugs.

THE DRUG SCENE – NARCOTICS OVERDOSE

Narcotics and cocaine are not commonly employed as suicide drugs. However, admission to hospital from inadvertent narcotic overdose is common, due to mistakes in intravenous shoot-ups and in underestimating the strength of some street-supplied drugs, which are of notoriously variable quality and concentration. If such victims of narcotic overdose can be kept alive by correct first aid, a specific antidote, naloxone, can have dramatic salvage effects. Comatose patients can be awake within minutes after such antidote rescue. Thus the history taken at the pre-hospital scene can be of great help to the medical team in the emergency department. The first aider or paramedic should try and obtain information about drug-taking habits, previous overdoses and recent events. A search of the victim's clothing, the room or car where they were found, and their other belongings may reveal drugs such as cocaine, other narcotics, filter-paper impregnated with LSD, or instruments or paraphernalia used in drug taking.

One new and disturbing syndrome is that of the teenager who has taken drugs, often for the first time, at a night club or discotheque. The drug Ecstasy and amphetamine are often involved. Effects come on within several hours and can be fatal. Anyone who lapses into coma under such circumstances is at grave risk. The pre-hospital drills of ABC (airway, breathing, circulation) must be continued until life support systems are connected at the intensive care unit.

AT THE ACCIDENT SITE

The victim may be found in circumstances which indicate a planned suicide attempt, or a scene of drug experimentation such as glue or solvent sniffing by teenagers. In the case of young children there may be foil strips of tablets some of which are missing, or bottles of fluid. A not uncommon cause of accidental poisoning in childhood is the action of adults in putting solvents and petrol into used soft-drink bottles or reused food containers.

Smelling the victim's breath or clothes often yields helpful information. Paper or plastic bags and the smell of petrol or glue will alert the paramedic to the likelihood of toluene abuse. Kerosene (paraffin) or dishwashing detergent may have been vomited after ingestion and may be saturating the victim's clothes. Ketones on a victim's breath smell fruity and indicate ketoacidosis – smelt in diabetic coma as well, but sometimes indicating acute alcohol poisoning. The garlic-like odour of arsenic, the almond-like odour of cyanide and the rotten-egg odour of hydrogen sulphide (sewage gas) all give clues to the danger of the accident site as well as the type of treatment medical officers may wish to use if the patient can be kept alive en route to hospital.

Pinpoint pupils and needle-tracks over veins suggest the likelihood of narcotic poisoning.

Whatever poison has been ingested and whatever the age of the victim, three clinical issues will confront the first aider at the accident site – altered consciousness, seizures, and temperature changes.

Altered consciousness

Most patients who are severely poisoned are confused, unsteady and incoordinate, and have slurred speech. This may progress to coma. The timed documentation of these states, and particularly the rate of change, is very important indeed. Downstream decisions about whether to induce emesis or not, or whether to use specific antidotes, are often partly determined by such information. Some poisoned patients are hallucinating. This is seen particularly in children who have ingested antihistamines and in adults experimenting with mushrooms and "designer" amphetamine-derived drugs.

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Seizures

Many patients who are severely poisoned start to convulse before they reach hospital, and the first aider or paramedic has to deal with this complication as well. Epileptic fitting can occur as the direct result of the stimulatory effect of a poison such as anti-asthma drugs or decongestants, or can occur secondarily from lack of oxygen due to depression of other vital centres, or can result from withdrawal of certain drugs such as alcohol or narcotics.

Temperature changes

Most poisoned children lose heat rapidly because of reduction of metabolism and because of coma. Loss of tissue perfusion can cause the temperature to drop rapidly. Many comatose victims who have attempted suicide with depressant drugs such as barbiturates, benzodiazepines (such as Valium) or alcohol are severely hypothermic when found. Gradual warming, and the prevention of further hypothermia, by protection with space blankets is usually all that is needed in the pre-hospital setting. Some poisoned victims are grossly hyperthermic and are in danger of "cooking up". Atropine overdoses and the ingestion of atropine-containing plants such as corkwood *(Duboisia)* can cause this syndrome, as also can overdoses of cocaine and amphetamine derivatives. If the patient is manic or incoherent and feels burning hot to the touch, cooling with wet cloths and exposure are helpful.

Pre-hospital care

Observe the DRABC protocol (Danger, Response, Airway, Breathing, Circulation). First aiders will protect themselves and their patients by identifying any further risk of poisoning, by rescue if need be, and by a quick history and assessment of the scenario. They will test and document the state of consciousness to form one baseline for assessing further deterioration as the effects of the poison develop, they will maintain the airway if the poison has acted directly on the protective reflexes which keep a gag reflex intact, and they will maintain CPR if the poison has overcome the functioning of the respiratory centre in the brainstem and the beating of the heart itself. The nearest ambulance will be summoned and the nearest Poisons Information Centre consulted.

Giving water or milk

In some types of poisoning, giving water will help dilute the noxious agent. This applies only to alkalis (corrosives) and to acids, and in practice this means things such as caustic soda, dishwasher powders, toilet (lavatory) cleaners, kerosene (paraffin) and petrol (gasoline). After the ingestion of alkaline corrosives all patients have some burns to the lips, tongue, cheek mucosa and oesophagus. Some 22 per cent have second or third degree burns to the oesophagus and stomach as well. The eyes and face are usually also burnt. Flooding the external skin and eyes with water dilutes the corrosive and allows the cellular and tissue fluid buffers some chance to compensate.

Milk has the added advantage of containing proteins (lactalbumin and casein) which absorb and bind some of the caustic. Never give more than 250 millilitres (ml) of water or milk to a child as too much bulk can induce or exacerbate retching. There is no point, and some danger, in giving fluids to

- any victim lapsing into coma, or
- any victim who has swallowed an unknown substance.

Giving specific antidotes

Antidotes are not commonly used, even in intensive care wards, in the management of poisoned victims. These antidotes are naloxone (for narcotic overdose), ethanol (for methanol and ethylene glycol ingestion), physostigmine (for atropine poisoning), atropine (for organophosphate and mushroom poisoning), digoxin-specific antibodies (for digoxin and oleander poisoning), flumazenil (for benzodiazepine eg Valium poisoning), and the cyanide antidote kit (amyl nitrite, sodium nitrite and sodium thiosulphate) for cyanide poisoning.

All these antidotes are potentially dangerous in their own right, and nice judgement and experience is required in their use. For this reason they have no real

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Plate 13 *Some weeks after the freezing cold injury, a black carapace or eschar forms. With time the line of demarcation between viable tissue and dead, mummified tissue becomes apparent and the eschar will separate and peel off along that line. (Photograph courtesy of R. Burdell, Antarctic Division.)*



Plate 14

Plates 14, 15 and 16 *The aftermath of accidents: nail through finger, toe amputated by lawnmower, body and limb lacerated by barbed wire. Several chapters in this book have relevance to these alarming pictures; see, for example, Wound Infections, Foreign Bodies in Wounds, and Tissue Healing and Scarring. But most relevant of all, perhaps, is Preventive First Aid. These accidents need not have happened.*

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Plate 15



Plate 16

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part to play in pre-hospital care – with one exception. This is the situation in the manufacturing and mining industries and on the farm where cyanides and organophosphates are used. First aiders and paramedics caring for workmen in these specific instances will have access to such antidotes, and there is a requirement that the carers know how the antidotes can be used. The correct drill is to talk, using a mobile telephone, with the intensivist of the nearest hospital, if such antidotes have to be used as a life-saving measure in the field or on the workshop floor.

Although it might seem logical, from the theoretical point of view, to use an alkali antidote to neutralise an acid that has been swallowed, in practice this makes the situation much worse. This must, therefore, never be attempted. Any acid or alkali used in this way will damage tissue before any theoretical neutralisation could occur, and the heat generated by the exothermic chemical reaction which results can be disastrous.

The use of charcoal

"Activated" charcoal is a purified powdery form of charcoal. It is completely inert from the biological point of view. At the ultra-microscopic level, the charcoal consists of tiny spheres and tiny hollow cups. This produces a vast surface area (from about 1000 to 2000 square metres per gram) which has the further property of physically binding drugs and chemicals of all types. Giving a conscious victim a slurry of this great absorbing surface of charcoal to drink means that poisons of all types, which are not yet absorbed from the gut, can be bound and then passed out with the charcoal which itself is not absorbed.

Activated charcoal can be bought in many countries over the counter. If it is available, and if the patient can be induced to ingest this unappealing slurry, it may be used in the pre-hospital situation following telephone consultation with a Poisons Information Centre, or with an emergency medicine department, or a physician. It is mixed up with water in a (non-critical) dose of 1–2 grams per kilogram body weight (eg 100 grams for the average adult). It is very effective in reducing the absorption (up to 70 per cent reduction of absorption of hundreds of different compounds) of ingested poisons. It can have some side-effects such as vomiting, damage to lungs if the charcoal is aspirated, and constipation. Of course it also causes the stools to be jet black and, if the patient – such as a toddler or hallucinating adult – is combative, black charcoal slurry can be spread over a lot of people and a large area. Under such circumstances the tiny granules of charcoal can scratch the cornea of the eye. Activated charcoal should *not* be used in the management of ingestion of acids, alkalis (corrosives), ethanol, ferrous salts, ethylene glycol or button batteries (used in cameras and computer games). The reason for this recommendation is that vomiting is contraindicated following acid and alkali ingestion, and that the other drugs do not bind to the microsurfaces of the charcoal.

If activated charcoal is given in the emergency department it is combined with a laxative to hasten the transit time through the bowel. Using magnesium citrate or sorbitol, such transit time is reduced from an average of 19 hours to 12 hours or less.

Syrup of ipecac

For several decades, good preventive first aid has taught that there should be "a bottle of ipecacuanha syrup in every home". The drill has been that if a child ingests a potential poison, then the first aider (usually a parent) should telephone the local Poisons Information Centre and in some cases administer 15–30 ml of the syrup. It is now strongly advised that the first aider discuss the matter by phone with a Poisons Information Centre before using ipecac or any other means of inducing vomiting.

Ipecac is obtained by an alcoholic extract (ie a tincture) of the root of the ipecacuanha bush. The syrup is sweet. It is rapidly absorbed and the drug acts on the vomiting centre in the brain. This not only causes vomiting but also some reverse peristalsis of the small intestine as well. It has a proven success of greater than 90 per cent in causing vomiting, if repeated. Its use is very old. Syrup of ipecac has, however, a limited use in first aid work. It is effective if given early, immediately after the swallowing of the poison, and now does not have any real use, if charcoal is available, later than an hour or so after ingestion of the poison. It has a place in

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some remote areas, where there may be a delay of more than three or four hours from ingestion to possible admission to an emergency department facility. Of course it must never be used in any poisoning by alkalis or acids, detergents or hydrocarbons such as paraffin (kerosene) or petrol. In these circumstances, vomiting up the low viscosity, low surface tension chemicals allows them to spread over mucous membranes into the trachea and lungs with serious consequences. Ipecac must never be used if the patient is lapsing into coma, as a vomiting patient without a protective gag reflex is in grave danger.

The main contraindication to the use of ipecac is that activated charcoal works better, and that, if the patient is vomiting from ipecac, it is difficult to start giving the charcoal.

The principle underlying the pre-hospital care of the poisoned patient is that central dictum of all health care – *primum non nocere* – first and foremost do no harm. An understanding of the types of poisoning that occur commonly, an adherence to basic DRABC doctrine, and the ability to interact by telephone with the nearest Poisons Information Centre will not only do no harm, but will preserve life.

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CHAPTER TWENTY-THREE

SHOCK

John Pearn

Shock is one of the universals of all trauma and its recognition, management and prevention form one set of universals of all first aid work.

The first aider thinks of shock as the state when there is an insufficient volume of blood to fill the vessels of the circulatory system. Under these circumstances insufficient oxygen-carrying blood can perfuse the tissues of the body. Within the fundamental units of the body – the cells – shock may be defined as respiratory failure at the cellular level.

In the short term – within seconds or minutes – the most vital organs of the body, the brain and heart, may react to this lack of oxygen-delivery by not functioning properly. In the case of the brain, clouding of consciousness or outright unconsciousness may occur. If the blood vessels of the heart are deprived of a sufficient flow of blood, the heart cannot pump as well and a vicious circle of shock/heart-failure/further shock may ensue. In the short term, organs other than the brain or heart can run up an oxygen debt in the shocked patient. This debt of course has to be repaid, and it is the first-aider's role to help in this repayment in the pre-hospital setting.

In some types of shock, if the blood flow to selected vital organs is insufficient and lasts more than just minutes, then permanent damage may result. This affects the kidneys and gut particularly. Kidney failure is not uncommon in shocked children who are dehydrated from fluid loss in gastroenteritis.

In a normal healthy person, a number of automatic servo-controlled reflexes control the output of the heart on the one hand, and the diameter of the blood vessels and the tension in their walls on the other. If whole blood is lost (as in haemorrhage) or if the blood's fluid component is lost (as in dehydration) the blood vessels constrict to make the intra-vascular volume less. Blood is also automatically shunted away from the gut and kidneys and these organs are asked to contribute to the preservation of the brain and heart, by themselves going into temporary oxygen debt. The heart beats faster to compensate also, with the result that there is no "dead-space" in the blood vessels and oxygen-carrying blood is still being pumped to the tissues. Nevertheless, the victim will look pale (owing to the constricted blood vessels in the skin), may feel weak or faint (owing to even a tiny reduction of oxygen to the brain), and will have an altered pulse rate. First aiders regard such individuals also as "shocked", even though the compensatory mechanisms are working overtime. Thus the term "shock" may be broadly defined as "That state of the body when there is insufficient blood flow to the tissues to maintain normal function; or when compensatory reflex mechanisms have to operate to maintain such normal tissue perfusion".

THE CHEMISTRY OF SHOCK

To function normally, every body cell needs oxygen delivered to it. It must then extract this oxygen from the blood bringing it. It uses this oxygen to generate a chemical, adenosine triphosphate (ATP). ATP is the fundamental chemical of life. It is necessary to maintain not only the second-to-second workings of the cells but also their structural integrity.

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Thus, all of life depends on the taking in of oxygen through our lungs, on its transfer to the blood in the lungs, on its circulation by the heart, on its perfusion of all tissues by the capillaries which supply them, on its extraction from the blood and fluids by the cells, on the use of this oxygen to make ATP, and on the role of ATP in being the substance involved in the workings of the cells.

If this wondrous chain of linked events is broken at any point, then the cells cannot function normally. Acid by-products, particularly lactic acid, build up in the cell – often to such a degree that acid spills back into the blood stream. Thus in shock there is oxygen debt, the tissues are forced to rely as long as they can on oxygen-free functioning (called "anaerobic metabolism"), and tissue acidosis occurs.

A more specific definition of shock than that given above, used by all doctors, nurses and paramedics, is: "shock is that condition in which oxygen delivery to the body is inadequate for the generation of sufficient ATP in the cells necessary to maintain their function and structural integrity".

NORMAL REFLEXES – THE SIGNS OF SHOCK

The perfusion of body tissues by oxygen-carrying blood is kept at a correct flow rate by a set of automatic reflexes. These are normally beyond conscious control, though some rare individuals (yogis for example) can modify them by training and some others can do the same following biofeedback exercises.

The heart is the main agent which ensures that the correct amount of oxygen-carrying blood reaches the tissues. It speeds up instantly if extra oxygen is required. It speeds up if any tissues are deprived of oxygen, and thus an increased pulse rate is almost always a sign of shock.

The rate of heart beat is controlled by three essential factors:

1 automatic reflexes,
2 the "slowing" effect of the vagus nerve, and
3 the amount of adrenaline in the circulating blood which it pumps.

It is the cardiac reflexes which produce the classic sign of an increased pulse rate, albeit often a *thready,* rapid pulse – in the patient who is shocked.

In all healthy mammals, the "instant" or quickest responding reflex determining heart rate depends on stretch receptors in the right side of the heart. One sees this in one type of shock, the "faint" that brings down a soldier who has stood too still for too long on parade. Some of the blood that should be returning to his heart is "pooling" in his legs. If there is insufficient venous return to the heart, there is insufficient stretching of these heart-wall receptors and the heart slows. This important reflex is called the "Bainbridge Reflex". As soon as we run or exercise, more blood returns to the heart and the Bainbridge Reflex causes it to speed up.

Similarly, when more blood returns to the right side of the heart, it beats more forcefully. This second reflex, known as "Starling's Law" of the heart, occurs within seconds of increased venous return. Starling's Law and the Bainbridge Reflex operate together as very powerful "real-time" responders to counteract a damming up of blood in venous systems. Under normal conditions of quiet activity, the cardiac output remains reasonably constant, the rate and stroke volume of the heart (ie the pump) being linked together – the so-called "ANREP Effect".

The Bainbridge Reflex and Starling's Law are instant responses, and it is important to understand these normal reflexes in the context of the first aider's recognition and management of shock. However, they can be overridden by a more powerful reflex system brought into play when there is oxygen lack in tissues. The heart responds to chemical sensors in the carotid arteries and in the brain. If there is insufficient flow of oxygen-carrying blood to these sensors, they are triggered, and nerve circuits from them to the heart cause it to speed up. Other sensors respond to the amount of acid in the circulating blood, again causing the heart to beat faster. These two chemically-driven reflexes are the dominant protective reflexes in the whole body. They compensate for detected oxygen lack by causing the blood to circulate faster. They are very powerful and persistent and, in a shocked patient, only fail when death is near.

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Other reflexes determine whether the blood vessels of the body, especially the capillaries, are held wide open or are constricted. The sensors for these reflexes are in the blood vessels themselves and in the brain as well. In addition, other reflexes determine just which sets of capillaries will be held open, thus ensuring maximum perfusion of the oxygen-carrying blood, and which capillaries can be safely shut down for a short period of time – usually minutes.

Under normal conditions of quiet activity, the capillaries in the skin are open, and the capillaries of the absorbing surface of the intestinal wall and of the kidneys are also open. However, if blood is suddenly lost (as in traumatic haemorrhage) or if extra blood is suddenly needed in the brain or muscles (in a sudden fight or fright, for example), the capillaries and other blood vessels in the skin (and for example in the intestine and kidneys) will constrict. The person will look pale, food absorption from the last meal will stop; and urine production will slow down. When the crisis is over, the constrictive reflexes will stop and the tissues will receive their oxygen again. The brain can do without oxygen safely for only eight seconds or so, the heart for perhaps three minutes, but skin will remain viable for up to 36 hours and skin grafts will sometimes "take" from a cadaver donor for as long as 40 hours after the latter's death.

SYNCOPE OR FAINTING

Fainting or syncope is the sudden loss of consciousness that occurs when the brain is inadequately perfused with oxygen-carrying blood for more than eight seconds or so. It is the simplest and most common form of shock.

It occurs particularly when blood "pools" in the veins and distended capillaries of the lower limbs. This may occur when the muscle-pump of the legs is not working and, as mentioned before, is commonly seen when soldiers stand on parade without moving. If one flexes one's calf and thigh muscles (surreptitiously so that the sergeant-major doesn't see) the blood will keep flowing and the Bainbridge Reflex and Starling's Law of the heart will not fail.

Another cause of fainting is a sudden shock, or the experience of a sudden bad smell or a frightful sight of something disgusting. Here the brain sends signals to the blood vessels which cause them to dilate. Blood pools in them and does not return to the heart. Under these unpleasant circumstances also, the brain sends "slowing" signals to the heart via the vagus nerve. The result is a sudden loss of cardiac output and a reduced supply of oxygen-carrying blood to the brain and within eight seconds or so the reserve of oxygen is used up and unconsciousness ensues. This type of shock is called "psychogenic shock".

Psychogenic shock is the cause of medical students fainting in the operating theatre, fathers fainting in maternity wards, and inexperienced first aiders or policemen fainting when they see blood or frightening wounds at accident sights. After the fainter loses consciousness, the conscious mental processes which have triggered the vagus (inhibitory) nerve to the heart are lost; and again when the victim is lying down, having been correctly diagnosed and positioned by the first aider, the normal Bainbridge Reflex and Starling's Law can take over, and the heart speeds up and pumps more effectively, and oxygen is again delivered to the brain.

HAEMORRHAGIC SHOCK

Lack of blood or reduced blood volume can result from trauma, from blood loss internally, from dehydration and, in severe cases, from anaemia. The end result of all these is insufficient perfusion of the body's cells by oxygen-carrying fluid. Shock results. The signs of shock – pallor, clammy skin, rapid thready pulse, confused mind – may come on instantaneously in cases such as severe cuts or lacerations or fractures with bleeding into or from the severed blood vessels. Sometimes the signs of shock come on more gradually, as in the case of a child with gastroenteritis. If such a child has (say) ten bowel motions and three vomits over several hours a very large amount of fluid is lost. Blood volume is lost. There is insufficient blood to perfuse the tissues, which go into oxygen debt. The resulting signs of shock may "creep

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up" on parents or on first aiders. Anticipation of the likelihood of shock is an important part of all pre-hospital management of any casualty.

CARDIOGENIC SHOCK

If the heart itself is damaged then it may not be able to pump sufficient blood around the circulatory system to perfuse all the tissues with enough oxygen-carrying blood. This occurs in a sudden coronary occlusion (a heart attack) or following some infections of the heart muscle (myocarditis). It occurs if the heart itself is damaged by other causes of oxygen lack, such as near-drowning or suffocation. It can occur after a blow, such as the chest hitting the steering wheel of a car in a front-on crash. It can occur following poisoning with some chemicals. If the heart cannot maintain the circulation by its own pumping effect, the condition is called "cardiogenic shock". It is always potentially fatal. The first aider's role is to do all that is possible to ensure as much oxygen-carrying or oxygen-enriched blood as possible still gets to the brain, whilst the patient is en route to the emergency department.

TOXIC SHOCK

In some severe infections, such as septicaemia, the circulating germs produce toxins which act directly on blood vessel walls, preventing them from maintaining tone. They dilate and cannot respond to the normal constrictive protective reflexes. The heart too may be poisoned by such germs and its pumping action may be reduced. The net result is blood pooling in capillaries and tissues instead of circulating vigorously. The tissues become starved of oxygen. The condition is called "toxic shock" and is often fatal. It can come on gradually over several days, or can develop very rapidly, sometimes within hours, in cases such as streptococcal septicaemia resulting from retained tampons.

RESUSCITATION AND SHOCK

A shocked patient looks pale, has a clammy skin, has a rapid pulse, may be breathing quickly, is often confused, and usually has a reduced blood pressure. A trained paramedic, looking for these signs and diagnosing shock, sees past the outward signs and in his or her imagination sees the cells of the body not making enough ATP to function properly. Looking at the pale, shocked patient, the paramedic sees the lactic acid building up in the patient's cells and then spilling out into the blood stream.

The aim of resuscitation is to stop this chain of events. Resuscitation from shock is only complete when all evidence of oxygen debt and of tissue acidosis has disappeared.

First aiders act especially upon the first part of this chain, through their ABC of first aid (airway, breathing, circulation). They do this by ensuring a clear airway, by giving supplemental oxygen, by artificial ventilation, and by external cardiac compression if required. When the shocked casualty is passed on to the casualty or emergency department of a hospital, a "D" and an "E" can be added to the chain of treatment. Drugs can be used to increase the delivery ("D") of oxygen to meet the metabolic demands of all tissues of the body, especially those of the gut and kidneys which have put themselves into oxygen debt to preserve the brain and heart. Other drugs can be used to facilitate the extraction ("E") of oxygen by shocked body cells, still affected by the oxygen debt and still in an acidotic state or worse.

LATER EFFECTS OF SHOCK

If a casualty remains in a shocked state, the oxygen-starved tissues may be permanently damaged. The brain is the organ most vulnerable to such oxygen lack. Sometimes a survivor will be left with a degree of intellectual retardation. Some 3 per cent of childhood survivors of near-drowning accidents, for example, have

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all their other body tissues salvaged by correct first aid and subsequent medical care, but remain in a permanently vegetative state because the oxygen debt and acid damage to brain cells was too great for recovery.

The kidneys are also vulnerable to the effects of oxygen lack, as the very brain-protective reflex which shunts blood from them may have to operate for so long that the oxygen debt becomes too great for repayment. Some children who are shocked for many hours following the dehydration of gastroenteritis may go into kidney failure after they have been successfully resuscitated and after all lost fluid has been replaced. An artificial kidney procedure may be needed for their survival.

All the internal organs of the body are vulnerable to this oxygen debt. The most severe effect is multiple-organ failure, which is commonly seen in intensive care units where patients have been successfully resuscitated in terms of life itself but reperfusion of oxygen-carrying blood has occurred too late to restore function in the intestines, kidneys and liver. This phenomenon of *compensated shock* is seen in many patients who have experienced major surgery and in the great majority (80 per cent) of patients with severe life-threatening infections. Sometimes, after the patient has been resuscitated in the intensive care ward, the gut is so damaged by its oxygen debt that it does not act as a barrier to the microbes normally contained within it. Mucosal injury and its consequences – migration of bacteria across the bowel wall into the blood stream and the release of toxins into the blood stream – is called *reperfusion injury*; this is a potent cause of multiple organ failure in patients who otherwise survive because of the skills of physicians working in intensive care wards.

Every action that the first aider takes to reduce shock in the pre-hospital setting reduces the risk of these downstream complications.

PRE-HOSPITAL MANAGEMENT OF SHOCK

The rationale of first aid management of shock is two-fold:

1. to protect the brain and heart from the effects of underperfusion; and
2. to remove, as far as is possible, the agents causing the shock.

Simply lying the patient down will help the venous return to the right side of the heart, thus allowing both the Bainbridge Reflex and Starling's Law to operate better. Loosening clothing about the neck, chest and waist will stop any restriction of venous return; this is the reason that this rule is such a fundamental one in all first aid.

Removing unpleasant sights or smells or objects that trigger horror or fear will relieve psychogenic shock. Stopping bleeding will arrest the need for the body's extreme reflex response to blood loss. Sips of water in some circumstances will give comfort, and so reduce psychogenic components of the shock syndrome, and in other circumstances will help increase blood volume. Fluids must never be given of course if an anaesthetic will be needed for the primary injury which caused the shock in the first place. Supplemental oxygen will help in many cases. Everything that can be done to increase oxygen levels in the body cells themselves forms the basis of shock management.

Keeping a patient warm is one of the important parts of the overall management of the shocked patient. This not only reduces the psychogenic component of shock, since warmth brings comfort, but also and very importantly will help to stop a victim shivering. Shivering is a dramatic form of muscular action to produce heat and is another protective reflex. But shivering in turn raises the need for more oxygen, and this can be dangerous in a casualty who already does not have enough to perfuse all the tissues. All shocked patients feel cold because the reflexes constricting the blood vessels are shut down and the temperature receptors are not bathed with warm blood. If an already shocked patient uncontrollably starts shivering because he or she feels cold, the shock will become worse rather than better as extra demands are placed on an already compromised system. It is important not to make a

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shocked victim too warm as then the blood vessels will dilate and blood will be pooled in them and the situation will be worse. Thus in keeping a shocked patient warm, it is important to realise that "not too little, not too much" is the rule.

One of the most dramatic causes of shock is internal bleeding. Patients who have been involved in trauma are particularly at risk. If a motor vehicle accident victim, or someone involved in a brawl, becomes gradually weaker and paler, and if their pulse continues to rise (the oxygen-sensor reflexes doing all they can to compensate), it is likely that internal bleeding is continuing, perhaps from a ruptured spleen or liver. Internal bleeding can occur in some types of pregnancy also.

The whole focus on the anticipation, recognition, understanding and treatment of shock has to do with the delivery of oxygen-containing blood to the cells of the body. All patients who develop shock, or who remain shocked for more than a few minutes after any triggering event, should therefore see a doctor. In the interim, the cornerstone set of first aid drills – reassurance, removal of offending cause, lying down, removing constricting clothing, keeping warm, close monitoring of vital signs – will keep them alive.

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CHAPTER TWENTY-FOUR

SNAKEBITE

Julian White

Snakebite strikes fear into the hearts of most humans. In most urban communities snakes are seen as essentially evil or frightening creatures; yet snakebite is not a common occurrence in most urbanised nations, other than in that community of individuals who keep or handle dangerously venomous snakes. Therefore, while often mentioned in textbooks, the subject of snakebite is rarely dealt with in any great detail and is usually regional in outlook, such as the problem of rattlesnake bite in North America. However, snakebite in the context of medical problems world-wide is of considerable significance. In many parts of Africa, Asia and South America snakebite causes significant morbidity and mortality and in a few countries ranks as a major health issue.

This chapter will outline the problem that snakebite presents from an epidemiology viewpoint, and similarly discuss the culprit snakes, their venoms, envenomation and its management. It will become clear to the reader that, while we now know a considerable amount about snakes and their venoms, diagnosis and treatment are still evolving. This is reflected in the diversity of advice available on first aid for snakebite, much of it conflicting and often with little sound scientific basis. The World Health Organization and the International Programme on Chemical Safety have embarked on an extensive review of all forms of poisoning including snakebite. The initial consensus of ideas on first aid has helped form the basis for the recommendations found in this chapter.

EPIDEMIOLOGY

The epidemiology of snakebite may be considered from several aspects. First, there is the national and international perspective of total numbers of people bitten by, or dying as a result of being bitten by, venomous snakes. Secondly, there are similar considerations based on bites by each particular species of snake. Lastly, the issue of bites with envenomation compared to the total number of bites may be considered. The last subject, the issue of "dry bites", is of considerable importance as it is poorly understood by many practitioners in the field of snakebite management and this lack of full understanding may lead to unsound conclusions and recommendations for therapy.

It is important for all involved in snakebite management to realise that a snake bite may not result in envenomation or, if envenomation occurs, it may be only minor, despite the capacity of the snake to deliver a dangerous quantity of venom with a single bite. In practical terms this means that not all bites by any given species of snake will cause significant injury or death. Indeed, for some potentially lethal species, the rate of envenomation is below 50 per cent of cases, and death even in the untreated case may occur in less than 10 per cent of cases. However, when significant envenomation does occur, it may rapidly cause a life-threatening illness. Correct first aid procedures for snakebite will hasten convalescence and save resources in terms of intensive care management.

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In one major international survey of snakebite world-wide it was estimated that the annual death-rate from snakebite was up to 40 000 persons per year, the majority occurring in the Indian subcontinent and in Asia. In another recent survey it was estimated that 23 000 deaths occur annually in west Africa alone. These figures, and others using new diagnostic laboratory techniques, point to the major health problem that snakebite presents in some regions. Indeed in some countries snakebite is a major cause of death; an example is Myanmar (Burma) where it is the 6th to 7th most important cause of death, ahead of heart disease and cancer. In one study in an Indian community in South America, based on residual levels of antibody to snake venom, it was estimated that up to 80 per cent of the adult population had been bitten by a dangerous snake at least once during their lifetime. It is likely that in excess of 1 000 000 people annually may be bitten by snakes.

In "westernised" countries the incidence of both bites and fatalities is clearly less, and a different group of victims enters the statistics, namely those individuals who handle venomous snakes either as a hobby or as part of their work. The hobbyists are a particular problem as they may sustain repeated bites and may account for 25 per cent or more of all snakebite cases and an even higher percentage of serious cases.

The epidemiology of bites for particular species of snakes is beyond the scope of this chapter but such information is available for a variety of species and for some regions (eg Australia – not Australasia, as snakes do not occur in New Zealand) is known in some detail for most of the fauna.

THE SNAKES

Snakes form one suborder (Serpentes) of the Class Reptilia (reptiles), and comprise about 3500 species world-wide of which only about 450 are classed as venomous and a considerable number of these are not dangerous to man. Venomous snakes fall into three broad groups.

1 Back fanged snakes (Family Colubridae).
2 Front fanged snakes with small fangs – the cobra type snakes (Families Elapidae, Hydrophiidae).
3 Front fanged snakes with large mobile fangs – vipers and rattlesnakes (Family Viperidae) – and the side fanged vipers (Family Atractaspidae).

It is the latter two groups which account for the vast majority of snakes dangerous to man. Most regions have representatives of both these groups but Australia has only the Elapidae. This is of significance from a first aid viewpoint as the first aid for Australian type elapid snakebite is substantially different from that advocated for viper bites in other parts of the world. In deciding on appropriate first aid it is, therefore, necessary to know what is the broad likelihood of envenomation and the type of snake which caused the bite. An extensive listing of dangerous venomous snakes of the world, with data on their distribution, venom, and appropriate first aid is given in Table 24.2 at the end of this chapter.

Family Colubridae

Colubrid snakes, in general terms, resemble the "classic" snake in appearance and until recently were considered as non-venomous with a few exceptions. Those exceptions had small fangs placed towards the back of the mouth, a position quite effective for envenoming the snake's natural prey, but less efficient for biting man. It is now understood that considerably more colubrids produce venom inoculated either by fang or simply around the tooth, and the list of species potentially harmful to man is increasing.

Species of note are the tree-dwelling colubrids of southern Africa such as the boomslang (*Dispholidus typus*) and the vine snake (*Thelatornis kirtlandii*) and the Asian species *Rhabdophis subminiatus*, all of which can cause significant and even life-threatening envenomation in man, principally affecting the coagulation system, causing haemorrhage.

Family Elapidae

Elapid or cobra-type snakes all have an effective fang structure (Figure 24.1) and many are dangerous to man, although some smaller species are, in practice, unable to

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bite and envenom a human. The fangs are anteriorly placed in the upper jaw on a small maxilla which, in at least some species, is capable of limited rotation allowing for fang movement during the act of biting. There are numerous other teeth in both the upper and lower jaws and, while smaller than the fangs, these teeth may also leave bite marks or scratch marks. Fang lengths are dependent not just on the size of the snake but also on the species. Some regional groupings of elapid snakes are of particular relevance in human envenomation.

Americas

Coral snakes are found in the southern United States through into South America *(Micrurus, Micruroides)*. They are generally small snakes of thin build and often have vivid coloured bands. Many species are capable of causing significant envenomation in man, the usual result being paralysis, including respiratory paralysis.

Africa

The cobras are widely distributed in Africa and are common in many regions, and all species are generally considered potentially dangerous. In addition to typical cobras *(Naja)* a few of which may spit venom, there are spitting cobras or rinkhals *(Hemachatus)*, the desert black snake *(Walterinnesia)*, the burrowing cobra *(Paranaja)*, the tree cobras *(Pseudohaje)*, the water cobras *(Boulengerina)* and the garter snakes *(Elapsoidea)*. Depending on the species, cobra bites may cause either systemic or local pathology or both. Some species can cause quite severe local tissue damage or necrosis around the bite (or spit) site. Neurotoxic envenomation is the classic systemic effect of cobra envenomation, although it only occurs with bites by some species. Cardiotoxins are described from a few cobra venoms but their role in human envenomation is less clear.

Asia

As Asia is the largest continent, it is not surprising that its elapid fauna are quite diverse and that several genera are dangerous to man. In western Asia, including the Middle East, there are few elapids *(Naja, Walterinnesia, Elapsoidea)*. In the Indian subcontinent, the cobras *(Naja)* (Figure 24.2) are common and can cause both local damage and systemic envenomation, especially neurotoxic paralysis. Also found here are the kraits *(Bungarus)* which cause severe neurotoxic paralysis. Both these groups extend across to east and south-east Asia and the adjacent Pacific (eg the Philippines). There are also some coral snakes *Maticora, Calliophis* in Indo-China and the Malay peninsula, and the king cobra *(Ophiophagus hanna)*.



Figure 24.1 *Skull of an elapid snake (Australian taipan* (Oxyuranus scutellatus)) *showing the fangs (plus old and reserve fangs) and other teeth in the upper jaw. (All photographs in this chapter courtesy of Dr J. White.)*

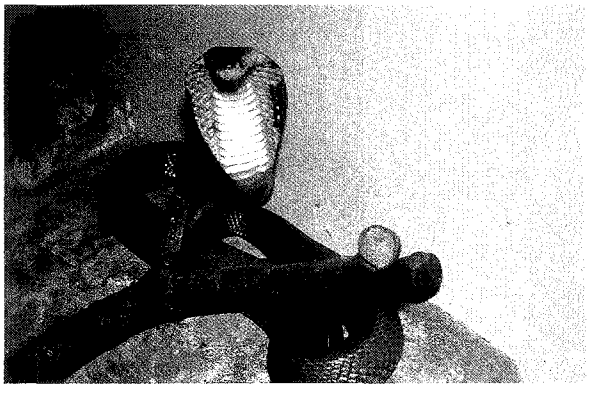


Figure 24.2 *The common or Indian cobra* (Naja naja) *showing the typical flared or hooded neck of the cobras, the most important group of elapid snakes.*

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Australia

Australia has the most diverse and rich elapid fauna of any continent. The majority of such elapids are small and harmless to man but there are about 20 species which are potentially lethal, including many of the world's most venomous snakes. The dangerous species fall into five major groups which correspond to the five types of specific (monovalent) antivenoms available in that country. These groups are:

1 the brown snakes *(Pseudonaja);*
2 the tiger snakes *(Notechis)* (Figure 24.3) including the copperheads *(Austrelaps)* and the rough scaled snake *(Tropidechis);*
3 the black snakes, notably the mulga snake or king brown *(Pseudechis; P. australis);*
4 the death adders *(Acanthophis);* and
5 the taipans *(Oxyuranus)* (Figure 24.4).

In addition, several other species may cause significant envenomation, for example the whip snakes *(Demansia)* and the broad headed snakes *(Hoplocephalus)*. The Australian elapids cause principally systemic envenomation and, with the exception of *Pseudechis*, do not cause significant local tissue injury. The pattern of systemic effects varies with species but may include any or all of neurotoxic paralysis, myolysis, defibrination-type coagulopathy and renal failure.

Europe

Europe does not have a native elapid fauna.

Family Hydrophiidae

The seasnakes are taxonomically closest to the cobras (elapids) and the fang structures are similar. There is some similarity between seasnake venoms and those of elapids, in particular the Australian elapids, which some taxonomists have suggested are closer to the seasnakes than to other elapids. Seasnakes are found in a wide band in the Indian and Pacific Oceans and associated seas extending from the east coast of Africa to the west coast of the Americas, and from the equator polewards a little beyond the tropics. Only one species is truly pelagic *(Pelamis platurus)*, the rest being mostly found in coastal and reef regions. There are several genera *(Laticauda, Enhydrina, Aipysurus, Astrotia, Hydrophis, Microcephalophis, Lapemis)* but not all appear to be dangerous to man. Those that do cause significant envenomation generally produce neurotoxins or myotoxins.

Family Atractaspidae

A unique group of snakes of medical importance is found in Africa and the Middle East. Genus *Atractaspis* occupies a taxonomic position which is controversial. These strange snakes are the side fanged or mole vipers.

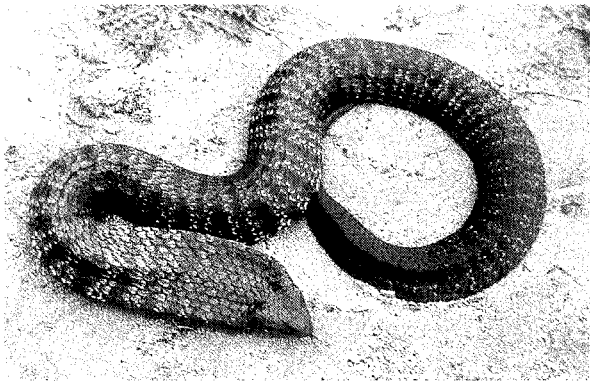


Figure 24.3 *Tiger snakes* (Notechis *spp.) are found in southern Australia and show more neck flare than other Australian elapids.*

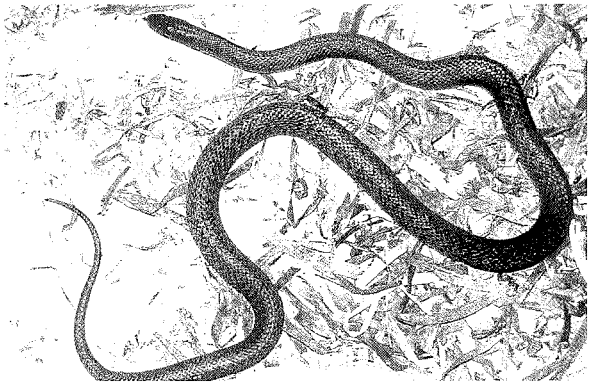


Figure 24.4 *The Australian inland taipan* (Oxyuranus microlepidotus) *has the most potent snake venom known, but bites are very rare.*

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They bite in an unusual fashion and often do so with the mouth closed yet effectively ejecting venom from only one fang. The venom of these snakes contains a unique class of cardiotoxins (sarafotoxins).

Family Viperidae

The vipers (Figure 24.5) are a diverse and widely distributed family of snakes represented on all continents except Australia (and adjacent New Guinea) and Antarctica. Vipers have a highly evolved skull with a mobile maxilla capable of rotation through nearly 90°. This allows the fangs to be much longer than in elapids, and in some vipers the fangs may exceed 20 mm in length. Most species, however, have fangs well under 20 mm and some of the smaller species have quite modest fang lengths. The large fang is usually teamed with large venom glands capable of producing from 100 to 400 mg (dry weight) of venom or more. Thus, in theory, vipers can inject deeply a large dose of venom, but in practice most viper bites are only subcutaneous. As with elapids the viper may bite and leave clear fang marks yet fail to inject a significant quantity of venom. Venom toxicity and effects are quite variable but the classic hallmark of viper envenoming is local tissue damage including oedema and even necrosis. Many vipers may also cause severe systemic effects as well, including coagulopathy and haemorrhage, renal damage, cardiotoxicity, myolysis, and even paralysis in a few species.

The typical viper body shape consists of a broad and large triangular-shaped head, quite distinct from the narrower neck, the latter quickly expanding into a large body, comparatively shorter but thicker than that of an elapid or colubrid snake. The pupil is usually slit-shaped consistent with the partially or wholly nocturnal or crepuscular activity patterns of most vipers. The vipers are divided into two distinct subfamilies.

Subfamily Viperinae

These are the classic vipers of the Old World. They lack heat-sensing pits.

Subfamily Crotalinae

These are the "pit vipers", so named because they have heat-sensing pits on their upper jaws that allow them to detect warm-blooded prey at night (Figure 24.7). This sophisticated prey detection system is used by only one other group of snakes, the non-venomous pythons, in which the heat-sensing apparatus is less highly evolved. Not surprisingly most pit vipers are principally nocturnal, though not necessarily exclusively so.

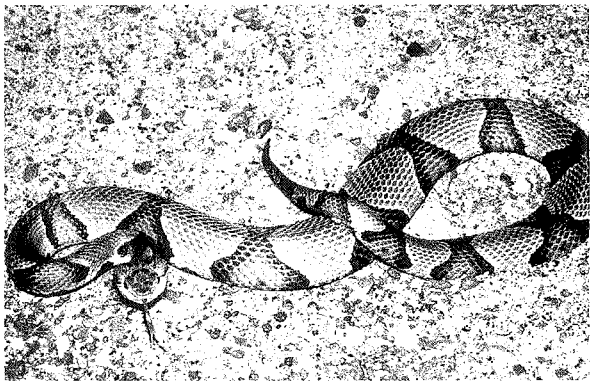


Figure 24.5 *Vipers are typically more robust in build than elapids and often have a more distinct triangular head, as seen in the North American copperhead* (Agkistrodon contortrix).

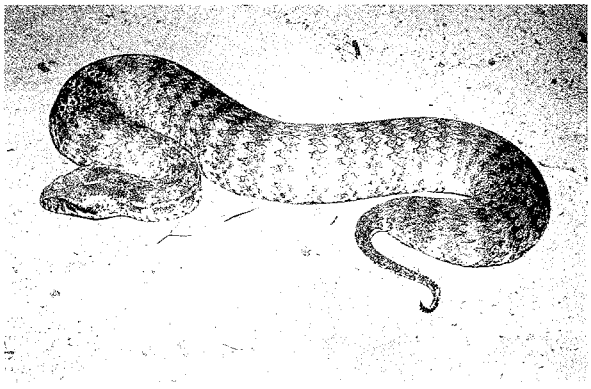


Figure 24.6 *The Australian death adders,* Acanthophis *spp., are elapids with viperid appearance and habits.*

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Americas

Vipers dominate the venomous snake fauna of South, Central and North America. All species are pit vipers. In North America the rattlesnakes (Genus *Crotalus)* predominate, and the diamondback rattlesnakes *(C. adamanteus, C. atrox)* probably account for most serious bites to man. The two other viper groups of significance in North America are the copperheads or moccasins *(Agkistrodon)*, and the pigmy rattlesnakes and massasauga *(Sistrurus)*. Most of the North American vipers have strongly cytolytic venoms causing moderate to marked local tissue swelling and damage at the bite site, but a few have quite different venoms causing only systemic effects, notably neurotoxic paralysis (especially the mohave rattlesnake, *Crotalus scutellatus*). Clinically this is an important differentiation to understand as the mohave rattlesnake is sympatric with the western diamondback rattlesnake (*C. atrox*) (Figure 24.8) and the two species appear superficially similar. In the same area may be found a coral snake *(Micruroides)* which may also cause neurotoxic paralysis, but which requires a quite different antivenom to that for viper bite.

In Central America there are several species of rattlesnake with varied local and systemic effects, and here also some representatives of the principal South American pit viper fauna may be found, of the Genus *Bothrops.*

Through into South America the bothropid vipers predominate (Genera *Bothrops, Bothriechis, Bothriopsis, Lachesis, Ophryacus, Porthidium*) many of which can inflict serious bites to humans, usually characterised by both local tissue injury and systemic effects, principally coagulopathy with haemorrhage, renal damage and for some species at least, myolysis. True neurotoxic paralysis, however, is not a significant feature of envenomation by these snakes, but it is the predominant feature of bites by the most important South American rattlesnake, *C. durissus terrificus.*

Africa

Vipers are widely represented in Africa, all being classic vipers (Viperinae). In terms of human bites the carpet viper *(Echis carinatus)* (Figure 24.9) is the most significant species, causing thousands of deaths each

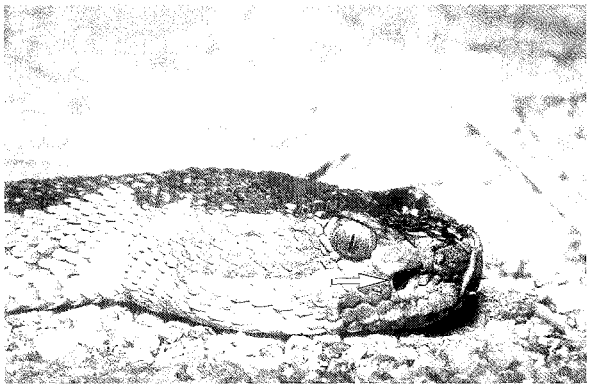


Figure 24.7 *Head of a rattlesnake, showing the heat-sensing pit just in front of and below the eye. This gives the snake effective "heat vision" to guide strikes on warm-blooded prey in the dark.*

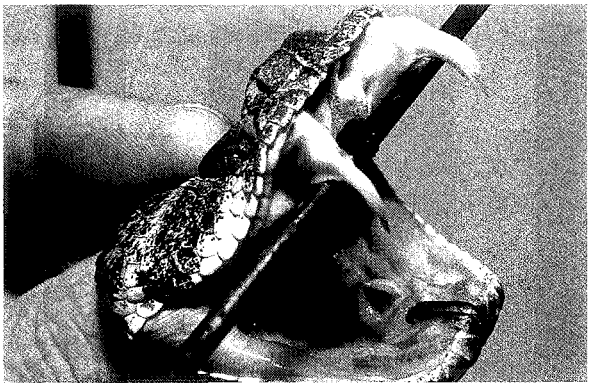


Figure 24.8 *Live western diamondback rattlesnake, showing the long fangs in erect position, as they would be when striking a human.*

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year, followed by the puff adder *(Bitis arietans)*. The gaboon viper *(B. gabonica)* is worthy of mention because of its size and lethal reputation. These species range from southern through central Africa, while the carpet viper is found across northern Africa and is of special medical significance in savanna west Africa. In the arid regions of northern Africa may be found the horned adders *(Cerastes)*. Other genera represented in Africa include *Causus, Vipera, Adenorhinos* and *Atheris*, although the snakes of the latter two genera are of doubtful medical significance.

Asia

Western and central Asia are the province of the true vipers (Viperinae) including several species responsible for many significant human snakebites, such as the carpet and saw scaled vipers *(Echis)*, Russells viper *(Vipera russelli)*, and related species (*V. ammodytes, V. palestinae* (Figure 24.10), etc.). In western Asia and the Middle East there are some lesser known vipers *(Pseudocerastes, Eristicophis)*. In eastern and south-east Asia there are also pit vipers including the Malayan pit viper *(Calloselasma rhodostoma)* noted for the marked defibrination type coagulopathy that its bite may cause; the green tree vipers *(Trimeresurus)* (Figure 24.11) which may also cause coagulopathy, though generally less severe; the habu *(T. flavoviridis)* of northern Pacific islands (Okinawa etc.) which may cause significant local tissue injury, and the Asian copperheads, notably the mamushi *(Agkistrodon blomhoffii)*. However, in its range in south-east Asia, Russells viper is generally responsible for many or most of the severe snakebites, particularly in Myanmar (Burma), where it is responsible for more than 95 per cent of all snakebite deaths.

Europe

The only venomous snakes of significance in Europe are the typical vipers *(Vipera)* and these generally cause either minor envenomation or principally local tissue damage, but the latter can cause significant fluid shifts and shock.

Australia

There are no vipers native to Australia or New Guinea.

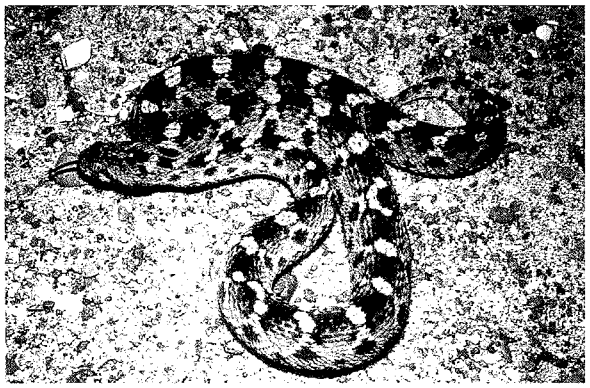


Figure 24.9 *The carpet viper* (Echis carinatus) *is responsible for more snakebite deaths world-wide than any other species of snake.*

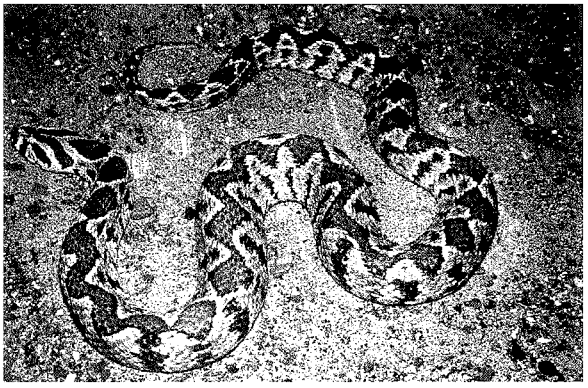


Figure 24.10 *A typical viper* (Vipera palestinae) *from the Middle East.*

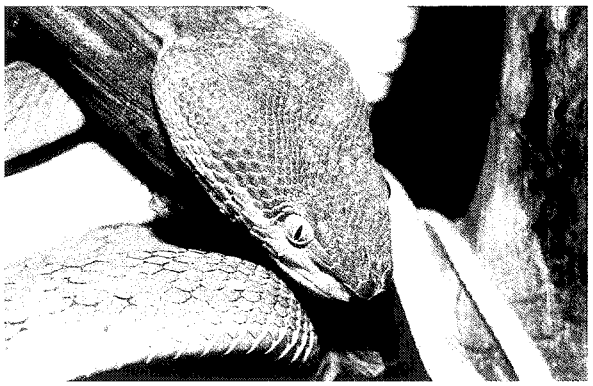


Figure 24.11 *The green tree pit vipers* (Trimeresurus *spp.*) *are an important cause of snakebite throughout Asia.*

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VENOMS

Snake venoms are essentially a modified digestive juice produced by glands evolved from salivary glands. Venom has probably evolved for two principal functions, namely, the rapid immobilisation of prey and the digestion of prey. It is a complex mixture of components some of which have multiple effects and many of which may work synergistically. While most of the major components are proteins, they may be glycoproteins, metaloproteins etc., and may range in size from less than 10 000 daltons to 200 000 daltons or larger. Each species of snake produces a distinctive venom and both within species and even for individual snakes there may be quite significant differences in venom depending on geographic location, or time of year, or age of the snake.

Venom is a precious resource for the snake and this may in part explain why some snakes fail to inject a significant dose of venom when inflicting a defensive bite against a human. At least for some species it has been shown experimentally (and many times clinically) that the snake can bite without injecting venom. The mechanism of expulsion of venom by compression of the gland by surrounding muscle contraction does leave some scope for voluntary control. The likelihood of any given snake biting and failing to inject a significant quantity of venom is dependent on several factors including the rapidity and angle of the strike, whether the bite is through clothing, the number of bites (multiple bites are nearly always associated with significant envenomation), when the snake last fed and, not least, the species and size of the snake.

There are many schemes of classification of venom components and toxicity, and this may lead to conflicting information in the literature. One classification is in terms of toxicity, the common measure is the Lethal Dose 50 per cent (LD_{50}) which is the dose of substance (venom) which, on average, will kill 50 per cent of the test animal for the particular lethality experiment. This is in many ways a crude assessment and is subject to important variables, namely, the species of test animal (some species are far more susceptible to a particular venom than others), the route of administration, the quality of the venom, and venom variability within each species of snake. Because of these variables, it is quite difficult to compare all the world's venomous snakes and assign to each a ranking in terms of their venom toxicity. Attempts to do so in the past have produced conflicting results.

One recent study compared species of note from around the world with particular reference to Australian species and concluded that Australia had most of the world's most venomous snakes. However, this gives a false impression of the medical importance of each species of snake which is based as much on local factors, such as population–snake interactions and availability and standards of health care, as it is on the toxicity and quantity of venom the snake may inject. From this perspective, the most important snakes medically are those from Africa, Asia and South America. The carpet vipers *(Echis)*, Russells viper *(Vipera russelli)*, the bothropids *(Bothrops)* and the cobras *(Naja)* are responsible for most of the snakebite deaths throughout the world. Although Australia has the most venomous snakes, deaths from Australian snakebite pale almost into insignificance when compared with the tens of thousands annually which occur in Africa.

Venom being a mixture of components, some of which may have multiple actions or be synergistic in action with each other, any scheme of classification will be artificial and incomplete. From a medical viewpoint it is often helpful to assess components on the basis of their clinical effects in envenoming man. Using such an approach it is possible to categorise venom components into two broad groups of actions: local and systemic.

- *Local venom actions* Components with local action are essentially cytolytic, causing local oedema, haemorrhage, tissue damage and necrosis, and are generally referred to as cytolysins. Such components are found in the venoms of some elapids (notably cobras) and many vipers.
- *Systemic venom actions* The range of clinically important systemic venom actions is extensive. Contrary to comments frequently found in the literature, it is not possible to categorise the vipers and elapids purely on the basis of clinical systemic

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effects; thus not all vipers are haemorrhagic and not all elapids are neurotoxic.

Neurotoxins

These are of three main types:

1 postsynaptic action at the neuromuscular synapse typified by alpha bungarotoxin and generally working by blocking binding of the neurotransmitter acetylcholine to its receptor on the muscle end plate;
2 presynaptic action at the neuromuscular junction typified by beta bungarotoxin and typically having a phospholipase A_2 component in the molecule and causing interference with release of acetylcholine from the terminal axon by a variety of methods, most often by acting on the ion channels of the terminal axon, such as potassium channel blockers; and
3 synaptic neurotoxins affecting transmission in the autonomic system typified by kappa bungarotoxin and of uncertain clinical significance at present.

There are several subgroups of postsynaptic neurotoxins, including the long chain and short chain neurotoxins, but their importance medically is their ability to cause often rapid and severe neuromuscular paralysis. However, because of their site of action, they are generally accessible to therapy including antivenom. This distinguishes them from the presynaptic neurotoxins which cause a more severe paralysis, often of slower onset but not reversible by antivenom therapy. Most snakes which have presynaptic neurotoxins in their venom also have postsynaptic neurotoxins but the converse is not true.

Most snakes with neurotoxic venoms are elapids but a few vipers also have potent neurotoxins.

Coagulopathins

This artificial term encompasses the wide variety of venom component actions on the haemostasis system. The two major actions are:

1 coagulopathy caused by reduction in the ability of the clotting system to produce blood clots; and
2 coagulopathy caused by interference with platelet function.

To these must be added those venoms that directly cause haemorrhage by their action on blood vessel walls, the haemorrhagins. The combination of a coagulopathin and a haemorrhagin can be devastating clinically, but coagulopathins alone may cause marked derangement of normal haemostasis without major deleterious consequences for most patients.

The mechanisms of action of these venom components are varied and some venoms contain a multitude of such components, each of which may affect the clotting system in a different way. In general terms, however, the effects on the clotting system usually result in a decreased ability of the blood to clot. This may be achieved by true anticoagulants which inhibit or block key steps in the formation of blood clots, or it may be the result of consumption of key clotting proteins as a direct or indirect effect of venom action. The common consequence of venoms in this latter group is to remove most or virtually all circulating fibrinogen (the protein which blood clots are actually made from). This process is commonly called "defibrination" and is distinct from the true complete consumption coagulopathy seen in response to many medical problems and known as disseminated intravascular coagulation (DIC), since in defibrination there is no consumption of platelets. In some venoms the consumption of fibrinogen is direct, with the venom splitting chains (fibrinopeptides A or B) off the fibrinogen molecule and opening the way for natural fibrinolysis of the resultant products. In other venoms prothrombin is converted to active thrombin and this activates a natural chain of events resulting in the destruction of fibrinogen. In either case the results are clinically the same, the ultimate effect being in essence an "anticoagulation". Any breach in vascular integrity will result in intractable bleeding, as no clot can be formed to seal the breach and even the initial platelet plug may be abnormal, since fibrinogen is an essential element in the binding of platelets to form the platelet plug.

The action of venoms directly on platelets is less well understood clinically, though for a few venoms it is well elucidated at the molecular level. Again venom actions are dichotomous as they may cause either inhibition of platelet function or promote platelet aggregation.

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Myolysins

The myolysins cause destruction of muscle. This action is usually systemic rather than local and largely or wholly confined to voluntary muscle. Experimentally, there is breakdown of the internal structure of individual muscle cells and two released products are of importance clinically, namely myoglobin and creatine phosphokinase (CK), both of which are readily measured. The muscle damage may be very extensive and cause wasting and other symptoms but clinically there is complete recovery over time, though this may take months. Experimentally, there is evidence that only the slow fibres recover and that the consequent loss of fast fibres is equivalent to premature ageing of muscle. This alarming finding has not yet been shown in human cases. The component causing myolysis is a modified phospholipase A_2 molecule.

Myolysins are present in several elapid venoms, particularly some Australian species, as well as a few viper venoms *(Vipera russelli, Bothrops* spp.*)*.

Cardiotoxins

These are essentially membrane toxins. They cause disruption of cardiac muscle function. The clinical significance is uncertain in most species where such toxins have been demonstrated (eg some cobras, gaboon viper, Australian brown snake).

Nephrotoxins

Kidney damage or failure is a serious complication of snakebite and, while in some cases this may be secondary to other venom induced damage, some venoms contain primary nephrotoxins. The distinction between the two groups is not clear at present and some species of both vipers and elapids cause renal damage.

ENVENOMATION

Envenomation is that clinical state which occurs when venom is circulating in the victim or when venom has been inoculated into the victim. It is the important but not inevitable consequence of a bite by a dangerous venomous snake. It encompasses all the diverse effects of venom on the human body by the equally diverse array of snakes. It may range from trivial or minor envenomation through to life-threatening envenomation.

The prevention or delay in onset of envenomation is the prime goal of first aid for snakebite. The second objective for the first aider managing a snakebite is to be aware of the manifestations of envenomation and watch for them, documenting them so that when the patient reaches a definitive treatment facility the treating practitioner will have the most accurate assessment possible on which to base treatment strategies.

When a snake bites and injects venom a mass of venom is usually deposited subcutaneously and will then move gradually proximally to reach the systemic circulation, from which it will be distributed throughout the body. Some or most of the venom on passing through the renal circulation will be filtered by the kidneys and may be excreted in the urine. The method of venom movement from the bite site is variable and incompletely understood. For elapid snakes (and probably all snakes) some or most of the venom is initially transported via the lymphatic system. Thus any activity by the victim will increase the rate of flow of the venom by increasing the muscle pump action of the limb muscles. Some venom may directly enter the blood stream at the bite site by simple diffusion. However, since venom molecules are of large molecular weight, it is unlikely this will apply to all components unless there is an alteration in the local vascular architecture as a result of venom damage. Some venoms, notably those causing local tissue damage, contain spreading agents such as hyaluronidase that open tissue planes and speed local dissemination of the venom. Some venom components may cause release of endogenous substances from the tissue around the bite site and these may then rapidly enter the blood stream and cause widespread effects. This mechanism is incompletely understood but may account for the rapid onset of symptoms in some patients.

The onset of envenomation may be very rapid or delayed by many hours, particularly if effective first aid has been instituted. In some cases where a bite has

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occurred, envenomation may never develop as the snake has failed to inject a significant amount of venom. This latter event, though not uncommon, will not be predictable at the time of the bite and all bites must be treated as potentially lethal (Figure 24.12). Envenomation will be more rapid in onset if one or more of the following pertain:

- the patient is of small body mass such as a child;
- if no effective first aid has been instituted in the first 3–15 minutes after the bite;
- if the patient has been physically active immediately following the bite; and
- if there have been multiple bites. Some species, such as the taipan and Russells viper, are more likely than others to inject a lethal or supralethal dose of venom.

The early symptoms of envenomation will depend on the species of snake. Some symptoms are non-specific and some are those of anxiety. These symptoms include headache, nausea, vomiting, abdominal pain, dizziness and non-specific visual disturbance. Collapse or convulsions may occur and are usually a manifestation of significant envenomation. For some snakes (eg Australian elapids), the first apparent manifestation of envenomation in young children may be collapse followed by generalised convulsions.

In those snakes which cause significant local tissue damage early symptoms may also include local pain and swelling and even bruising and blistering. In severe cases the fluid shift may be rapid and result in development of shock with its own attendant symptoms.

As envenomation progresses, symptoms and signs of specific venom action may become apparent. Depending on the species of snake, there may be evidence of neurotoxins, myotoxins, haemotoxins, nephrotoxins, or cardiotoxins. Even for bites by a given species of venomous snake, the likelihood of these complications and their severity and timing of onset are quite variable. Thus there may be an ineffective bite with no envenomation of significance, or at the other extreme first aiders may be confronted by a victim of multiple bites with rapid profound envenomation. Even within a single species of snake there may be variability in both venom content and toxicity. Small children and patients who have failed to institute appropriate first aid or who have been physically active after the bite and those who have received multiple bites are most likely to have rapid onset and progression of envenomation.

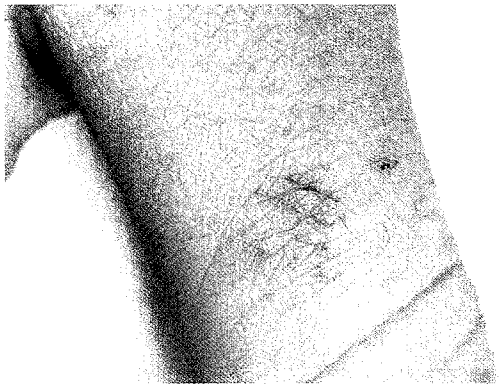


Figure 24.12 *An Australian brown snake bite, showing faint scratch marks rather than fang marks, with no local reaction or pain. This bite looks trivial but certainly was not, as this child had life-threatening envenomation within 30 minutes of the bite!*

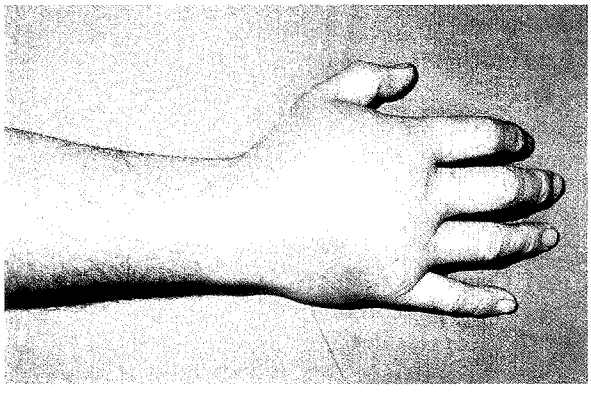


Figure 24.13 *The Australian mulga snake causes swelling of the bitten limb but, in most cases, no tissue damage.*

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However, while death is a potential outcome it is unlikely to occur very rapidly. In a study in Australia nearly a century ago – when neither antivenom nor intensive care facilities and ventilatory support were available - of those patients who died from snakebite only 15 per cent did so in under 6 hours after the bite. Nearly 50 per cent died 24 hours or more post bite.

Neurotoxic complications

Neurotoxins are probably the best known cause of snakebite mortality, but in a modern medical environment, where patients can be maintained on a respirator for prolonged periods, death from neurotoxic paralysis should be a rarity. Snake venom neurotoxins generally work at the neuromuscular junction by blocking transmission of the signals for muscle to contract. This is usually only at the skeletal muscle neuromuscular junction and so principally affects voluntary and respiratory muscles. The extent of paralysis is variable but may be complete and, without external respiratory support, this will prove lethal. The presynaptic neurotoxins cause a more severe and longer lasting paralysis than the postsynaptic neurotoxins and may not be reversed by antivenom therapy, but the rapidity of onset of symptoms is usually slower than with postsynaptic neurotoxins. For both types, the earliest symptoms and signs will not usually be evident for at least one hour post bite and are often delayed by several hours.

The earliest manifestations of developing paralysis are seen most often in the muscles of the face, particularly eye muscles (Figure 24.14). The patient may complain of sleepy or drooping upper eyelids and on testing be unable to fully raise the upper eyelid (ptosis). There may be blurred or double vision, the latter due to paralysis of some but not all ocular muscles. If the patient is asked to look to the left or right or upwards, this may stimulate double vision (diplopia). Eventually, however, all the eye muscles may become paralysed and the patient can only stare fixedly forwards. At this stage the ptosis may be complete and the pupils may be fixed dilated. This latter finding is important to note as fixed dilated pupils in a severely paralysed and possibly unconscious snakebite patient may purely reflect the effect of venom directly on the pupillary muscles and may not in any way reflect underlying devastating intracranial pathology. As paralysis progresses there will usually be progressive slurring of speech, peripheral and



Figure 24.14 *The earliest signs of neurotoxic paralysis are seen in the face, particularly drooping of the upper eyelids (ptosis), as in this case of Australian tiger snake bite.*

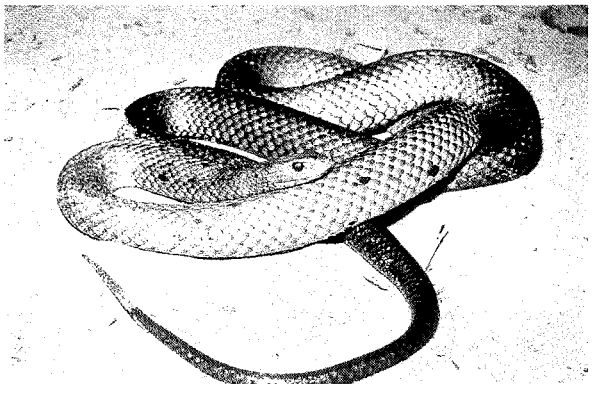


Figure 24.15 *The brown snakes are the most frequent cause of both snakebites and snakebite-related deaths in Australia.*

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then generalised muscle weakness. Accessory muscles of respiration come into play and finally only the diaphragm will be working and the patient will be in obvious respiratory distress. Eventually, even the diaphragm may be paralysed.

Myotoxic complications

Destruction of skeletal muscle (myolysis, rhabdomyolysis) has not always been recognised as a complication of envenomation, but in recent years a wide variety of snake venoms have been shown experimentally to cause direct muscle damage. An increasing range of snakes are now known to cause myolysis in man and in many cases this is a major and potentially lethal complication. While the onset of symptoms and signs of myolysis may be delayed several hours, it is likely that the venom-induced muscle damage commences much earlier, as soon as the venom reaches the circulation and is distributed throughout the body. Often the first indication that myolysis is occurring is a change in urine colour to red or even brown due to excretion of muscle pigment (myoglobin) in the urine. This may also damage the kidneys causing kidney failure. By this time there will be detectable rises in enzymes in the blood (eg creatine kinase). The first and often only symptoms are pain on moving muscles and muscle weakness. Thus myolysis may mimic paralysis.

Haematological complications

The effects of snake venom on the blood are many and varied but, even when quite severe changes have occurred, they may not be reflected by obvious symptomatology. Thus the blood may be rendered completely unclottable but, unless the patient has a bleeding lesion, there may be no external indication of this potentially lethal complication. Fortunately, if there is such a severe coagulopathy, there is often at least a snakebite wound from which there will be a constant ooze of non-clotting blood. Any venepunctures or insertions of intravenous lines will also usually ooze blood, an important point to remember in performing such procedures on snakebite patients. Some species of snakes may cause much more obvious bleeding or bruising, often because the venom also contains a haemorrhagin which damages blood vessels. This may result in bleeding from the gums, superficial bruising of the skin (which can be very extensive and associated with major blood loss), or evidence of internal bleeding such as coughing up blood (haemoptysis), vomiting blood (haematemesis) and passing blood in the urine (haematuria), the latter easily confused with myoglobinuria due to muscle damage, and bloody bowel motions may occur. Internal haemorrhage secondary to snake venom-induced coagulopathy is usually a serious and potentially lethal complication, the common problem being haemorrhage into or around the brain. This in turn may result in collapse, unconsciousness, convulsions, a flaccid paralysis mimicking neurotoxic paralysis and pupillary changes. A few snakes, notably Russells viper *(Vipera russelli),* can also cause haemorrhagic infarction of selected organs, particularly the pituitary gland resulting in Sheehan's syndrome. Most effects of venom on the blood are based on relatively small amounts of venom reaching the circulation at any given time. If there is a sudden bolus dose of venom, as might occur if the fangs entered a vein (fortunately quite rare), then the effects are quite different. In this situation there may be sudden catastrophic massive clotting of blood which will rapidly cause cardiovascular collapse, cardiac arrest, and inevitable death within a few minutes of the bite.

Nephrotoxic complications

Kidney damage is still one of the more common causes of snakebite mortality world-wide, reflecting both the potential of many snakes to induce kidney damage and the lack of resources to treat kidney failure in many snakebite-prone parts of the world. The damage to the kidneys may be a primary effect of specific nephrotoxins in the venom, or the secondary effect of other venom-induced abnormalities such as shock, coagulopathy, haemorrhagic infarction and myolysis. Immunologic mediated damage may also play a role. For some snake species, such as Russells viper, kidney failure is the usual cause of fatal outcome in human victims.

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The extent of kidney damage is variable, from mild glomerular or tubular damage, through acute tubular necrosis, and occasionally even renal cortical necrosis. The latter may require either long-term dialysis or renal transplant for survival, but for most of the other more common and lesser degrees of kidney damage there will be a partial or total recovery of kidney function if the patient can be supported through a few days to weeks of acute renal failure. The time of onset of kidney damage is both variable and incompletely understood, but in many cases probably occurs quite early after onset of systemic envenomation. However, symptoms and signs may not be apparent for some hours and so both the patient and their care givers, including first aiders, may be quite unaware of the damage occurring internally.

Cardiotoxic complications

The role of cardiotoxins in humans envenomed by snakes is not well understood, reflecting uncertainty in our understanding of the nature and mechanism of action of snake venom cardiotoxins. They are best known from cobra venoms (*Naja* spp.) and may play a role in collapse seen after envenomation by these snakes. They appear to have a similar role in envenomation by the gaboon viper *(Bitis gabonica)* and are probably responsible for cardiac arrhythmias and even cardiac arrest which may occur after bites by this species. It is uncertain how many other venoms may contain similar components.

Cytolytic complications

A significant consequence of bites by many diverse species of venomous snakes is tissue injury at the bite site. This may vary from mild swelling through to major tissue damage including necrosis, long-term damage, and even loss of the bitten limb. In venoms which result in such damage a wide variety of components may individually or synergistically produce the tissue injury. Such components are found in most snake venoms but are of clinical significance in many of the viper venoms and a few of the elapid venoms, particularly some of the cobras (*Naja* spp.). Local tissue swelling usually commences soon after the bite and may be accompanied by bleb formation, local subcutaneous haemorrhage, and even swelling or necrosis of underlying structures including muscle. A compartment compression syndrome may develop which if untreated may cause ischaemic necrosis. With all these local changes pain is a common feature and may be severe. If there is compression of vital structures, this may cause further symptoms such as numbness distal to nerve compression. The local tissue swelling may be accompanied by a significant fluid shift to the injured tissues similar to that seen with major burns, and can result in hypovolaemic shock which is potentially lethal. This latter effect is commonly seen following severe bites by some viper species such as the diamondback rattlesnakes of North America, but should be considered following any snakebite where there is major local swelling or tissue damage.

DIAGNOSIS

The signs of snakebite may range from the very obvious to the very obscure, and it is likely that a number of unexplained deaths are due to snakebite. At one end of the spectrum, the reptile fancier who presents bitten by his pet snake would seem a fairly clear-cut case. Yet even here there may be doubt, first as to whether they have been bitten, secondly as to the true identity of the snake (not all amateur herpetologists are knowledgeable taxonomists), and thirdly as to symptomatology. They may exhibit symptoms such as nausea, even vomiting, headache, abdominal pain, and local pain, and may even feel short of breath or sweaty, or conceivably collapse, all due to anxiety and hyperventilation rather than true envenomation. All the above symptoms could equally be due to advancing envenomation. If these problems may occur in an apparently straightforward case of snakebite, they are multiplied many-fold in people bitten in rural areas while going about their business, and the situation is often even more complex in children from whom no useful history may be obtained. In a young child, the first an adult may be aware that anything is amiss is a crying child suddenly

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collapsing and convulsing. Alternatively, the patient, either child or adult, may present unconscious with no available history.

The diagnosis of snakebite is, therefore, sometimes complex and may rely on both a high index of suspicion by the treating health-care workers and a knowledge of the possible effects of envenomation. There are two crucial decisions that must be made, namely, is the patient definitely or possibly suffering from a snakebite, and is there evidence of significant envenomation.

For the first aider the first of these questions is often the most important as it will determine if the patient needs appropriate first aid for snakebite. As the aim of such first aid is to minimise morbidity and mortality, the quicker it is instituted the better. Therefore, if there is a reasonable chance that the patient may have been bitten by a dangerous venomous snake the working diagnosis should be snakebite and appropriate first aid instituted immediately (see Table 24.1).

The role of the first aider can be more extensive than simple application of a bandage or splint. They can make a real contribution to the process of diagnosis, and therefore treatment, by noting both the circumstances of the bite and the subsequent development of symptomatology.

TREATMENT

The treatment of snakebite has been controversial throughout history, with innumerable folk remedies and almost as numerous "reputable" medical treatments, some of which have proved to be far from reputable. First aid for snakebite has been equally contentious and remains so. Different methods are recommended in different parts of the world.

First aid principles

Any first aid must adhere to the maxim "do the patient no harm". Unfortunately, many types of first aid for snakebite – some of them still actively advocated in some regions – contravene this basic rule. The best known of these are the practice of cutting and sucking the wound and the use of tourniquets. The latter has been advocated to slow movement of venom from the bitten limb, but is usually incorrectly applied and left on too long. It has resulted in many cases of severe limb injury, sometimes requiring amputation for gangrene. An arterial tourniquet can only be left on safely for about 20–30 minutes and, when briefly removed after this time, may be associated with rapid onset of envenomation symptoms which may be harder to control than if no first aid had been used. Cutting and sucking the wound may actually accelerate the absorption of venom and so make envenomation more rapid and severe. There is some limited evidence that specifically designed medical devices which produce local cuts and then apply high level suction, may be effective in some types of viper bite (those causing major local effects), but their role remains uncertain and they are not recommended at this time.

First aid recommendations

The current guidelines on first aid for snakebite as agreed by the World Health Organization Working Group on Natural Toxins (IPCSD-INTOX Project) are as follows.

1 Elapid snakes

For some elapid snakes (eg Australia, possibly some elsewhere) use pressure immobilisation bandage and splint. This method applies for bites by species which do not cause local tissue injury or significant oedema. It has not been proven safe for species (eg some cobras, vipers) which cause significant local tissue injury.

Method Apply a broad compressive bandage (eg crepe or similar), bound firmly over the bite site, then bind over as much of the bitten limb as possible, over the top of clothing (Figure 24.16). Pressure should approximate that used for a sprained ankle. It should not act as an arterial tourniquet. Peripheral pulses should still be detectable. Severe pain in the bandaged limb may indicate that it is too tight. The bitten limb should be securely immobilised using a splint. Immobilisation of the limb is as important as the compressive bandage.

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Once correctly applied, the compressive bandage should only be removed by medical staff at a treatment facility, as removal may result in rapid development of systemic envenoming.

2 Wound care

Wounds should be cleaned to remove venom on the skin.

Important exception In Australia the wound must *not* be washed. Current venom detection tests (Figure 24.17) used in Australia rely on swabs from the skin adjacent to the bite. There is no evidence that leaving venom from Australian snakes on the skin causes the patient harm.

3 Contraindicated first aid

Many harmful "first aid" treatments are advocated for snakebite throughout the world.

In particular *do not*:

- Excise the wound.
- Incise the wound.
- Apply suction to the wound.
- Use a tourniquet or constricting band.
- Use electric shock therapy.
- Apply or inject chemicals or medicines to the wound.
- Apply ice packs.
- Use any proprietary "snake bite kits".

4 Patient reassurance

Panic is a common and a detrimental response to snakebite. The patient should be reassured that not all bites result in envenoming, even when the snake is dangerous, that many snakes are not dangerous to man, and that modern hospital treatment is effective.

5 Patient immobility

Movement or exercise, especially of the bitten limb, speeds up spread of the venom. Patients should be discouraged, as far as possible, from using the bitten limb; immobilisation is beneficial.

6 Food and drink

In general do not give the patient anything by mouth, including food, alcohol, drugs (especially aspirin or sedatives) or drinks. However, where there is likely to be a considerable delay in receiving medical treatment, consider giving water only by mouth to avoid dehydration.



Figure 24.16 *Pressure immobilisation bandage, before application of the splint. Suitable first aid for Australian elapid bites.*

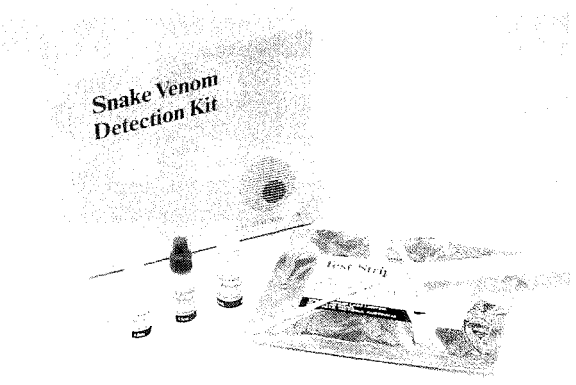


Figure 24.17 *In Australia, the diversity of dangerous snakes stimulated the development of kits to detect venom and more accurately target antivenom therapy. This is the current CSL kit. These techniques are being adapted for other countries.*

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Table 24.1 Key diagnostic information for snakebite

History	
Geographic location where bite occurred.	Delineates likely snake fauna.
Description of the snake.	May assist in identifying snake.
Description of the bite: chewing? glancing? multiple?	Multiple bites are usually associated with significant envenomation.
Patients activity after the bite.	Physical activity speeds venom absorption.
Symptoms of envenomation Local: pain, numbness, swelling. Pain in draining lymph nodes.	
General: headache, nausea, vomiting, abdominal pain, collapse, convulsions.	Indicates venom spread. Some of these symptoms may also reflect anxiety.
Neurotoxic: ptosis, diplopia, dysarthria, muscle weakness, respiratory difficulty.	Indicates developing paralysis.
Myotoxic: muscle weakness, muscle movement pain, red or brown urine.	Indicates myolysis.
Haematologic: oozing of blood from bite site, haemoptysis, haematemesis, melaena, haematuria, bleeding gums.	Indicates coagulopathy.
Nephrotoxic: decreased urine output, nausea, vomiting.	Indicates kidney damage.
Cardiotoxic: racing or irregular heart, faintness, collapse.	May indicate cardiac dysfunction.
Past history Past exposure to antivenom, allergic problems, kidney disease, other major diseases.	Past exposure to antivenom may increase the chance of untoward reaction to antivenom, as may a tendency to allergic disease.
Examination	
Local Inspect the bite site for evidence of fang marks, multiple bites, local swelling, bruising, haemorrhage.	IMPORTANT NOTE: Do not delay application of first aid to examine bite site and do not remove first aid to inspect bite site unless prepared and able to definitively treat envenomation.
Draining lymph nodes: local swelling or tenderness.	If positive, indicates venom spread.
General Vital signs: pulse, blood pressure, respiratory rate, temperature.	If local swelling, look for tachycardia and hypotension as indicators or shock. If unconscious or fitting, look for bradycardia and hypertension as signs of rising intracranial pressure.

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Table 24.1 (Continued)

Neurotoxic: drooping of upper eyelids or inability to open eyelids fully (ptosis), double vision, commonly seen when asked to look to side (diplopia), slurring of speech (dysarthria), peripheral muscle weakness such as poor hand grip or inability to raise one leg normally, respiratory weakness, respiratory failure (eg shallow respirations, cyanosis).	All indicate paralysis developing, the range of such signs indicative of the extent of paralysis.
Haemotoxic signs: These may be few, such as persistent bleeding from the bite site, but also look for bleeding gums, haematuria etc. Severe internal bleeding may produce further signs such as shock or signs related to intracranial bleeding.	Indicative of significant coagulopathy.
Myotoxic signs: initially there may be none; look for red or brown urine, muscle weakness or pain on movement. Ultimately may cause muscle wasting.	Indicative of significant myolysis.
Nephrotoxic signs: Initially there may be none; look for decreased urine output.	Indicative of kidney failure.
Cardiotoxic signs: Look for irregular pulse.	Arrythmias.
Unconsciousness: Is there flaccid paralysis or increased muscle tone, are the pupils fixed dilated, is there fitting, is there an associated hypotension?	Unconsciousness is a moderately common early event in systemic envenomation and may be associated with hypotension of brief duration. Fixed dilated pupils do not necessarily indicate brain problems (see paralysis).
Investigations While not strictly part of first aid they will be mentioned for completeness.	
Basic Whole blood clotting time. Urine check for blood (myoglobin).	Usually can be done without need of laboratory. Evidence of coagulopathy if prolonged. Evidence of coagulopathy or myolysis.
Haematology CBP, clotting studies (INR, APTT, TCT, fibrinogen, FDP, platelets)	Evidence for coagulopathy.
Biochemistry Electrolytes, renal function, CK.	Evidence for myolysis, kidney damage, dehydration etc.
Venom detection Depending on technique may be wound swab, urine, or blood. NOTE: Do not wash the wound if there is a chance of venom detection (ie all bites in Australia).	Snake venom may be detected using either commercial kits (Australia only) or locally developed assays (selected centres in a few countries).

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7 Identification of the snake

Where possible, try to identify the cause of the bite, except where this puts the patient or others at risk. Note descriptive features. If the snake is dead, bring it to hospital with the patient, but handle with extreme care, remembering that even a severed snake's head can bite and inject venom.

8 Medical advice

Arrangements should be made to seek medical advice at the earliest opportunity, and in most cases the patient should be transferred as quickly and passively as possible to a place where medical attention can be given. Patients can be transferred on a stretcher or trestle, on the crossbar of a bicycle, or in a boat, if road transport or conventional ambulances are not available.

9 Life supportive treatment

Bearing in mind that patients may vomit or faint, they should be laid on their side and the airway kept clear. Life supportive treatment, such as expired air ventilation and external cardiac massage, should be used if the patient's condition warrants it. If more than one first aider is present, the most experienced first aider should stay with the victim whilst others get help.

The above guidelines were produced with all countries in mind and, therefore, are quite general, except for the specific mention of Australia, where the management of snakebite is relatively advanced and the specific first aid measures apply, and have been shown to be effective both experimentally and in clinical practice.

The Australian pressure immobilisation method of first aid was developed by researchers in Melbourne in the late 1970s headed by Dr Sutherland of the Commonwealth Serum Laboratories. It was based on past observations that much venom appeared to travel from the bite site initially in the lymphatic system, as evidenced by high concentrations of venom in lymph nodes draining the bitten area, and parallel observations that immobilising the bitten limb in a splint significantly delayed the onset of envenomation. This was combined with knowledge of local lymph flow characteristics and tested as a pressure bandage and splinting in monkeys given doses of venom and then carefully monitored, including measurement of circulating levels of venom using very sensitive assays. The combination of local pressure bandage at the same pressure as for a sprain, and the immobilisation of the limb with a splint proved the most effective safe and reliable method. Given that lymph vessels are small and easily compressed by local pressure, and that movement centrally of lymph is mediated by local muscle contraction so that immobilising muscles dramatically slows lymph flow, it is easy to understand why such a first aid technique is effective. Because blood flow to vital structures in the limb is not critically impaired this method is safer than the tourniquet. Furthermore, as it does not require periodic release, unlike a correctly managed tourniquet, there is no danger of sudden worsening of envenoming due to release, prior to treatment at a hospital. Indeed the researchers were initially suggesting that this method be used for all snakebites throughout the world. However, this is not advocated as such bandaging of the limb may cause worsening of local tissue damage if the bite is by a type of snake causing significant tissue damage, such as many vipers and some cobras. For those non-Australian snakes (principally elapids) which do not cause significant local effects, but rather systemic effects, especially neurotoxic paralysis, the pressure immobilisation technique should be applicable.

Hospital treatment

Clearly the aim of first aid is to present the patient to a hospital or other appropriate medical treatment facility in as good a condition as possible, and at that point hand over responsibility to the medical officer. Most snakebite victims receive definitive diagnosis and treatment at hospital and the mainstay of treatment, world-wide, is antivenom. If there is significant envenomation, then antivenom therapy is virtually always the single most effective therapeutic measure and can be credited with dramatically improving the chance of survival from snakebite. However, other therapies also have a key role to play, especially modern methods of ensuring continued respiratory function up to and including mechanical ventilation. The management of

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renal failure may be important. But the crucial factor in reducing morbidity and mortality from snakebite will be the speed and effectiveness of first aid, as the earlier the patient is presented for treatment and the fewer complications of envenomation that have occurred, the better the final outcome will be.

PREVENTION

The best form of treatment of snakebite is to prevent it happening in the first place. Snakes are an important and integral part of our environment. To advocate their wholesale removal as a measure to prevent snakebite would be both impractical and ecologically dangerous. However, a policy of removing and relocating snakes found in metropolitan areas may have some merit if resources are available and the process is carefully supervised. This method is being tried in parts of Australia but so far there are no results proving it has reduced the incidence of snakebite, though it has certainly relieved the anxiety of many householders! It is probably more relevant to educate the population about snakes, particularly how to avoid being bitten, and what to do if bitten.



Figure 24.18 *Carelessness accounts for many snakebites. Wear appropriate footwear, look where you are putting your feet and, above all, avoid treading on a snake!*

Some basic preventive measures are universal. These include:

- look where you are walking or putting your hands or feet;
- if you encounter a snake endeavour to leave it alone and allow it to escape;
- avoid travel by foot in areas or at times when seeing a snake may be difficult, such as in long grass and at night or around dusk;
- prevent easy access by snakes into houses or sleeping areas or areas which people use as thoroughfares; and
- ensure all adults and older children know what snakes live in the local area, what their habits are, how to recognise them, and what action to take if someone is bitten including having appropriate first aid equipment readily available.

Nevertheless, it may prove very difficult to prevent some snakebites as circumstances conspire to bring human and snake into close contact. An example is the rice paddy fields in parts of south-east Asia where snakes, such as the Russells viper, may become common at rice harvest time as they feed on rodents which also increase at this time. Farmers need to be in the fields in conditions where the snakes may prove hard to see. In these circumstances control seems problematic. Most snakebites, however, will be prevented if people do not attempt to catch and kill these important creatures of the world's ecosystems.

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Table 24.2 Consolidated information on taxonomy, distribution, lethality, venom effects, first aid and antivenom, for all dangerous snakes by Family, Genus, and for some by Species

Snake	Scientific name	Distribution	Lethality	Venom effects	First aid
Family COLUBRIDAE					
Brown tree snake	*Boiga irregularis*	Australia, NG, Guam	Low (?Mod in infants)	L, ?N	BI
Boomslang	*Dispholidus typus*	SubSaharan Africa	Mod to Sev	C, H, R	BI
Argentine blackheaded snake	*Elapomorphus bilineatus*	South America	Mod	H, C	BI
Montpelier snake	*Malpolon monspessulanus*	Mediterranean, ME	Mild to Mod	L, ?N	BI
Rednecked keelback	*Rhabdophis subminiatus*	SE Asia, Japan, China	Mod	L, C, H, R	BI
Yamakagashi	*Rhabdophis tigrinus*	Japan	Mod	L, C, H, R	BI
Bird snakes	*Thelatornis* spp.	Southern Africa	Mod to Sev	H, C, R	BI
Culebra de cola corta	*Tachymenis peruviana*	South America	Mild to Mod	L, H, C	BI
Family ELAPIDAE					
Death adders	*Acanthophis* spp.	Australia, NG, Indo	Sev	N	BI
African coral snakes	*Aspidelaps* spp.	Southern Africa	Mild, occ deaths	N	BI
Australian copperheads	*Austrelaps* spp.	Australia	Sev	N, W	BI
Water cobras	*Boulengerina* spp.	Central Africa	?Mild	?N	BI
Kraits	*Bungarus* spp.	East Asia	Sev	N	BI
	Bungarus caeruleus		Sev	N	BI
	Bungarus ceylonicus	Sri Lanka	Sev	N	BI
	Bungarus fasciatus		Sev	N	BI
	Bungarus multicinctus		Sev	N	BI
Asian coral snakes	*Calliophis* spp.	East Asia	Mild	?	BI
Australian whip snakes	*Demansia* spp.	Australia, NG	Mild	?N	BI
Mambas	*Dendroaspis* spp.	Southern Africa	Mod to Sev	N	BI
	Dendroaspis angusticeps		Mod to Sev	N	BI
	Dendroaspis jamesoni	Mod to Sev	N	BI	
	Dendroaspis polylepis		Sev	N	BI
	Dendroaspis viridis		Mod	N	BI
African garter snakes	*Elapsoidea* spp.	Southern Africa	Mild	L	I
Rinkhals spitting cobra	*Hemachatus haemachatus*	Southern Africa	Mod	L, ?n, spits	BI
Broad headed snakes	*Hoplocephalus* spp.	Australia	Mild	?N	BI
Asian coral snakes	*Maticora* spp.	East Asia	Mild, ?fatalities	?N	BI
Small eyed snake	*Micropechis ikaheka*	New Guinea	Mod to Sev	?N	BI
American coral snake	*Micruroides euryxanthus*	SAm, CAm	Mod, occ. deaths	N	BI
American coral snakes	*Micrurus* spp.	CAm, SAm	Mod, occ deaths	N	BI
Chinese cobra	*Naja atra (N. n. atra)*	SE China	Mod	N	BI
Egyptian cobra	*Naja haje*	Africa, ME	Mod to Sev	N	BI
Monocled cobra	*Naja kaouthia*	SE Asia	Sev	L, N	BI
Forest cobra	*Naja melanoleuca*	Central to Sth Africa	Mod to Sev	N	I

KEY

SNAKE = Family and an accepted common name (other common names may also exist).

SCIENTIFIC NAME = Generally accepted scientific name (other names may also occur in the literature).

DISTRIBUTION = Generally accepted distribution, by name of country, or by region or continent: E = Europe; Af = Africa; NAf = northern Africa; SAf = southern Africa; ME = Middle East; NAm = North America; CAm = Central America; SAm = South America; Aust = Australia; NG = New Guinea; Ind = Indonesia (or part thereof); SEA = south-east Asia; SWA = south-west Asia; Ind = Indian subcontinent (includes Sri Lanka); Ch = China; Jap = Japan; Tai = Taiwan.

LETHALITY = Generally accepted severity of confirmed bite in humans: Sev = severe (often potentially lethal); Mod = moderate (bites often cause significant envenoming, sometimes potentially lethal); Mild = mild (most bites cause only minor envenoming, generally not likely to be lethal); ? = clinical information on severity either lacking or conflicting.

VENOM EFFECTS = Generally accepted clinical effects of venom in man, where known: ? = considerable uncertainty or lack of information on effect(s); A = cardiotoxic; C = coagulopathy; E = haemolysis; G = local tissue necrosis; H = haemorrhagic; L = local tissue swelling/blisters/haemorrhage/damage; M = myolysis; N = neurotoxic paralysis; P = antiplatelet actions; R = renal damage/failure; S = shock; W = anticoagulant.

FIRST AID = Generally accepted method recommended for first aid: I = immobilisation; BI = pressure bandage and immobilisation (Australian method for snakes which do not cause significant local tissue damage).

Note For all information in this table please note that clinical and venom data represent a synthesis of published information available to the author. For many species listed, there is little such information available. Individual medical practitioners may have extensive but unpublished experience of bites by various species of snakes which may not agree with the information given here. Should this be the case, we cordially invite these colleagues to write to St John Ambulance detailing this new information.

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Table 24.2 (Continued)

Snake	Scientific name	Distribution	Lethality	Venom effects	First aid
Mozambique spitting cobra	*Naja mossambica*	Southern Africa	Mod	L, G, spits	I
Indian cobra	*Naja naja (N. n. naja)*	India, adjacent areas	Sev	N, ?A	BI
Black necked spitting cobra	*Naja nigricolis*	Africa	Sev	L, G, H, ?A	I
Cape cobra	*Naja nivea*	Southern Africa	Sev	N	BI
West Asian cobra	*Naja oxiana (N. n. oxiana)*	SE Asia	Mod to Sev	L, G, occN, ?A	
Philippines cobra	*Naja philippinensis (N. n. p.)*	Philippines	Sev	L, N	BI
Malayan spitting cobra	*Naja sputatrix (N. n. sputatrix)*	SE Asia	Mod to Sev	L, G, occN, ?A, spits	I
Sumatran cobra	*Naja sumatrana (N. n. s.)*	SE Asia	Mod to Sev	L, G, occN, ?A	
Australian tiger snakes	*Notechis* spp.	Australia	Sev	N, C, M, N, occL	BI
King cobra	*Ophiophagus hanna*	SE Asia	Sev	L, N, ?A	I, ?BI
Taipans	*Oxyuranus* spp.	Australia, NG	Sev	N, C, M, R, H in NG	BI
Burrowing cobra	*Paranaja multifasciata*	Africa	?	?	BI
Mulga snake	*Pseudechis australis*	Australia, NG	Sev	M, R, ?W, L	BI
Spotted mulga snake	*Pseudechis butleri*	Australia	?Sev	?(M, R, ?W, L)	
Colletts snake	*Pseudechis colletti*	Australia	Mild to Mod	M, L	BI
Spotted black snake	*Pseudechis guttatus*	Australia	Mild to Mod	M, L	BI
Papuan black snake	*Pseudechis papuanus*	New Guinea	Mod to Sev	C, ?H	BI
Red bellied black snake	*Pseudechis porphyriacus*	Australia	Mild to Mod	L, E	BI
Tree cobras	*Pseudohaje* spp.	Africa	?	?	BI
Australian brown snakes	*Pseudonaja* spp.	Australia, New Guinea	Sev	C, R, N	BI
Rough scaled snake	*Tropidechis carinatus*	Australia	Mod to Sev	N, C, M, R	BI
Desert black snake	*Walterinnesia aegyptia*	ME, NAf	Mod	N	BI
Family HYDROPHIIDAE					
Subfamily HYDROPHIINAE					
Horned seasnake	*Acalyptophis perenii*	Indo-Pacific Ocean	Mild	?M	BI
Seasnakes	*Aipysurus* spp.	Indo-Pacific Ocean	Mod	?M	BI
Olive seasnake	*Aipysurus laevis*	Indo-Pacific Ocean	Mod	N	BI
Stokes seasnake	*Astrotia stokesii*	Indo-Pacific Ocean	Sev	N	BI
Turtle headed seasnakes	*Emydocephalus annulatus*	Indo-Pacific Ocean	Mild	?M	BI
Beaked seasnake	*Enhydrina schistosa*	Indo-Pacific Ocean	Sev	M	BI
Mangrove seasnake	*Ephalophis greyi*	Indo-Pacific Ocean	Mild	?M	BI
Black ringed mangrove seasnake	*Hydrelaps darwiniensis*	Indo-Pacific Ocean	Mild	?M	BI
Banded seasnakes	*Hydrophis* spp.	Indo-Pacific Ocean	Mild	?M	BI
Spine bellied seasnake	*Lapemis hardwickii*	Indo-Pacific Ocean	Mild	?M	BI
Northern mangrove seasnake	*Parahydrophis mertoni*	Indo-Pacific Ocean	Mild	?M	BI
Yellow bellied seasnake	*Pelamis platurus*	Indo-Pacific Ocean	Mod	N	BI
Subfamily LATICAUDINAE					
Sea kraits	*Laticauda* spp.	Indo-Pacific Ocean	Mod	N	BI
Family ATRACTASPIDAE					
Mole vipers	*Atractaspis* spp.	Africa, ME	Mod to Sev	L, A, ?A	I
Family VIPERIDAE					
Subfamily VIPERINAE					
Bush vipers	*Atheris* spp.	Africa	Mild	?	BI
Puff adder	*Bitis arietans*	Africa	Sev	L, G, H, R, S	I
Mountain adder	*Bitis atropos*	Africa	Mild	N	BI
Horned adder	*Bitis caudalis*	Africa	Mild	L, G	I
Many horned adder	*Bitis cornuta*	Africa	Mild	L, G	I
Gaboon viper	*Bitis gabonica*	Africa	Sev	L, G, H, C, R, S, A	I
	Bitis heraldica	Africa	?Mild	?L	I

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Table 24.2 (Continued)

Snake	Scientific name	Distribution	Lethality	Venom effects	First aid
Rhinoceros viper	*Bitis nasicornis*	Africa	Sev	L, G, H, R, S	I
Peringueys viper	*Bitis peringueyi*	Africa	Mild	L	I
Dwarf adder	*Bitis schneideri*	Africa	Mild	L	I
	Bitis worthingtoni	Africa	?Mild	N	I
Desert mountain adder	*Bitis xeropaga*	Africa	?Mild	?L	I
Night adders	*Causus* spp.	Africa	Mild	L, ?N	I
Horned vipers	*Cerastes* spp.	N Africa	Mod	L,C	I
Carpet vipers	*Echis* spp.	Africa, ME, Ind	Sev	L, G, C, H, R, S	I
Horned vipers	*Pseudocerastes* spp.	ME, WAf	Mod to Sev	?N	BI
Long nosed viper	*Vipera ammodytes*	SE Europe, WAf	Mod to Sev	L, N	BI
Asp	*Vipera aspis*	Europe	Mod	L, H, S	I
European viper	*Vipera berus*	Europe	Mod	L, H, S	I
	Vipera cerastes	?WAf, ME	?	?	I
Caucasus viper	*Vipera kasnakovi*	Russia	?Mild	?L, H, S	I
Latastes viper	*Vipera latasti*	Iberia, NAf	Mod	L, H, S	I
	Vipera latifi	ME	Mod	L, H, S	I
Blunt nosed viper	*Vipera lebetina*	ME, WAf	Mod	L, H, S	I
	Vipera meridionalis	Europe, ME	Mod to Sev	L, H, S, ?N	I
	Vipera mesocoronis	Europe	Mild to Mod	L, S, H	I
Palestine viper	*Vipera palaestinae*	ME	Mod	L, H, S	I
Russells viper	*Vipera russelli*	Ind to SE Asia	Sev	L, G, H, C, M, R, S	I
	Vipera seoanai	Spain	?Mild to Mod	L, S, H	I
Meadow viper	*Vipera ursinii*	Europe, WAf	Mild	?L, H, S	I
	Vipera xanthina	Europe, WAf	MIld	L, H, S	I
Subfamily CROTALINAE					
Cantil	*Agkistrodon bilineatus*	CAm	Mild to Mod	L, S, G, H	I
Mamushi	*Agkistrodon blomhoffii*	Russia, China, Japan	Mild to Mod	L, S, G, H	I
Dusky mamushi	*Agkistrodon caliginosus*	Korea	Mild to Mod	L, S, G, H	I
American copperhead	*Agkistrodon contortrix*	NAm	Mod	L, S, G, H, C	I
Karaganda pit viper	*Agkistrodon halys*	Russia, China	Mild to Mod	L, S	I
Himalayan pit viper	*Agkistrodon himalayanus*	Himalayas	?Mod	?L, S	I
Central Asian pit viper	*Agkistrodon intermedius*	Russia, China	?Mod	?L, S	I
Likiang pit viper	*Agkistrodon monticola*	China	?Mod	?L, S	I
Cottonmouth	*Agkistrodon piscivorus*	NAm	Mild to Mod	L, S, G, H	I
Tibetan pit viper	*Agkistrodon stauchi*	Tibet, China	?Mod	?L, S	I
Palm pit vipers	*Bothriechis* spp.	CAm	Mild	L, G	I
Eyelash palm pit viper	*Bothriechis schlegelii*	CAm	Mod	L, G	I
Forest pit vipers	*Bothriopsis* spp.	CAm, SAm	Mild to Mod	L, G	I
Chocoan pit viper	*Bothriopsis punctata*	SAm	Mod to Sev	L, G, ?(C, H M, R)	I
Lanceheads	*Bothrops* spp.	CAm, SAm	Mod to Sev	L, G, C, H M, P, R	I
Urutu	*Bothrops alternatus*	SAm	Mod	L, G	I
Patagonian lancehead	*Bothrops ammodytoides*	SAm	Mod	L, G	I
Terciopelo	*Bothrops asper*	SAm	Sev	L, G	I
Common lancehead	*Bothrops atrox*	SAm	Sev	L, G	I
Brazils lancehead	*Bothrops brazili*	SAm	Mod	L, G	I
Saint Lucia lancehead	*Bothrops caribbaeus*	SAm	Sev	L, G	I
Cotiara	*Bothrops cotiara*	SAm	?Mod	?L, G	I
Golden lancehead	*Bothrops insularis*	SAm	Sev	L, G	I
Jararaca	*Bothrops jararaca*	SAm	Sev	L, G, C, H, M, R	I
Jararacussu	*Bothrops jararacussu*	SAm	Sev	L, G, C , H, M, R	I
Fer de lance	*Bothrops lanceolatus*	Martinique	Sev	L, G	I

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Table 24.2 (Continued)

Snake	Scientific name	Distribution	Lethality	Venom effects	First aid
Brazilian lancehead	*Bothrops moojeni*	SAm	Sev	L, G	I
Neuwieds lancehead	*Bothrops neuwiedi*	SAm	Sev	L, G	I
Desert lancehead	*Bothrops pictus*	SAm	Sev	L, G	I
Malayan pit viper	*Calloselasma rhodostoma*	SE Asia	Mod to Sev	L, G, H, C, R, S	I
Rattlesnakes	*Crotalus* spp.	NAm, CAm, SAm	Mild to Sev	L, G, C, H, E, P, M, N, R	I
Western diamondback RS	*Crotalus atrox*	NAm, CAm	Sev	L, G, S, C	I
Eastern diamondback RS	*Crotalus adamanteus*	NAm	Sev	L, G, S	I
Mexican westcoast RS	*Crotalus basiliscus*	CAm	Sev	L, G, S	I
Sidewinder	*Crotalus cerastes*	NAm, CAm	Mod	L, G	I
Cascabel, neotropical RS	*Crotalus durissus durissus*	CAm, SAm	Mod	L, G, S	I
Cascabel, neotropical RS	*Crotalus d. terrificus*	SAm	Sev	N, M, R, H, S, C	BI
Timber RS	*Crotalus horridus*	NAm	Sev	L, G, S	I
Rock RS	*Crotalus lepidus*	NAm	?Mod	?L	I
Speckled RS	*Crotalus mitchellii*	CAm, NAm	Mild	L, G	I
Black tailed RS	*Crotalus molossus*	NAm, CAm	Mod	?L, G	I
Mexican lance headed RS	*Crotalus polystictus*	CAm	Mod	L, G, S, C	I
Twin spotted RS	*Crotalus pricei*	NAm	?Mod	?L	I
Red diamondback RS	*Crotalus ruber*	NAm, CAm	Mild	L, G	I
Mojave RS	*Crotalus scutellatus*	NAm, CAm	Sev	depends on specimen some L, G, S, C, H etc. some N (usually mild)	I
Tiger RS	*Crotalus tigris*	NAm	Mod	L, S, G	I
Mexican dusky RS	*Crotalus triseriatus*	CAm	Mod	L, G, S	I
Western or prairie RS	*Crotalus viridis*	NAm			I
Ridge nosed RS	*Crotalus willardi*	NAm, CAm	Mild		I
Hundred pace viper	*Deinagkistrodon acutus*	China, Taiwan			I
Hump nosed vipers	*Hypnale* spp.	Sri Landa, India	Mild to Mod	L, S	I
Bushmaster	*Lachesis muta*	CAm, SAm	Sev	L, G, S	I
Mexican horned pit viper	*Ophryacus undulatus*	CAm	?Mod	?L, S	I
Hognosed and montane pit vipers	*Porthidium* spp.	CAm	Mild to Mod	L, G	I
Godmans montane pit viper	*Porthidium godmani*	CAm	Mild	L, G	I
Rainforest hognosed pit viper	*Porthidium nasutum*	SAm, CAm	Usually Mild, occasionally Sev	L, G	I
Jumping pit viper	*Porthidium nummifer*	CAm	Mild	L, G	I
Slender hognosed pit viper	*Porthidium ophryomegas*	CAm	Mild	L, G	I
Massasauga	*Sistrurus catenatus*				
Pigmy rattlesnake	*Sistrurus miliarius*				
Tree pit vipers	*Trimeresurus* spp.	East Asia	Mild to Sev	L, S, H, C	I
White lipped tree viper	*Trimeresurus albolabris*	India, China, SE Asia	Mild to Mod	L, S, H	I
Okinawa habu	*Trimeresurus flavoviridis*	Amami and Okinawa islands	Mild to Mod	L, S, H, G	I
Indian green tree viper	*Trimeresurus gramineus*	India	Mild to Mod	L, S, H	I
Chinese habu	*Trimeresurus mucrosquamatus*	Taiwan, Sth China, Indochina, Myanmar	Mild to Mod	L, S, H, G	I
Popes pit viper	*Trimeresurus popeorum*	SE Asia	Mild to Mod	L, S, H	I
Shore pit viper	*Trimeresurus purpureomaculatus*	Bengal to SE Asia	Mild to Mod	L, S, H	I
Chinese mountain pit viper	*Trimeresurus monticuola*	Nepal, China to Malaysia	Mild to Mod	L, S, H	I
Chinese green tree viper	*Trimeresurus stejnegeri*	Taiwan, China	Mild to Mod	L, S, H	I
Waglers pit viper	*Trimeresurus wagleri*	SE Asia	Mild to Mod	L, S, H	I

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CHAPTER TWENTY-FIVE

MARINE ENVENOMATION

John Williamson

The sea and its shores are beautiful places. Therein, however, may lie work for the first aider and paramedic. In the sea and among its reefs is a world of great biodiversity – a world of fish and mammals, of crustacea and medusae, of molluscs and of corals, all of which defend themselves vigorously against the intrusion of humankind.

In the Indo-Pacific waters is found the world's most venomous creature, the box jellyfish *Chironex,* whose Latin name means "the assassin's hand". In the Atlantic and the Mediterranean are to be found venomous jellyfish, and everywhere are to be encountered poisonous fish of great beauty and seasnakes of great elegance.

Experienced first aiders and paramedics can play an important role, not only in treating the victims of marine envenomations and intoxications but also in raising the general awareness of risks and thereby reducing the incidence of death (Table 25.1) and morbidity.

By convention and common usage, "envenomation" implies the introduction of a biological poison, a toxin, via penetration of the intact skin or mucous membranes. "Poisoning" is a more general term. "Intoxication" has come to mean that poisoned state of the body which results from the ingestion of a chemical substance such as alcohol or a toxin such as ciguatoxin.

Morbidity from coral lacerations, prostration from jellyfish stings and poisoning from eating tetrodotoxic and ciguatoxic fish are important subjects for all health professionals – first aiders, paramedics, nurses and doctors alike.

It is clear that morbidity is very common indeed, and constitutes one of the major public health problems in several world regions (eg the Pacific Island nations). It has been significantly underestimated – even ignored – in the past. Table 25.2 lists, in descending order, an "informed guesstimate" of the relative international incidence of morbidity from marine envenomations.

A world-wide database for the recording and collation of human injuries from marine toxins has now been established by the International Consortium for Jellyfish Stings. It is based in Australia and the United States.

PHARMACOLOGICAL AND CLINICAL FEATURES, AND THE MANAGEMENT OF SPECIFIC MARINE INTOXICATIONS

Rationale and general principles

Most marine toxins are complex mixtures of proteins, polypeptides and enzymes. Consequently, they are often locally pain-producing, dose-dependent in their effects, antigenic, and capable of initiating allergic reactions in susceptible individuals. They exert their harmful effects predominantly by their toxic properties. They may be thermolabile (ie denatured rapidly by heat), and may exert their clinical effects locally (ie at the site of their entry into the body), systemically (ie affecting the body as a whole), or both.

Importance of first aid

While the first aid management of marine poisonings may present difficulties, correct on-the-spot first aid for any marine envenomation is of the utmost importance.

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Table 25.1 The causes of human mortality from marine toxins which could be verified by firm documentation and/or first-hand interview (based upon data from *The Marine Stinger Reference Book 1996*, edited by J. Williamson, P. Fenner, J. Burnett, J. Rifkin)

Toxin source	Mortality
Puffer fish (tetrodotoxin) poisoning – "fugu"	approx. 10% lethality rate
Paralytic shellfish poisoning	approx. 6% lethality rate
Cnidarian (jellyfish) envenomations	hundreds of deaths
Ciguatera poisoning	approx. 0.1% lethality rate
Seasnake envenomation	approx. 150 deaths annually
Stingray spine injury/envenomation	19 documented deaths
Asiatic porpoise flesh poisoning	–
Weeverfish injuries (most deaths from secondary infection)	–
Catfish injuries	–
Zebrafish injuries	–
Stonefish envenomation	2–3 documented cases found
Blue-ringed octopus envenomation	3 confirmed cases
Sea anemone envenomation	1 confirmed case

Notes:
1 *Where no indication of numbers or percentages is given, no reliable data is available.*
2 *The relative incidence varies considerably from country to country.*
3 *Mortality, while not confined to, is commonest in tropical oceans.*
4 *Cnidarian envenomations world-wide are one of the more common causes of deaths.*
5 *Actual mortality from ciguatera poisoning has probably been overestimated in the past.*
6 *Deaths from most of the animals listed below "seasnake envenomation" in the table are very uncommon to rare.*
7 *Fatalities from stonefish envenomation are rare; no confirmed deaths have occurred to date in Australia*

Table 25.2 Approximate relative incidence of the causes of human morbidity resulting from marine envenomations and poisonings (based upon data from *The Marine Stinger Reference Book 1996*, edited by J. Williamson, P. Fenner, J. Burnett, J. Rifkin)

Relative incidence	Morbidity cause
1 (most common)	Coral cuts and abrasions
2	Cnidarian (jellyfish) stings
3	Spiny fish injuries
4	Ciguatera poisoning
5 (least common)	Poisoning from the ingestion of other marine animals (eg shellfish)

Notes:
1 *Morbidity is world-wide, in all oceans and seas.*
2 *Some morbidity (eg jellyfish envenomations) is very serious, even permanently disabling or disfiguring.*
3 *The extent of world-wide morbidity from jellyfish stings has been significantly underestimated in the past.*

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Virtually no marine envenomation need have a fatal outcome provided early and correct first aid is applied. Systemically acting marine venoms may have their effects reduced or controlled by the application of compression immobilisation bandaging. Both systemic and local effects of certain toxins may be minimised by the administration of specific antivenoms.

Marine antivenoms

Antivenoms against the venoms of chirodripid (multi-tentacled) box jellyfish, seasnakes, stonefish, and European weeverfish have been produced (though antivenom production in Zagreb has now ceased) and for the most part are dramatically effective. It is now clear from recent international research that the correct use of specific antivenoms is the method of choice in managing any serious envenomation. The cost and availability of antivenoms world-wide, in the regions of greatest need (ie "third world" marine locations) remain obstacles to the improved management of marine intoxications. Exciting research advances in antivenom production are happening, and these include lowering of costs, improved specificity, reduced side-effects, and reduction in antivenom molecular size (through "fractionated antibody preparations" or FABs), permitting more appropriate pharmacological distribution within the body tissues.

IMPORTANCE OF PREVENTION IN MARINE INTOXICATIONS

Most marine intoxications are easy to prevent. The major impediments to this are lack of awareness and of public education, even in advanced countries. The principles of successful prevention are intertwined with those of conservation of the environment.

- Never handle or interfere unnecessarily with marine animals (whether alive or apparently dead) in their natural environments.
- Always wear protective clothing (eg stinger suits for swimmers or windsurfers, wet suits and gloves for divers, protective clothing when fishing) when entering the marine environment during the summer risk season in known risk areas, such as tropical beaches, on isolated beaches unequipped with stinger enclosures or unpatrolled by Surf Life Saving patrols.
- Carry 2–3 litres of household vinegar, several broad elastic bandages, a small supply of ice or cold packs, some hot water, and some communication facilities (eg mobile phone with appropriate emergency ambulance numbers) when spending time in isolated tropical beach or marine regions.
- Learn and practise basic resuscitation skills by attending a recognised first aid course, prior to undertaking such journeys.

SPECIFIC MARINE INTOXICATIONS

Intoxication by tetrodotoxin

Pufferfish poisoning

Tetrodotoxin is a unique and potent molecule with a curious distribution in nature, for example, puffer fish, *Hapalochlaena* species (blue-ringed octopus), certain species of gobies and frogs. Atypically for a marine toxin, it is virtually non-antigenic and thermostable with a low molecular weight of 319 (Figure 25.1). Hence there is presently no antivenom available. This poisoning may occur world-wide, but is more prevalent in warmer oceans.

It is readily absorbed via the gastro-intestinal tract, producing quite selective blockade of sodium (Na^+) ion channels in excitable membranes. Clinically, this results in systemic neurological dysfunction, neurogenic paralysis of respiratory muscles and hypoxia. Death may ensue from brain hypoxia in the absence of effective first aid or subsequent medical management.

The principal source of tetrodotoxin poisoning ("tetrodotoxication") is ingestion of the flesh of pufferfish. This flesh is an eating delicacy in Japan, where it is known as "fugu"; there poisonings, with some deaths, still occur. Accidental poisonings due to lack of awareness or recognition of the danger still occur world-wide.

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Management of tetrodotoxication

The early induction of vomiting following ingestion would seem practical, as the toxin is non-corrosive. However, this manoeuvre must be attempted only in the fully conscious patient with no muscle weakness, within the first minutes following ingestion, and then only under skilled direction.

Once weakness and paraesthesiae have supervened the patient requires airway protection, and sustained ventilatory assistance, by expired air resuscitation (EAR) and/or intermittent positive pressure ventilation (IPPV), preferably with oxygen supplementation. There is no specific antidote, so airway protection and IPPV must be sustained until clinical recovery occurs (approx. 12–24 hours, depending upon dose and patient size and condition). Provided hypoxia, aspiration and injury are avoided, full neurological and clinical recovery can be anticipated. Remember that during the paralysis and "apparent unconsciousness" phase (in reality total paralysis with full awareness), the unfortunate victim may be able to hear what is being said around him or her by the rescuers and attendants. Reassurance and tact are required.

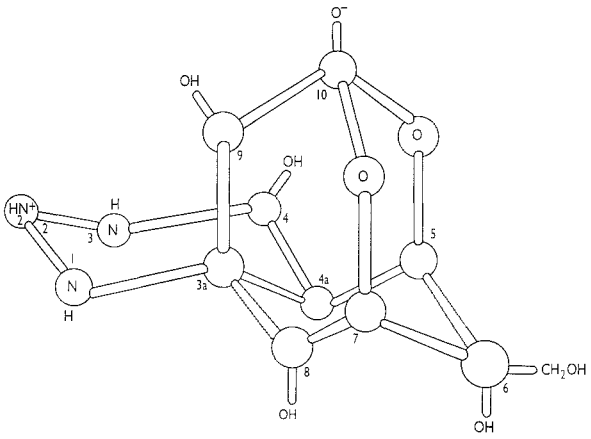


Figure 25.1 *The molecular structure of tetrodotoxin, the principal toxin from the flesh of pufferfish and in envenomation by the blue-ringed octopus (*Hapalochlaena *species). (Illustration courtesy of J. Pearn and Queensland Health Department.)*

Envenomation by the blue-ringed octopus

At least two venomous species of *Hapalochlaena* are known, *H. maculosa* and *H. lunulata*. The known distribution includes Australia (the entire coastline), south-east Asia, Japan, the Indian Ocean, eastern Papua New Guinea and New Zealand.

Intoxication is from a bite by a tiny, parrot-like beak located on the underside of the body, which injects a toxin-containing secretion of the salivary glands. Most bites do not result in serious envenomation, and all bites to date (with one known exception, in a scuba diver) have occurred from uninformed persons handling the small octopus while out of the water. Their attraction to fossickers (especially children) is enhanced by their small size (Figure 25.2) and its ability to iridesce its blue rings prettily when irritated. The bite is commonly painless, and may be ignored initially by the unsuspecting victim. A spot of blood on the skin may be the only sign. In a dose-dependent manner, the onset of

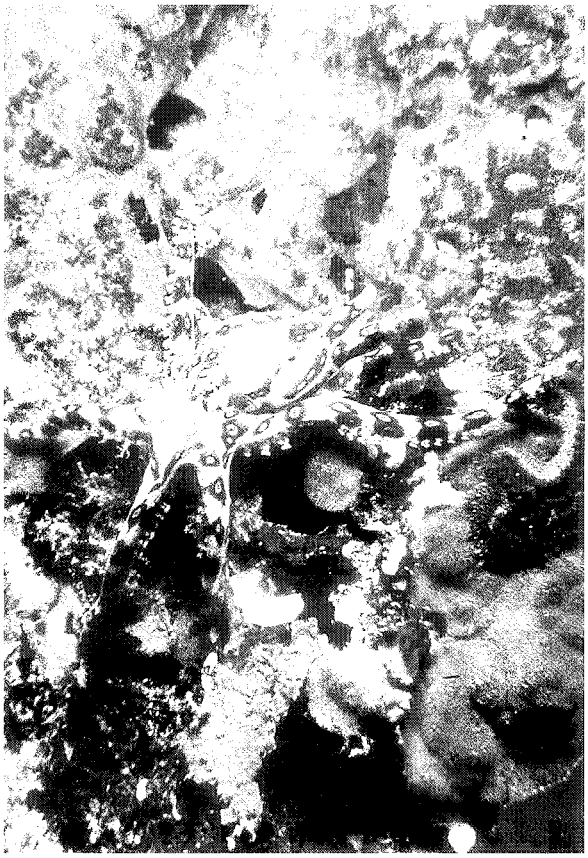


Figure 25.2 Hapalochlaena maculosa, *the smaller species of blue-ringed octopus. Note the effective camouflage and the blue rings superimposed on the dark brown markings. (Photograph courtesy of Surf Life Saving Queensland Inc.)*

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circum-oral paraesthesiae progressing to muscular weakness and respiratory difficulty may develop within minutes in seriously envenomed persons.

In principle the management of blue-ringed octopus envenomation is identical to that of pufferfish poisoning, but emphasising the more rapid onset caused by its injection through a bite. However, there is one crucial first aid difference, namely the early application of compression immobilisation bandages and splinting of the bitten limb, together with the immobilisation of the patient. This technique has the capability of trapping the venom at the injection site, thus preventing any serious consequences. This bandage and restraint must be maintained until the patient is delivered to direct and skilled medical care.

Molecular studies are suggesting the future possibility of the development of immune complexes against tetrodotoxin.

Cnidarian (jellyfish) envenomations

Jellyfish inhabit every marine ocean and sea (some freshwater species also exist but are of no stinging significance). Some mortality but frequent morbidity continue to be produced world-wide from these envenomations. The taxonomy of the many hundreds of genera and thousands of species of cnidarians is still being elucidated, and many remain undescribed.

Stinging mechanism in cnidarians

Cnidarians are characterised by the possession of intrinsic nematocysts. The nematocyst (Figure 25.3) is one of nature's most remarkable structures. It is a combined harpoon and injection syringe of microscopic size, and is the natural mechanism used by cnidarians for the capture of their prey. Thousands to millions of nematocysts populate the tentacles and sometimes the body (bell) of each stinging jellyfish, hydroid and sea

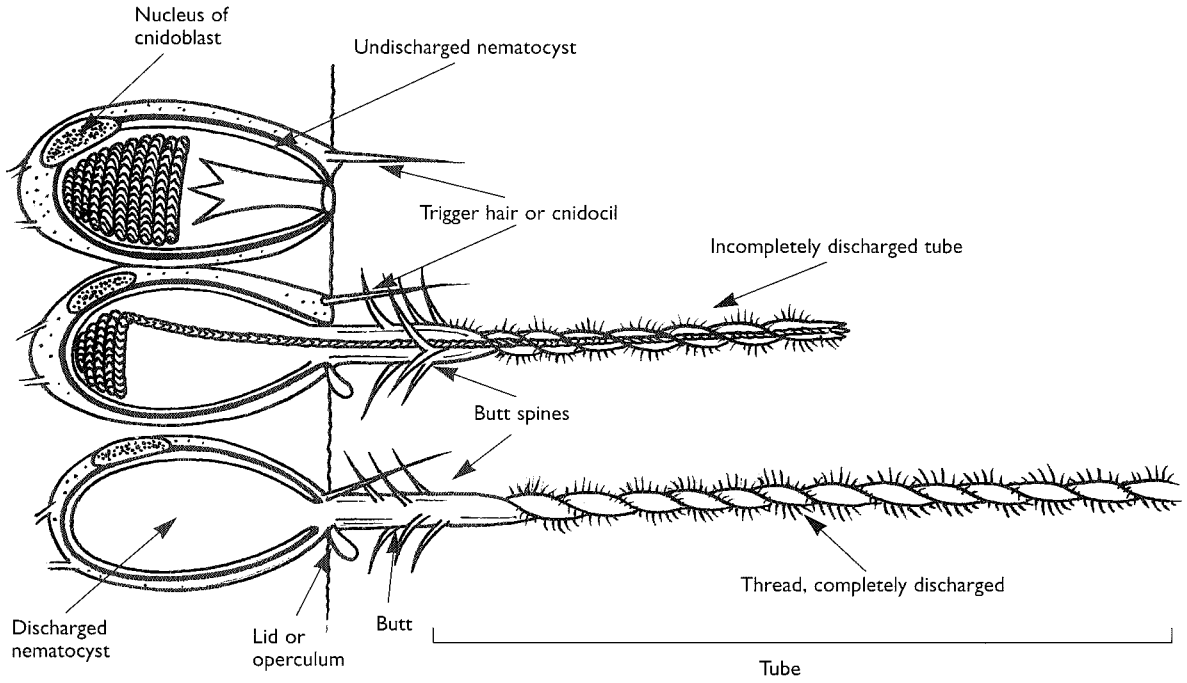


Figure 25.3 *A diagrammatic representation of the functional features of a typical nematocyst by Dr Ron Southcott. (Reproduced with Dr Southcott's permission from* Injuries to Man from Marine Invertebrates in the Australian Region.*)*

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anemone. Nematocysts are "fired" by mechanical and/or osmotic (chemical) stimuli, and each injects a micro-dose of venom (usually into the vascular dermis); some of this venom is deposited directly intravascularly. This plus the simultaneous deposition of scores of thousands of micro-doses of venom into a vascular area of a struggling victim leads to very rapid rises in blood levels of venom. This is in fact the most rapid envenomation process known.

The multiple nematocyst threads thus pin the tentacle to the skin of a stung victim, so that tentacular material, which may contain in addition thousands of *unfired* nematocysts, will be retained on the patient's skin. Hence, any early rescue and first aid manoeuvres which will fire these adherent microscopic nematocysts will increase the total envenomation dose delivered, and should be avoided. For example, jellyfish stings should never be rubbed with sand or a towel.

Several poorly understood factors (eg recent feeding, ageing, mobility and injury) probably influence the stinging propensity of jellyfish from day to day. This may contribute in part to the apparent variation in the dose of delivered venom and the wide spectrum of clinical severity of stings which are seen.

Nematocyst inhibition

This important consideration has caused some confusion among first aiders. With increasing research and clinical experience it has become apparent that the inhibition of firing in undischarged nematocysts is a complex and perhaps genus-specific affair. The present understanding is as follows, but is subject to modification as further research and understanding occurs.

- 2–10 per cent acetic acid in water irreversibly inhibits all presently known box jellyfish (cubozoan) nematocysts within about 30 seconds (Figures 25.4A, B and C).
- Household vinegar (from any source) is 4–6 per cent acetic acid in water, and is an ideal substance for this purpose.
- Acetic acid or vinegar does *not* relieve the pain of a sting resulting from the venom already injected by the fired nematocysts.
- Acetic acid or vinegar should not be used as a nematocyst inhibitor on the fresh sting from any other presently known cnidarian. (There is evidence that it may actually cause discharge in certain species such as *Cyanea* and *Physalia*.)
- No other substance has yet been convincingly identified as an effective nematocyst inhibitor for any cnidarian nematocysts from any species.
- While the identification of a chirodropid sting (see below) is possible on the beach in many cases, indicating the need to douse the sting area with vinegar, where the identity of another stinger is unknown, it is best not to apply any substance for nematocyst inhibition, but to commence with pain-relief measures immediately.
- Nematocysts retrieved from stung skin and examined by simple microscopy, may permit identification of the offending cnidarian in some cases. This retrieval is most simply achieved by placing a piece of sticky tape, sticky side down, over a small area of the fresh sting, then removing it and placing it under a low-power light microscope.

Pain relief in cnidarian stings

This is of the utmost importance as all cnidarian stings are instantly painful, they frequently involve children, and pain can be savage in large stings, rendering the victim incoherent and irrational. Restraint may be necessary.

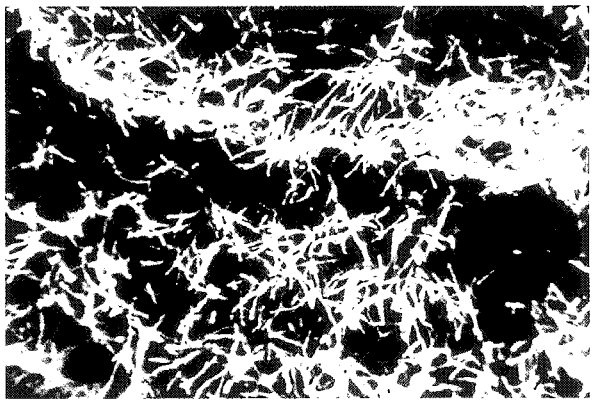
The immediate application of wrapped ice or ice-cold packs directly to the sting site has been well shown to provide effective, if not total, relief. In the case of a major chirodropid sting (see below), the best analgesia is provided by the injection of the specific box jellyfish antivenom. Direct application of heat has proved disappointing in cnidarian stings.

It is a frustrating fact that the application of any water to a cnidarian sting on human skin that has been allowed to dry off after exit from the water, may cause discharge of the unfired nematocysts still present on the skin. Water should not be used to douse jellyfish stings. There is also some concern regarding later showering by the victim following a major sting. Where vinegar

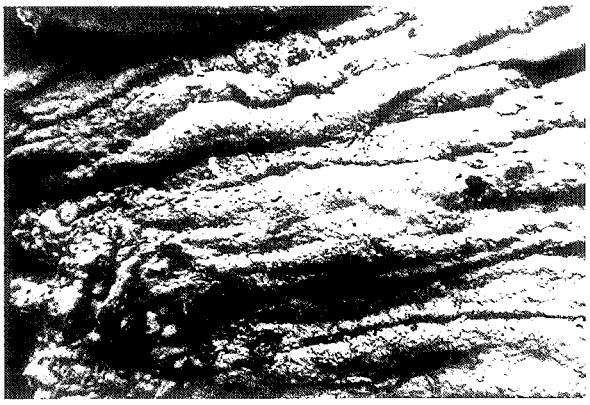
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A



B



C

Figures 25.4 A, B and C *A picture series, by scanning electron microscopy, from the original experimental observations by Dr Robert F. Hartwick, of James Cook University, illustrating:* **A** *the undisturbed appearance of the surface of a* Chironex fleckeri *tentacle;* **B** *a similar piece of tentacle following exposure to methylated spirits; and* **C** *a similar piece of tentacle treated first with household vinegar for 30 seconds followed by exposure to methylated spirits. Note the complete nematocyst discharge in* **B** *and the equally complete inhibition of nematocyst discharge in* **C**. *(Copyright* © The Medical Journal of Australia, *1980, 1:15–20; reprinted with permission.)*

application has not been used it is probably advisable to wait 24 hours before showering. Even then some increase in the sting site pain may occur.

Cnidarian venoms

These are complex antigenic mixtures of proteins and polypeptides, some of which are extremely potent. Their effects generally include potentially lethal effects upon human myocardium and perhaps central nervous function, vascular reactivity (both constriction and dilatation), dermatonecrosis (skin-killing), and the release of intense acute inflammatory kinins and components locally. There are no known direct effects upon neuromuscular function or on peripheral nerve function. The complex molecular mixtures exhibit mechanical and thermal lability, with a ready tendency to disaggregate and rearrange with experimental isolation and handling; some previous toxinological studies may have been rendered partially inaccurate because of these properties.

The venoms generally are antigenic, producing some specific immunoglobulin (Ig) responses with titratable and long-lasting (2–5 years) IgG antibodies. Recent immunological research by Burnett and colleagues in Baltimore is developing genus- and even species-identifiable titres from cnidarian venoms, against human serum from stung victims. This will permit the identification of the offending cnidarian, and even people at risk of a serious reaction, from the examination of their serum. (Interestingly, it could also theoretically permit the identification of an unidentifiable tentacle fragment of cnidarian by titrating its toxin against a serum with known antibodies.)

Allergic reactions (including anaphylaxis) although less common, are well documented. Such reactions to cnidarian stings are most likely in patients with a pre-existing allergic diathesis tendency such as atopic conditions, eczema, contact allergies, allergic asthma and hay fever.

Specific cnidarian envenomations

Box jellyfish (cubozoans)

These are the elite of the jellyfish world, possessing the most potent envenomation apparatus and the most

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evolved biological characteristics. Cubozoans are classified into chirodropids (multiple tentacles attached to each of the four corners of the trailing edge of the bell – ie up to 60 tentacles in total – Figure 25.5), and carybdeids (a single tentacle on each of the four corners of the trailing edge of the bell – ie only four tentacles in total).

Chirodropid stings are presently known in tropical Australia, the Indo-Pacific (especially Malaysia and the Philippines), Okinawa, the Gulf of Mexico, Myanmar (Burma), Sri Lanka and western Africa.

Carybdeid stings are presently identified in Australia (almost the entire coastline), the Pacific Ocean (mainly tropical), Okinawa, Oman, and the eastern Mediterranean Sea.

Chirodropid envenomation

This is the most rapid-onset envenomation process known. Hundreds of fatalities have been recorded in the Philippines alone, 63 in Australia and one each in the United States and Japan. The majority of stings are not life-threatening, but morbidity is legion. Some stings are disfiguring; all produce severe pain.

Full grown chirodropids, such as *Chironex fleckeri* in Australia, attain a bell size equal to that of a human head, with a curtain of tentacles streaming out behind the swimming animal for up to 5 metres. Despite their size, their milky white translucent colour makes them virtually invisible, even in clear, sunlit sea water (Figure 25.6). Hence the uninformed swimmer or duck diver can all too easily blunder into this massive envenomation armament.

Contact with the tentacles produces instant and savage local pain with the immediate initiation of a severe skin inflammatory reaction, pallor, weal formation, surrounding erythema and oedema, and in worst cases progressive full-thickness skin death where tentacle contact has been made (Figure 25.7). Scarring and even keloid formation may result, and secondary infection is documented. Tentacle material is avulsed from the animal by the struggling and rapidly withdrawing wader (stings usually occur close to the shore in water seldom deeper than 2 metres), and tentacle pieces remain adherent to the victim's skin as they emerge, often screaming, from the water. Women and children are particularly susceptible to serious envenomation because their

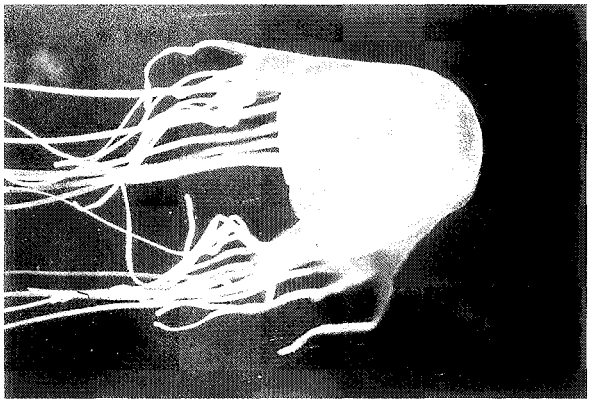


Figure 25.5 *An adult specimen of a north Queensland* Chironex fleckeri, *photographed at James Cook University by Dr Robert F. Hartwick. Note the multiple tentacles attached to each corner of the bell, one of the classic morphological features of chirodropids.*

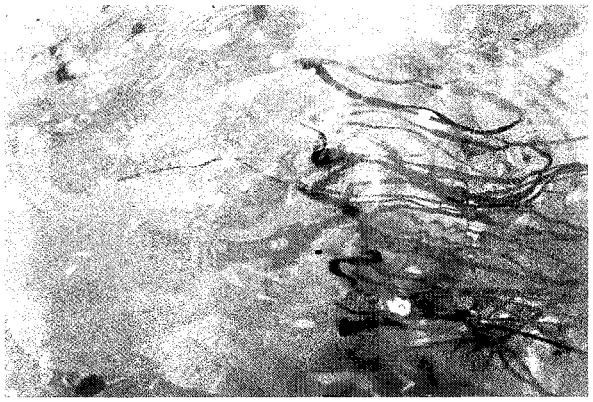


Figure 25.6 *The natural appearance of a large adult* Chironex fleckeri, *photographed in clear, sunlit shallow sea water in Nhulunbuy, in tropical Australia, in summer. Note the near-invisibility, even under such ideal observing conditions, of the bell, which occupies the left centre of the photograph. The animal is swimming from right to left. (Photograph courtesy of Dr Robert F. Hartwick.)*

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hairless skin permits more intimate tentacle contact. Most stings are on legs and hands, but the lower trunk is often involved. An approximate estimate of the seriousness of the situation may be made by measuring the total length of the tentacle weals. However, any sting which occupies a total area equal to or greater than one half of one limb should be treated as potentially life-threatening.

Management of serious chirodropid envenomation

- Restrain the distraught and struggling victim on the beach from rubbing and grabbing at the adherent tentacles.
- Commence resuscitation immediately in victims whose breathing ceases. *Nothing takes priority over immediate resuscitation.*
- Douse the whole sting area with 2–3 litres of household vinegar for 30 seconds. Do not attempt to remove adherent tentacle material.
- Next, in any major chirodropid sting, apply compression immobilisation bandages and splints over all limb stings and any vinegar-treated tentacle material still adherent.
- If available, administer specific Commonwealth Serum Laboratories (CSL) Box Jellyfish Antivenom (3 ampoules intramuscularly) into the outer aspect of the thigh muscles, above the compression bandage. Maintain resuscitation as necessary.
- Apply cold packs directly to the sting of conscious victims.
- Contact medical/para-medical aid, but do not leave the victim.
- Do not abandon resuscitation attempts until skilled medical decision-making is possible.
- Document the events of the rescue and resuscitation, including times.
- Where prudent to do so, a piece of sticky-tape applied briefly to a fresh sting spot will permit adherent nematocysts from the skin to be identified subsequently by simple microscopy.

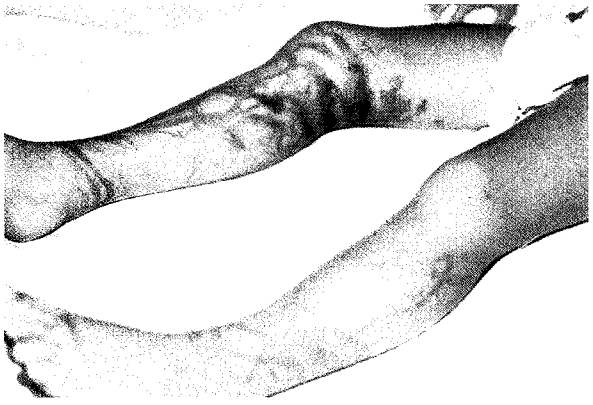


Figure 25.7 *The legs of a young girl 24 hours after a life-threatening* Chironex fleckeri *sting in northern Australia. Note the full-thickness skin necrosis behind the right knee. This area subsequently became secondarily infected, leading to scarring which required cosmetic surgery. (Photograph courtesy of Dr Peter Fenner.)*

Carybdeid envenomation

The known stinging carybdeids are:

irukandji (*Carukia barnesi* and other related species),
jimble (*Carybdea rastoni*), and
large carybdeids including *Tamoya* spp., morbakka, *Carybdea alata* and others

Undescribed species undoubtedly exist. With the exception of irukandji-type stinging, and certain large carybdeid stings (eg on the Sultanate of Oman coastline, and in eastern and western Australia) all carybdeid stings can be managed by vinegar application and pain relief. They do not produce life-threatening effects, although with larger stings the victim may have nausea and vomiting. No antivenoms presently exist, but the potential for their development exists.

Irukandji syndrome

The impressive reaction to the sting from a number of these widely distributed small carybdeids, which are found at least in tropical Indo-Pacific and Hawaiian seas, commences with a relatively minor sting pain leaving no skin mark or only a small one.

Some 30–90 minutes later there is the onset of ascending leg, trunk, lower back and chest pains, severe headache, nausea and vomiting, complete prostration in great distress, and a characteristic subjective feeling in the victim of impending doom.

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In seriously affected individuals this will be accompanied by severe systemic hypertension, pallor, sweating, and a clinical appearance causing great concern to the attendant first aiders. In a small percentage of such seriously affected patients there may be progression, some 12–24 hours later, into acute pulmonary oedema and hypoxia requiring ventilatory assistance. No deaths in Australia are presently known, but the potential for a life-threatening hypoxic crisis exists in such patients.

This syndrome is believed to result from the massive endogenous (from within the body) release of catecholamines (adrenalin-like substances). This concept is supported by minimal animal studies and the response of patients to intravenous (I.V.) alpha-blocking drugs such as phentolamine. The "catecholamine-storm" concept is also supported by the development of cardiac dilatation in such persons, demonstrable by echocardiography. Spontaneous myocardial recovery usually occurs over two weeks or more, in healthy individuals.

If an irukandji-type jellyfish sting is suspected (from the locality, multiple stings, skin appearance or knowledge of local occurrences), prevent the swimmer from re-entering the water in anticipation of the delayed onset of the full-blown syndrome. A swimmer in deep water so affected would be at risk of drowning. Management of an irukandji-type sting consists of the following steps.

- Seek medical aid and referral at the earliest opportunity following diagnosis. Unfortunately measures normally available to first aiders are inadequate to manage the severe pain and distress of a patient with an irukandji syndrome.
- The early administration of systemic narcotic analgesia, combined with appropriate direct medical supervision of cardio-respiratory status, pharmacological control of the hypertension, and judicious respiratory assistance and oxygen therapy intervention, both reduce the risk and severity, and shorten the duration of severe prostration in such patients.
- Most patients require a convalescent period of 2–3 weeks following such an envenomation with persisting lassitude, muscle soreness, and diminished energy levels. Spontaneous, if gradual, recovery is the rule.

Envenomation from large carybdeids

Known as "fire jellies" in some Pacific regions, these large jellyfish, with one broad ribbon-like tentacle on each corner of their large (head-sized) bell, can inflict very painful but usually non-dangerous stings. Management is as described above for box jellyfish stings generally (vinegar and cold packs). Occasionally, large carybdeid stings are associated with nausea, vomiting and pain-induced syncope. However, large carybdeid *(Tamoya)* stings in the Omani region may be life-threatening, with shock and documented renal decompensation. Management is symptomatic; it should include dousing with vinegar in the first aid stage, and may require intensive care facilities.

Bluebottle and Portuguese man o'war (*Physalia *spp.) envenomations

These animals produce the world's most common jellyfish stings. In eastern Australia alone, many thousands of painful but mostly harmless stings occur each summer from the Pacific bluebottle *(Physalia utriculus)*. However, the Atlantic Portuguese man o'war *(Physalia physalis)* is a larger and more dangerous animal which can produce a severe envenomation syndrome with muscle pains, nausea, vomiting and a distraught mental state. It has three documented human deaths to its credit on the eastern United States coastline. Unfortunately, a Pacific counterpart (the Pacific *Physalia physalis* or Pacific man o'war) has recently been formally identified. Its existence along the Pakistani coastline is also probable. This animal also can produce a relatively severe and prostrating syndrome requiring occasional hospital admission. No Pacific deaths are known.

The management for such stings is as follows.

- Apply cold packs for the pain.
- Do not apply vinegar.
- Do not wash the sting with water. Picking off the stubborn tentacle piece using fingers may be necessary.
- Do not apply compression immobilisation bandages.
- Oral analgesics such as paracetamol or aspirin are useful.

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Selected cnidarian stings of significance

Stomolophus nomurai, a species of "cabbage-head jellyfish", causes thousands of stings and some deaths in China. Management is symptomatic, with respiratory support for acute pulmonary oedema in severe cases. No antivenom exists. Nematocyst inhibition studies are unknown.

Pelagia noctiluca, the "mauve stinger", is a widespread and important stinging jellyfish found in the Mediterranean Sea and occasionally in the Indo-Pacific. This animal is responsible for the only well documented (and near-fatal) anaphylactic reaction; this occurred in an asthmatic patient on a Mediterranean beach. Its venom appears to be more than commonly antigenic. In the absence of serious allergic manifestations sting management is symptomatic, with sea water washing (for a still wet sting only) and cold packs.

Gonionemus sp., a tiny swarming jellyfish, causes human stings in the Sea of Japan, especially in the Vladivostok region. The sting results in the production of a spontaneously reversible 24-hour syndrome of mental disturbance bordering on psychosis, sometimes requiring physical restraint. Nothing is yet known about the venom, and management is symptomatic.

Sea anemone stings may be very severe. These have a world-wide distribution, in tropical and temperate seas and at all depths. Their venoms are very toxic, and severe and spectacular local reactions, sometimes with crippling pain, are well documented. Vesiculation (blister formation) is characteristic. Despite this, their toxicity remains relatively unappreciated. One recently documented death from fulminant hepatic failure is known. Management is entirely symptomatic. Local pain relief by cold pack application is helpful. Serious stings require medical referral.

Allergic reactions to cnidarian stings

These are characterised by local oedema and itching, with progression sometimes to widespread swelling, itching and erythema remote from the sting site. Patients with a pre-existing allergic history are susceptible. Anaphylaxis, although very uncommon to date, is the most severe allergic manifestation. (It is of historical interest that anaphylaxis was first described in the medical literature by French scientists working with venoms of sea anemones and Portuguese men o'war.) Anaphylaxis is treated in the same urgent manner as anaphylaxis from any other cause:

- resuscitation (airway, breathing, circulation),
- adrenalin injection at the earliest opportunity, and
- I.V. access and fluid administration.

Hospital admission is essential.

Recurrence of local allergic signs and symptoms in some cnidarian stings (erythema, skin rash, itching, clear discharge) is well documented. This may happen 4–6 weeks after apparent healing, and can recur more than once.

The early application of compression immobilisation bandages over the envenomation site in a patient known to be at risk of a severe systemic allergic reaction may possibly diminish the severity of the subsequent reaction. This method has not been applied in practice to date in a marine envenomation.

SPINY FISH INJURIES

These are among the most common of all marine injuries, since the majority of fish spines are venomous. Fish equipped with venomous spines are found in marine, brackish and fresh water.

It may be noted, too, that although the sea star *Acanthaster planci,* the infamous crown-of-thorns sea star, is not a fish, injuries from the spines of this animal are common, and the general principles that apply to spiny fish injuries apply equally to crown-of-thorns spine injuries. Therefore, the following remarks on first aid management should be taken to include such injuries.

General considerations

Venomous fish spines may be located on any fins – dorsal, ventral, pectoral or caudal. They may also be associated with mouth parts. Some spines are erectile. They are always used for prey capture or for defence. No known spiny fish attacks human beings.

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Venomous fish spine injuries are most commonly sustained from accidental or intentional handling of the animal when it is captured, for example, in a fish net, on a line, or in an aquarium. Scuba divers are also at risk. The spines have remarkable penetration powers, and can transfix clothing, even thin shoes or thin gloves. Injuries can also be sustained by handling a dead fish.

Venom may be introduced into the wound during the puncture by the spine integument (eg stingray), or by actual injection through a hollow or grooved spine from an associated venom sac (eg stonefish).

Spines are frequently brittle, and fragments may break off and be retained in the depths of the wound. These are usually visible on radiographs.

Most fish spine injuries are accompanied by instant and severe local pain, which may be sustained for hours or days in untreated cases. Such pain (eg that caused by stonefish, weeverfish or bullrout injuries) may in specific cases be devastating.

Fortunately, most fish spine injuries pain may be greatly diminished on site by the immersion of the envenomed part in hot (not boiling!) water.

All significant fish spine injuries should receive early medical attention and follow-up.

Compression bandaging has no place in the first aid management of any known fish spine injury.

Specific identification of the offending fish is usually possible and is assisted by knowledge of the distribution of the different venomous fish. For example, the bullrout is known only in Australia and venomous weeverfish appear to be confined to the European coastline.

Stonefish sting (*Synanceia* spp.)

The archetypal spiny fish injury is the stonefish sting. This injury will be described in detail and its management serves as a model for any significant spiny fish sting. (Fortunately, not every spiny fish sting produces such severe effects.) Where special or unusual features apply to a particular sting, these will be described.

Stonefish are known on coral reefs and coastlines throughout the tropical and, less commonly, temperate oceans. Stonefish sting is quite common in some regions, but only two or possibly three human deaths (two of which were in Madagascar) can be discovered in the literature. Despite this, the stonefish is still popularly believed to be, and is repeatedly reported by the media as, a "killer". Nevertheless a stonefish sting is a terrible experience.

At least two venomous species are known, *Synanceia trachynis* (= *S. verrucosa*) the reef stonefish and *S. horrida* the estuarine stonefish, but from the first aider's point of view these may be regarded as identical.

The stonefish sting is considered by most victims as the most painful experience of their lives. Most stings, from one or more of the 13 venomous dorsal spines, occur on the foot or the hand. The stonefish is the master of camouflage, lies perfectly still, and will not be seen before, or often even after, the injury has been sustained. The grooved spines each have connection at their bases with a venom sac, and as the unsuspecting foot or hand presses down upon and is pierced by the razor-sharp spine, the venom sac is compressed against the animal's back, forcing its contents through the spine and into the depths of the wound.

Stonefish venom is protein in nature, antigenic, and intensely irritant to local tissues. The pain is instantaneous and savage, rendering most victims helpless and incoherent. Sometimes only a single puncture wound is present, but typically local oedema develops rapidly and is usually accompanied by a characteristic blue discoloration around the edges of the wound. Systemic effects, other than indirectly from the terrible pain, have never been seen by the writer. There is no compromise of local blood supply to the injured tissues, and significant bleeding does not occur. The tip of the spine may be retained in the depths of the wound.

Allergic reactions to stonefish envenomation appear to be unrecorded. All significant effects appear to be toxic in nature.

Management of stonefish sting

- Plunge the envenomated part into hot (not boiling!) water. Removal of the part will result in immediate return of the severe form of the pain. Keep the part immersed, and the water hot. Do not ask the agonised victim to test the water temperature – the first aider should test it first.

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- Bring the patient to immediate medical aid.
- Oral analgesics such as codeine and aspirin preparations are seldom of use, but may be tried.
- The medical management is essentially pain relief. This is the first priority, and is achieved by a regional nerve block with local anaesthetic, or by specific antivenom injection if identification is verified, or (least effectively) by local anaesthetic infiltration around the puncture wound.
- I.V. access – essential prior to regional blockade and antivenom injection, and the use of any I.V. narcotic analgesia.
- X-ray of the wound to exclude the possibility of retention of a major fragment of the spine or other foreign body.
- Specific stonefish antivenom injection – as with any antivenom usage, this is performed by intramuscular injection, and in the presence of full resuscitation facilities and skills. Dosage is titrated against clinical response.
- Exploration of the wound is controversial, not always necessary, and should involve surgical consultation. Washing venom from the depths of the wound sounds appealing, but presents difficulties and may be unnecessary.
- Tetanus prophylaxis is important and should always be considered. The current state of tetanus immunity of the patient should be enquired into, and appropriate booster or immunisation performed.
- Prevention of secondary infection – this requires adequate early wound toilet, removal of significant foreign bodies from the wound, possible antibiotic prophylaxis or therapy, and adequate follow-up.

Other specific spiny fish envenomations

Stingray spine injuries

The stingray spine, wielded on the tail shaft with great power, purely as a defensive response, may cause significant trauma, as well as envenomation. Most injuries are on the lower leg of waders who step upon the animal as it lies on a sandy shallow bottom. The spine is very brittle and may snap off in the wound, but the venom is contained in the integument of the spine, and is ruptured and released into the wound tissues. This venom produces inexorable local tissue necrosis, and any stingray injury requires skilled medical assessment as soon as possible. Any stingray wound to the trunk (chest or abdomen) is a potentially grave medical emergency and requires immediate referral to a major medical centre. Painful limb injuries respond to hot water immersion.

***Australian bullrout* (Notesthes robusta)**

This fish is found in brackish marine streams, is also well camouflaged, and produces an injury bearing considerable resemblance to a stonefish sting. It is not known for certain whether stonefish antivenom is effective for a bullrout envenomation; it should probably not be used for this purpose without informed medical consultation.

Secondary infection (including tetanus) of stingray and bullrout injuries is always a possible complication, and must be guarded against by appropriate and early wound management. Several of the documented deaths from stingray, catfish and (probably) zebrafish injuries have resulted from this complication.

Aquarium injuries are among the commonest sources of spiny fish injuries. Both amateur and professional aquarium keepers are affected. Consequently, tropical spiny fish injuries may present in any part of the world, including urbanised cold temperate cities.

There are three first aid measures which are contraindicated for spiny fish injuries.

1 As there are no confirmed spiny fish envenomations which carry the invariable potential for acute systemic effects clinically (eg paralysis or myocardial decompensation), compression immobilisation bandaging should not be used unless an anaphylactic reaction is diagnosed.
2 Wound incision or exploration should not be attempted by the first aider. If a small foreign body is visible in the wound, it should not be removed by first aiders without prior and early medical consultation.
3 Local injection or application of emetine for spiny fish stings is no longer indicated.

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SEASNAKES

Seasnakes are air breathers, equipped with lungs. They may be distinguished from eels by the absence of gills, and by the small tissue flaps over the paired nostrils. They have a paddle-shaped tail.

Seasnakes are curious, ubiquitous marine animals. (They may venture upstream in coastal rivers and streams.) They are graceful swimmers and are found at all depths in the sea, and may remain submerged for up to 20 minutes. The oft-stated claim that seasnakes are invariably aggressive and always attack humans in the water is not borne out by experience. Other common but mistaken beliefs are that seasnakes cannot bite effectively and that they possess weak venoms. In fact some seasnake venoms are among the most toxic known (see Table 24.2), and deaths and near deaths are well documented in Madras, Myanmar, Oman and Malaysia. Near deaths have occurred in Australia and Japan.

Seasnakes are most common in warm tropical waters. Interestingly, their presence in the Caribbean region is not yet confirmed.

One of the seasnakes most widely implicated in human envenomation is the beaked seasnake *(Enhydrina schistosa)*. The antivenom against seasnake bite is prepared against the venom of this species and of the Australian tiger snake (a horse serum) and is effective against most known seasnake venoms. This makes the accurate identification of the offending seasnake unnecessary.

The bite of a seasnake may be completely painless. The venom exerts neuromuscular blockade and somatic myotoxicity, but no coagulopathies (blood clotting disturbances).

There are no venom detection kits generally available for seasnake bite.

For practical first aid purposes, Australia has no non-venomous seasnakes.

CONE SHELLS

While there is firm documentation of at least 12 human deaths from cone shell envenomation, serious envenomation remains uncommon and is due to excessive handling of specimens. In comparison with other causes of serious marine envenomation, the evil reputation of the cone shell is scarcely deserved, especially since envenomation can be completely prevented by leaving cone shells alone.

The two species implicated in human death are *Conus geographus* (mainly) and *C. textile*. They are distributed widely on tropical and warmer temperate sea floors and reefs. They possess an elegant envenomation apparatus, consisting of an ejectible radula or tooth, which is shot out from a proboscis to spear and envenomate its prey. The proboscis may protrude from anywhere along the slit in the shell, but more frequently it appears at the pointed end of the shell. The radula may penetrate clothing in someone's pocket! Envenomation is usually painful.

Cone shell venom is protein and antigenic. Past attempts to develop an antivenom have not been carried to completion. The venom produces neuromuscular blockade, respiratory paresis and resulting hypoxia. Death is completely preventable by the application of expired air resuscitation. The venom has no direct effect upon the heart or brain.

Management of serious cone shell envenomation

- Protect the airway.
- Expired air resuscitation (EAR) if indicated.
- Early application of compression immobilisation bandaging and splinting. Most envenomations occur on the hand.

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CORAL CUTS AND ABRASIONS

These injuries are essentially traumatic, and less commonly involve envenomation (stinging coral injuries may be regarded as, and treated as, cnidarian stingings). None the less they are very common, and capable of producing chronic and even serious morbidity when early and correct first aid is not administered.

The injury is a direct abrasion/laceration and/or cutting from impact between bare skin and the coral. The wound is usually impregnated with proteinaceous coral polyp material and sometimes fragments of calcified coral skeleton. Some bleeding may occur, but initially the injury may pass unnoticed. Within hours, in the absence of early first aid, the wound becomes inflamed, painful and swollen, with regional lymphadenopathy and secondary infection common. Occasionally, in susceptible individuals elements of clinical allergy may develop locally, with itching, extending oedema and serous discharge.

Secondary infection is a most important complication to prevent; this is achieved simply by early first aid. Secondary bacterial infection, facilitated by the presence of proteinaceous material embedded in an untended wound, may be from the usual organisms such as staphylococci or streptococci, but may also develop from pathogenic marine bacteria, such as vibrios or corynebacteria organisms. These latter do not respond to the usual antibiotics, and their laboratory culture and sensitivity testing requires first the suspicion of their presence in the wound, and the use of special culture media by the microbiologist. *Vibrio vulnificus* is a particularly dangerous marine pathogen, and limbs and even lives have been lost from this marine infection in neglected wounds. The reader is referred to more appropriate texts for more detailed information on these matters.

The first aid for coral cuts and abrasions is simple and effective, but must be immediate following such wounds – that is to say, within 3–4 hours.

- Control any serious bleeding.
- Manually but gently remove any visible larger coral fragments from the wound, using clean washed hands, preferably wearing latex gloves.
- Using a gentle detergent, such as chlorhexidine diluted in warm fresh water (not sea water), and a soft bristle brush, gently but firmly scrub the fresh bleeding wound to clean out all debris. Increased bleeding may well occur but is not a contraindication. Pain will increase, and for extensive or deep wounds, especially in children, medical assistance with anaesthesia may need to be sought; this requirement must not be permitted to delay the wound toilet unduly.
- Apply a clean, firm dressing and bandage, and arrange for medical referral.

Medical attention will include X-ray of the wound if considered appropriate, tetanus immunisation as required, wound inspection and further attention as indicated, possible antibiotic cover, and follow-up arrangements.

It cannot be overemphasised that such simple early first aid measures for these injuries may prevent months of disabling and even serious morbidity from infection, which tends to be unresponsive to delayed medical treatment.

CONCLUSION

As for so many other first aid situations, simple and common-sense measures applied immediately after most marine envenomations will relieve suffering and reduce the duration and severity of the injury and intoxication. No amount of later, skilled or technologically advanced medical management can substitute for immediate first aid, correctly applied. *Those on the spot play the most vital role.*

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PART

IV

INFECTIONS *and* PREVENTION

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CHAPTER TWENTY-SIX

WOUND INFECTIONS

Joan Faoagali

The management of wounds is one of the most frequent tasks of the first aider; the prevention of infection is one of the most important contributions the first aider can make to the uncomplicated convalescence of the victim.

The skin and mucous membranes are the most important protective organs of the body. Intact skin surface and mucous membranes keep resident and transient microorganisms out of the underlying tissues.

Any break in an epithelial or mucous membrane will allow organisms normally on the surface (resident or normal flora) and organisms from the environment (transient flora) access to the blood vessels and deep tissues. There they may multiply and cause local infection, or spread widely throughout the body, causing septicaemia and possibly even distant abscesses. The skin surface is a complex physical and chemical barrier. It is a true living organ, metabolising actively and composed of defensive cells, protective chemicals and physical agents which repel invasion. There are three factors which may increase or diminish the likelihood of a breach of this protective barrier.

1 Patient (host) factors
2 Organism factors
3 Environmental factors

The patient's natural humoral immunity (eg immunoglobulins), white blood cells (including polymorphonuclear leucocytes and macrophages), cellular immunity and normal metabolism (eg the production of tissue chemicals) will usually confine any introduced microorganisms to the area of the breach. This may produce local redness (inflammation) or the development of an abscess (pus) followed by repair.

All external body surfaces and some internal surfaces – either skin or mucous membranes – have a normal flora of microorganisms. This population of living organisms is relatively stable for each individual. These normal organisms also have important metabolic and protective functions. Some normal gut flora and skin organisms produce vitamins (eg vitamin K) and other chemicals essential for good health. These normal flora organisms constitute a stable population, which can be altered by the use of antibiotics or may change following metabolic defects or surgery. When such changes occur, serious consequences may result.

The skin, upper respiratory tract, lower genital tract, gut and wounds not only have this normal flora, but may also be transiently colonised by ubiquitous environmental organisms. Sometimes these micro-organisms are selected by the use of antibiotics to which they are resistant. Some microbes produce toxins which enable them to penetrate or bypass natural barriers and become infecting agents.

The following surfaces have a stable normal flora: the intestinal tract from mouth to anus; the skin; the upper respiratory tract, including the nose, sinuses and external ear; the conjunctival surfaces of the eye; and the genito-urinary tract.

The following surfaces normally have no microbial flora: the meninges bathed by the normally sterile cerebrospinal fluid; the bladder, ureters and kidneys; the uterus and fallopian tubes; the peritoneum; the pleura; the pericardium and joint surfaces.

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By contrast, organisms may normally be found transiently in blood; bone marrow; the alveoli, bronchioles, bronchi and trachea below the laryngeal vocal cords; and the upper urethra. Neutrophils (phagocytes) clear these rapidly, or normal body fluids (eg urine or mucus) and cilia flush or carry these transient organisms to the exterior and enable them to be expelled into the environment where they die or multiply depending on their place of deposition.

If foreign material is introduced to a wound or into a normally sterile cavity, this material may protect contaminating organisms from the body's defensive activities and allow the organisms to multiply unchecked. If the patient's white cells, blood supply and body chemicals are not working normally and normal epithelial barriers are bypassed, then the risk and severity of any infection is increased (Figures 26.1 and 26.2).

In Figure 26.2, the microorganisms likely to be present in the black eschar will be:

- normal skin bacteria, including staphylococci, and
- contaminating transient flora including coliforms, *Bacillus* species and other spore-forming organisms such as *Clostridium* species.

The eschar will harbour these microbes and they will have the potential to invade the underlying tissues, particularly if they are traumatised during walking or by pressure from clothing or shoes. The plantar surface of the left great toe shows an epithelene break with a puckered scab through which normal and environmental flora (bacteria) could gain entrance to the subcutaneous tissues and even the phalangeal bone.



Figure 26.1 *Ischaemic feet. Note the shiny and scaly skin and the dry gangrene at the tip of the left great toe which occurred after nail cutting. The second right toe is also showing dry gangrene associated with vascular insufficiency of this area. No current infection. Normal and transient microbial flora would be found in any swabs taken from these areas.*

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Figure 26.2 *Plantar surface of a patient with peripheral vascular insufficiency and dry gangrene on the tips of the second right toe and the second and third toes on the left foot.*

The shiny and scaling skin indicate the poor vascularity and nutrition of the feet.

NORMAL (RESIDENT) FLORA

The normal flora of the skin and mucous membranes form a complex ecological system of interacting saphrophytic microorganisms which are not pathogenic while they remain on their specific surface.

Each area of the skin and each of the mucous membranes have different microorganism types and numbers. The mix of microbes depends on multiple factors including the temperature, pH and atmosphere of the area.

Skin organisms vary from place to place on the skin surface. *Staphylococcus epidermidis* is the most commonly found organism in all areas. *Staphylococcus aureus* may be found in the noses of some 60 per cent of people.

Propionibacteria (anaerobic Gram-positive rods) are also widely distributed on the skin and have an important role with *S. epidermidis* in maintaining the normal physico-chemical environment of the skin. These "good" microbes create an inhibitory milieu which discourages pathogenic or "bad" microbes.

Where mucous membranes and the skin join there are flourishing mixed colonies of microorganisms from the mouth, from the respiratory tract and from the gastro-intestinal tract.

In the practice of first aid, one deals mostly with cuts and lacerations which involve the skin. To remember which organisms can infect such wounds it is helpful to imagine the body divided by a belt around the waist. Above the belt *S. epidermidis* is the predominant organism

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of normal skin. Below the belt bowel flora (Gram-negative aerobes and anaerobes) and environmental flora including spore-forming organisms are commonly found.

This means that cuts and scratches of the legs and feet are more likely to be colonised or infected with spore-forming organisms or Gram-negative organisms than are similar lesions on the upper part of the body.

The mouth and pharynx have a large number of different organisms normally present. This includes streptococcal species, alpha and non-haemolytic *Neissera* species, diphtheroids (short aerobic Gram-positive rods), *Staphylococcus* species and a variety of anaerobic bacteria.

Viruses and yeasts are not normally found in the mouth.

The normal stomach has an acid pH, and the microorganisms found here include those swallowed from the mouth and pharynx as well as those ingested with food and fluids. Normally, these organisms are destroyed in the stomach and the resting stomach has few microorganisms.

The small bowel has an almost equal mixture of aerobic and anaerobic organisms in small to moderate numbers. These organisms colonise the bowel wall and interchange freely with others living in the bowel contents. They assist with digestion and the production of various products, such as vitamin K.

The large bowel has a heavy bacterial load made up principally of anaerobic bacteria. Up to 10^{10} organisms per gram of faeces will be anaerobic Gram-negative and Gram-positive bacilli and cocci. Up to 10^{9} organisms per gram of aerobic Gram-positive and Gram-negative bacilli and cocci may also be found in faeces and colonising the wall of the colon.

The presence of these enormous numbers of microbes in stool and excrement are the reason that handwashing after defaecating or handling or dispensing of bowel contents is so important.

MICROORGANISM FACTORS

Some harmful microorganisms (eg beta haemolytic streptococci) are able to establish themselves in tissues when only a very few are present. This is because they produce potent toxins which help them to break down the body tissues or immobilise and destroy the white cells. Streptokinase, phospholipases and hyaluronidases are some such enzymes, which break down normal body tissues and are produced as exotoxins by these microbes.

Other organisms (eg staphylococci), require larger numbers to be deposited before they can establish an infection. Foreign material such as dirt, gravel, soot or even surgical sutures can protect staphylococci and allow them to multiply to sufficient numbers to produce infection.

Still other invaders require several different organisms to be present before an infection can be established. Gas gangrene, a potentially deadly complication of wounds, is usually the result of a mixture of microbes multiplying in the tissues to create the right oxygen-free conditions for the gas-producing clostridia to multiply.

Organisms commonly found in wound infection

The organisms commonly associated with wound infections are listed in Table 26.1.

Most of these organisms are found as normal flora on the individual's skin or mucous membranes and are thus autogenous infections. Most may also be spread from patient to patient, resulting in cross-infection.

Washing hands between patients, the use of sterile or disposable equipment and careful disposal of contaminated objects such as dressings, scalpels, or bed sheets will minimise the risk of cross-infection.

Fungi such as *Candida* – the microbe which causes thrush – rarely cause wound infections but may be found contaminating wounds in patients who have been treated with antibiotics. The antibiotics destroy the susceptible bacteria and allow the fungal species to multiply unchecked because they are not inhibited by the antibacterial antibiotics.

Viruses rarely cause wound infections. Occasionally, herpes simplex virus (the virus which causes "cold sores") may be inoculated into a skin breach resulting in a local herpes infection such as herpetic whitlow. It is important that persons with cold sores wash their hands

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Table 26.1 Common organisms causing wound infections

Organism	Likely source
Staphylococcus aureus (Gram-positive cocci including MRSA)	Patient's normal (skin) flora First aider/staff hands Occasionally from the environment (dust)
Streptococcus pyogenes (Group A lecta-haemolytic streptococci) (Gram-positive cocci)	Patient's normal (respiratory) flora First aider/staff respiratory tract (via contaminated hands) Unwashed staff hands contaminated with those organisms from lesions on other patients
Coliforms (Gram-negative bacilli such as *E. coli, Proteus* species, *Klebsiella* species)	Patient's normal gut flora Unwashed hands of first aider/staff
Pseudomonas sp.	Contaminated water reservoirs, eg tap spouts, sink drains, flower vases Unwashed hands of first aider/staff Weak disinfectants
clostridial species (*Clostridium tetani,* causing tetanus, *C. welchii,* causing gas gangrene.)	Environmental dust and dirt Patient's own normal bowel flora Unwashed hands of first aider/staff

prior to treating wounds to ensure that the herpesvirus in the cold sore is not inadvertently transmitted to the wound being treated by the carer's hands.

Carer's with extensive cold sores or any other infection should not treat patients with wounds, except in an emergency, because of the increased risk of cross-infection that occurs when the carer has an active infection.

ENVIRONMENTAL FACTORS

Environmental factors influencing the risk of wound infection include physical agents such as moisture, heat and cold. Such factors can affect both the organism and the host, thereby improving or limiting the conditions which predispose to the development of wound infections. Most organisms infecting humans grow most actively at 35–37°C, but cold can decrease the otherwise protective blood flow, allowing organisms to become established in the tissues.

Microorganisms exist everywhere in nature. They form a diverse ecology which includes excreted human microbes and plant, soil and animal microbes. Some of these organisms are not viable at normal body temperatures (37°C) and, therefore, will not be able to cause invasive infections. They may, however, be able to multiply on the wounded skin surface or nose where the normal temperature is 28–32°C. These microorganisms are often resistant to antibiotics and disinfectants. A wound allows them access to areas normally inaccessible to them, and subsequently they may multiply and cause infection. There is no way that the general environment can be sterilised (ie freed of all microbes). We live in a highly populated world of potentially harmful microorganisms kept at bay by the skin. Infection-free living is a balance between reducing harmful microorganisms in the environment by good hygiene and sensible cleanliness including handwashing; and by removing any inoculated "foreign" microorganisms from wounds.

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Examples of removable reservoirs of microorganisms include bodily wastes, unrefrigerated food, damp, reusable cloths and mops, dust and dirt. If weak solutions of disinfectants are left undiscarded, not only are organisms able to survive, but resistant strains emerge. If a sick patient with reduced resistance to infection is being nursed in bed, other potential reservoirs of harmful microorganisms, such as pot plants and flowers, should also be removed.

Prevention of wound infection can be seen to involve the following features.

1 Limit the access of microorganisms to wounds, by physical cleaning, the use of sterile dressings, surgical debridement, and aseptic procedures.
2 Remove all foreign material such as dirt, metal and dead tissue.
3 Maintain a good blood supply and nutrition to the affected area.
4 Use appropriate antibiotics.

The practical measures which can be implemented to minimise the occurrence of wound infections revolve around the appropriate use of effective antiseptics or disinfectants for the environment and equipment, the hands of attendants, and the wound itself.

Appropriate antiseptics and disinfectants are widely available, but their effective use depends on knowledge of their actions, advantages and disadvantages, their cost and where they should be used.

Infection can be spread by air or by direct contact. Although hand transmission is probably the most important route of contact spread, equipment or materials can also be a major source or route of transmission, and effective decontaminants should be applied to these items. This applies not only to the prevention of bacterial infection but to the transmission of viruses such as HIV from contaminated instruments or fluids.

Microbial decontamination is a process which reduces the number of organisms to a level which will be insufficient to initiate an infection in a susceptible site. It is achieved by cleaning, disinfection, or sterilisation. Each of these processes tends to be progressively more effective, but more costly and more likely to damage the treated item.

Cleaning involves the removal of organisms and soil. Washing with soap or detergent and water will remove most of the organisms and protein or other organic material which may provide a source of nutrients to organisms as well as protecting them against drying and the killing power of disinfectants.

Disinfection is a process which kills or inactivates organisms, but not necessarily spores. It may also be associated with removal of organisms and does not necessarily eliminate all organisms, but reduces them to a safe level.

Spores

Spores are a "resting" form of bacteria and are the means by which some bacteria are able to withstand adverse conditions such as drying, heat, lack of nutrients or exposure to antibiotics. Bacteria with the ability to form spores "round up" and become metabolically inert which protects them from destruction by brief periods of heating or exposure to disinfectants or antibiotics which act on metabolising organisms.

The most important spore-forming bacteria are the *Clostridium* species which are responsible for the diseases of tetanus *(C. tetani)*, gas gangrene *(C. welchii)* and botulism *(C. botulinum)*.

Other spore-forming organisms which live naturally in the environment and which may be associated with food poisoning and occasionally invasive infection are the *Bacillus* species.

Washing with soap or detergent and water will remove or kill 80 per cent of organisms on a surface.

Careful use of aseptic techniques and appropriate disinfectants and antiseptics as well as an understanding of how microorganisms spread will ensure that wounded patients are not exposed unnecessarily to microorganisms. Establishment of a disinfectant policy with regular review will ensure the cost-effective use of these products.

The subject of disinfection is dealt with in more detail in Chapter 28.

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CHAPTER TWENTY-SEVEN

FOREIGN BODIES IN WOUNDS

Gael Phillips

In most real-life and practical situations involving open trauma, wounds are contaminated with foreign material. Foreign bodies – such as dirt, clothing, metal and wood – increase the incidence of wound infection; and complex reactions occur involving the cells of the wounded host and the various types of foreign bodies which are introduced into traumatised tissue.

Wounds containing foreign bodies may be accidental, suicidal, homicidal or iatrogenic. Iatrogenic wounds are those which are medically inflicted during the course of surgical operations or procedures. A wound may be further defined as "damage to the tissues of the body by mechanical forces" and several types are recognised. These include abrasions, bruises or contusions, lacerations and incised wounds. Incised wounds include cuts, stab wounds and intentional (iatrogenic) surgical wounds. Unless there is a break in the surface of the skin or mucous membrane, foreign bodies will not gain access to the tissues, so foreign bodies will only be a practical problem in abrasions, lacerations and incised wounds. They are not a feature of uncomplicated bruises or contusions. A wound containing a foreign body may be very small, such as a small wound on the surface of the conjunctiva of an eye harbouring a penetrating foreign body. Foreign bodies may be small or large and they vary widely in their composition.

The commonest type of wound seen by first aiders and paramedics is an accidental wound, but it should be borne in mind that not all suicides succeed and unsuccessful homicides may also result in wounds which present for treatment, contaminated with foreign bodies. Iatrogenic foreign bodies in wounds are those introduced during surgical treatment, such as suture materials, surgical prostheses and implants.

It is of fundamental importance that the presence of one or more foreign bodies in a wound should be detected, although the management of the foreign body depends on the level of treatment and the type of foreign body. A stake impaling a victim's abdomen in an agricultural accident must not be removed by a first aider, but left to the surgeon in the operating theatre. Eventually, all foreign bodies must be removed so that wound healing is facilitated. Foreign bodies in a wound may introduce bacteria, resulting in infection, but the presence of a foreign body will also complicate wound healing in other ways.

NATURE OF FOREIGN BODIES

The types of foreign bodies which may be present in a wound vary widely. Large foreign bodies, such as impaling stakes, spears or projectiles, may cause death very quickly, so they do not present for first aid; exceptions do occur. Bizarre cases of accidental, suicidal and homicidal injury in which the individual has survived the presence of one of more large penetrating foreign bodies are described in the literature. Such an episode was described in which a patient was struck by a bread knife which entered her face immediately above the right eye, passed upwards and medially through the ethmoid bulla on the right, crossed the midline at the level of the tuberculum sellae and came to rest with the

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tip of the knife close to the left internal carotid artery with back of the blade in contact with the right carotid artery. The knife was carefully removed by the treating neurosurgeons and the patient made an uneventful recovery. Patients with large penetrating foreign bodies present special difficulties to first aid personnel and they need to be treated individually.

Some examples of foreign body types are:

Metal, ferrous and non-ferrous
Wood and other vegetable material
Glass
Plastic
Gravel, soil and clay
Animal stinging apparatus (bee stings, nematocysts) and teeth
Fragments of clothing, fabric
Firearm projectiles
Weapons or fragments of weapons
Suture material, surgical implants or prostheses
Soot, ash

The classification of foreign bodies may be likened to the childhood guessing game "Animal, Vegetable or Mineral?", with most foreign bodies falling into the Vegetable or Mineral categories.

Common small foreign bodies are those seen following accidents in the home or garden in which thorns or wood splinters are introduced into the skin (Figure 27.1). These are always contaminated with bacteria, and severe infections such as tetanus may result from quite a simple injury such as a rose thorn penetrating the skin in those who are not immunised against *Clostridium tetani*. Other infections associated with retained foreign bodies include cellulitis, osteomyelitis, and gas gangrene (caused by *Clostridium perfringens* – sometimes known as *Clostridium welchii*) and fungal infections, sometimes with mycetoma formation. Another common foreign body, typical of the amateur fisherman, is a fish hook in a finger or hand. Fish hooks are easily detected, but some other foreign bodies may be occult. Following a fall, wounds may become contaminated with fragments of soil or glass, depending on the circumstances of the accident. Gravel or small fragments of rocks may contaminate wounds.

Paint fragments may be found in wounds on pedestrian victims of vehicular accidents, which may provide useful evidence to identify a particular vehicle when legal proceedings are undertaken. Identification of the nature of some foreign bodies is not only useful for medical management but also sometimes necessary for medico-legal or forensic purposes.

Foreign material is found in wounds resulting from firearm injuries. These include projectiles and elements of the gunpowder or propellant which will be present in varying amounts, depending on the distance of the victim from the firearm. It is important that the clothing

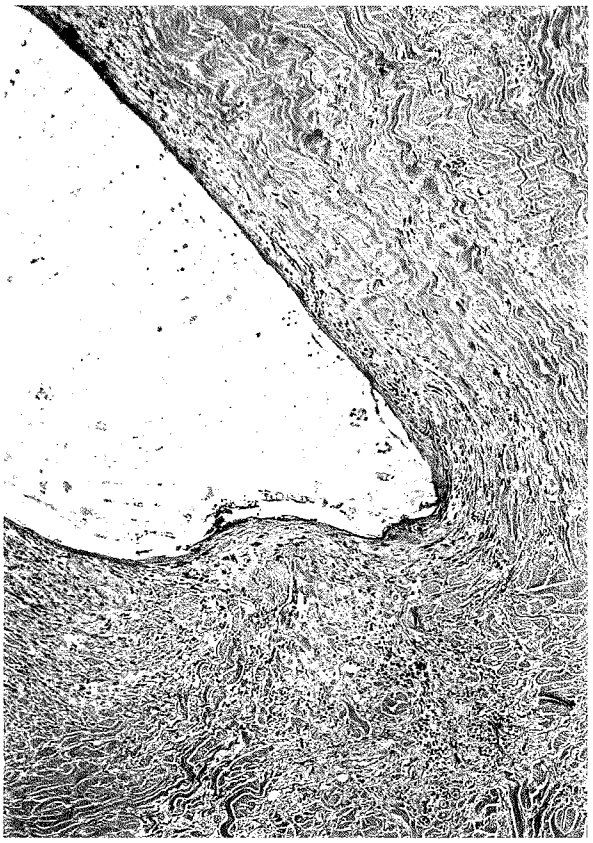


Figure 27.1 *Microphotograph of a plant thorn in the dermis showing a mild chronic inflammatory reaction in the surrounding connective tissue. There are a few attenuated foreign-body-type giant cells surrounding the thorn. The cellulose walls of the plant cells are identifiable. H & E magnification × 294.*

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be retained for examination in such cases, and the clothing should be cut off through areas not contaminated with gunpowder residues or damaged by the projectile so that forensic scientists may study the clothing in relation to the wound to establish the circumstances of the wounding. The clothing and any residues removed from the victim should be sealed in plastic or specimen bottles and labelled correctly so that a chain of evidence can be preserved. This is necessary so that evidence collected in cases of unlawful wounding will be acceptable in a court of law. The size, location, number and nature of wounds need to be carefully documented, especially if there is any suspicion of legal proceedings following the episode. This can be done by the first aider at the site, and is the duty of the treating surgeon or, in the case of fatal wounds, the forensic pathologist. Photography of wounds will also be necessary in these circumstances, to be tendered as evidence in court proceedings.

ANIMAL FOREIGN BODIES

Sometimes, foreign bodies are introduced into the skin by the bites of animals, or fragments of animal parts may be left behind in a wound, such as the sting of a bee or nematocysts (stinging capsules) from toxic jellyfish. These foreign bodies further complicate the first aid scene because of their inherent toxicity to the individual. Anaphylaxis may occur following a bee sting which may be fatal in those individuals with hypersensitivity. The toxic box jellyfish *Chironex fleckeri,* which lives in the warm coastal waters off northern Australia and the western Indo-Pacific area, has been responsible for many deaths. This is the most toxic jellyfish known to science, but first aid treatment is effective in helping to save lives. (See Chapter 25.)

In sutured surgical (iatrogenic) or accidental wounds, the presence of excessive suture material may delay wound healing, but even small fragments of suture material will often be accompanied by a localised foreign body reaction. Some types of synthetic suture material are relatively inert but the traditional cat-gut type of suture material invariably evokes a foreign body giant cell response.

DETECTION OF FOREIGN BODIES

How are these foreign bodies detected?

Making a diagnosis of a retained foreign body is often difficult and up to 38 per cent of foreign bodies are not detected on initial examination. As the first step in assessing any clinical problem, a history should be obtained, if possible. If the victim is unconscious, witnesses may provide clues as to the likely events leading up to the wounding. The history may suggest what foreign bodies may be present. A history of the victim's having used woodworking tools, for example, raises the possibility of an intra-ocular foreign body if the patient is presenting with symptoms related to the eye.

The examining doctor will probe traumatic wounds to detect the presence of foreign bodies. It is usually the case that a probe will reveal a characteristic grating sound as soon as a foreign body is located. Sometimes, very finely divided foreign particles, such as soil, will be removed as part of a wound cleansing and debridement, but other foreign bodies will need to be removed individually using forceps.

Soft tissue foreign bodies may be detected using various imaging techniques. These include plain radiography or X-ray, computerised tomography (CAT scanning) ultrasonography and xerography. All these techniques will reveal glass foreign bodies, but wooden and plastic foreign bodies are more difficult to demonstrate. *In vitro* tests reveal that ultrasonography is the most reliable test to detect the presence of a foreign body. Using this technique, wooden and plastic foreign bodies may be imaged, in addition to glass foreign bodies.

If there is any suggestion of the presence of a foreign body in a wound, radiology is mandatory. This is particularly so in cases involving possible penetrating wounds of the orbit, where small metallic particles may have penetrated the eyeball, which will lead to severe complications and even loss of vision if undetected. When the metallic foreign body has been removed, the

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pathologist has a role in determining its nature. If the intra-ocular foreign body is of iron or steel, further treatment of the patient will be necessary to prevent continuing intra-ocular inflammation. A copper foreign body, on the other hand, is relatively inert and the prognosis for the eye is much better, although progressive loss of vision from continuing intra-ocular inflammation can also occur with copper foreign bodies in the eye. Small foreign bodies are common on the conjunctival and corneal surfaces of the eye and these are frequently metallic. Because of their location, they cause great irritation to the eye and may result in infections gaining access to the conjunctiva and cornea. It is for this reason that the first aid treatment of all ocular foreign bodies includes covering the eye with an eye-pad. This is also necessary to rest the eye from unnecessary accommodation adjustments.

The nature of an intra-ocular foreign body can be determined using the electron microscope for elemental analysis. Scanning electron microscopy at the same time will demonstrate the shape of a foreign body, such as the copper wire intra-ocular foreign body illustrated in Figure 27.2.

Nuclear magnetic resonance imaging is now also being used to detect foreign bodies, although the presence of possible iron-containing foreign bodies may be dangerous.

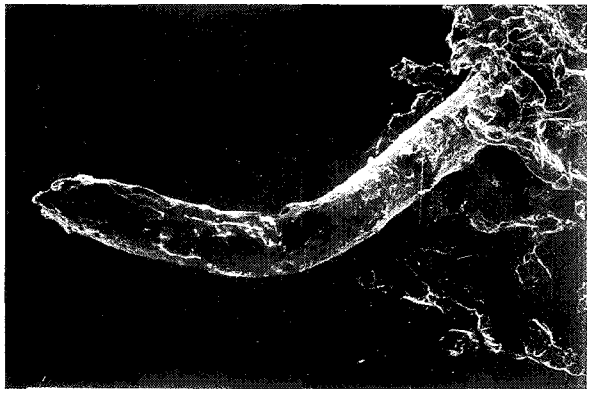


Figure 27.2 *Scanning electron micrograph of a small piece of copper wire which was removed from the eyeball of a male patient who had been trimming a garden border with a "whipper snipper". (The irregular material in the upper right hand corner is colloidal graphite mounting medium.) Elemental analysis demonstrated that the wire was pure copper.*



Figure 27.3 *Unidentified degenerate foreign body of organic origin in the dermis with a surrounding acute inflammatory reaction and abscess formation. H & E magnification × 196.*

WOUND INFECTIONS

The contamination of an accidental wound with foreign material is invariably accompanied by contamination of the wound by bacteria and/or fungal organisms, and wounds containing foreign bodies are particularly prone to develop and maintain wound infections. *Staphylococcus aureus* infections and Gram-negative bacteria are common organisms causing infections in wounds containing foreign bodies. An abscess will usually result if a contaminated foreign body is left behind in a wound (Figure 27.3). Fungal organisms introduced with a foreign body may lead to a fungal infection in the dermis or deeper tissue and a

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Figure 27.4 *Healing surgical wound of approximately 10 days duration showing a foreign-body-type giant cell reaction to suture material and naked hair shafts in the dermis. There is also (arrow) a fragment of implanted stratified squamous epithelium from the epidermis. H & E magnification × 98.*

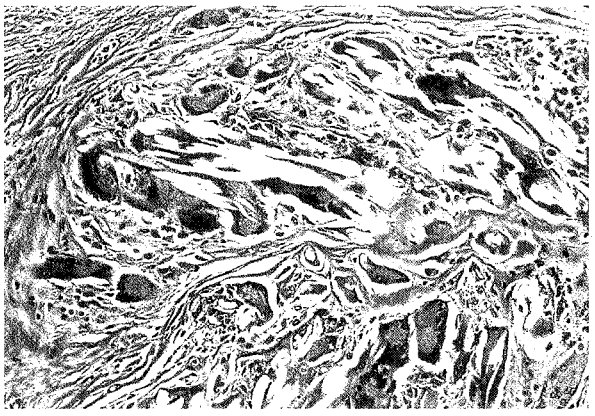


Figure 27.5 *Florid foreign-body-type giant cell reaction to suture material in deep soft tissue identified several years following surgery. H & E magnification × 294.*

mycetoma, or tumour-like mass lesion caused by a proliferation of fungal organisms in association with inflammatory cells, may result if left untreated.

FOREIGN BODY REACTIONS

The presence of a foreign body usually leads to a foreign body reaction which is a response by the host tissue to the foreign body. This is seen as the formation of multinucleate cells from the macrophage series of cells, which surround and apparently attempt to engulf the foreign body. Some foreign bodies are relatively inert, and do not produce this reaction. These are usually iatrogenic, surgically implanted foreign bodies which are designed to elicit a minimal host response. The foreign body giant cell reaction is thought to impair wound healing.

The presence of a foreign body may lead to failure of wound healing and the formation of a fistula, which is really a tunnel lined either by inflamed granulation tissue or epithelium, usually opening to the surface of the skin, and which may be associated with ulceration on the surface. Foreign bodies may be discharged spontaneously from the wound via a fistulous tract if they are not removed during the initial treatment of the wound.

EPIDERMAL IMPLANTATION CYSTS

Another complication associated with the introduction of a foreign body into the wound is the carriage of small fragments of epidermis into the wound which may form implantation epidermal cysts within the healing wound (Figures 27.6 and 27.7). These are seen as cystic structures, frequently in the dermis or less usually in the subcutaneous tissue, lined by keratinising stratified squamous epithelium. Abscess formation may also be associated with such cysts. Expansion and rupture of implantation epidermal cysts leads to further foreign body reactions due to the presence of naked keratin squames or flakes of normally superficially located keratinised cells in the dermis. Sometimes such

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implantation epidermal cysts may form in the dermis or subcutaneous tissue following intradermal or subcutaneous injections for therapeutic purposes. Very rarely, epidermis may be implanted into the cerebrospinal fluid space during lumbar puncture, which may lead to severe complications. A chemical type of chronic meningitis develops due to release of keratinised cells into the cerebrospinal fluid resulting from a proliferation of benign squamous cells producing space-occupying lesions around the spinal cord.

MECHANISM OF FORMATION OF FOREIGN BODY GIANT CELL REACTIONS AND FOREIGN BODY GRANULOMATA

The foreign body reaction is a response to the presence of foreign material in an attempt to protect the host against the "non-self" material. In some instances material derived from the host will be perceived as "foreign" when it is in an abnormal location, such as keratinising squamous cells released into the dermis from a benign epidermal cyst. In other instances, the material eliciting the foreign body reaction is truly "non-self" or foreign, such as splinters of glass or particles of talc from a surgeon's glove.

Parasites such as threadworms *(Enterobius vermicularis)* will also elicit a foreign body reaction by giant cell reaction.

A granuloma may be defined as "a chronic inflammatory reaction characterised by a localised



Figure 27.6 *Microphotograph of an implantation epidermal cyst derived from a fragment of epidermis carried into the dermis along with a foreign body of botanical origin. H & E magnification × 98.*

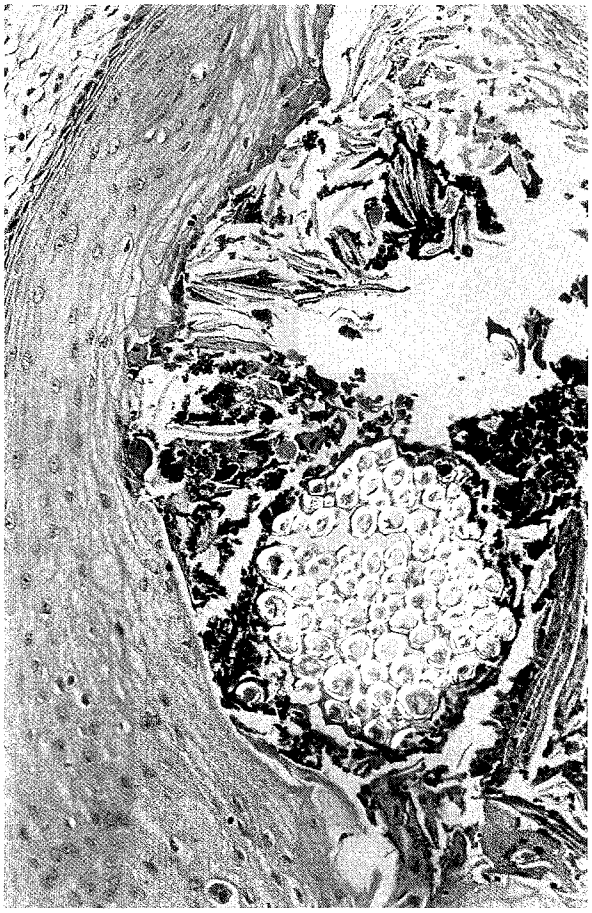


Figure 27.7 *Microphotograph of a detail of an implantation epidermal cyst, highlighting the cellulose in a fragment of plant material. The foreign body is surrounded by a cystic structure composed of stratified squamous epithelium, also containing desquamated keratin squames, acute inflammatory cells and necrotic cell debris. H & E magnification × 294.*

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collection of macrophages". The basic process of the formation of a foreign body reaction or foreign body granuloma is the accumulation of activated mononuclear cells in the tissue surrounding the foreign material. They then develop into epithelioid histiocytes and multinucleate polykaryons (multinucleate giant cells) which attempt to engulf the foreign material by a process of phagocytosis. This is followed by the events leading to processes such as chemotaxis and the release of cytokines to attract the mononuclear cells, which originate in the bone marrow, to the site of the foreign material. In order that mononuclear cells should accumulate at the site of the foreign body there is a differential chemotaxis which favours those cells as compared with neutrophil polymorphs. Macrophages present locally may also divide and proliferate to take part in the inflammatory reaction. If phagocytosis is incomplete or the foreign material cannot be "digested" by the cell, the macrophages enlarge to form so-called "epithelioid" histiocytes which are the characteristic cell of granulomata. (Histiocytes and macrophages are really the same type of cell, masquerading under two different names.) The macrophages may also fuse to form the multinucleate giant cells which are the cells which predominate in a foreign-body-type giant cell reaction. Nuclei in giant cells may also undergo mitosis to regenerate the giant cells. Although multinucleate giant cells are seen around exogenous foreign material, or "real" foreign bodies, they are also seen reacting to endogenous material which is perceived by the cells as being foreign, such as urate crystals in gouty tophi and cholesterol crystals in a variety of locations such as within atheromatous plaques in arteries and in areas of tissue breakdown.

Lymphokines are chemical mediators released by lymphocytes into the tissue which act on other cells to produce an effect such as a chemotactic effect or activation of macrophages. The crucial step in the formation of a granuloma is the maturation of the mononuclear cells, which have been attracted to the site, into macrophages, and their proliferation into aggregates which characterise granulomata. Lymphokines play a prominent role in the development of granulomata. A foreign body granuloma is regarded as being a non-immune granuloma, in contrast to the granulomata which develop in infections such as tuberculosis or leprosy, which are immune or hypersensitivity granulomata.

RARER COMPLICATIONS OF RETAINED FOREIGN BODIES

Retained foreign bodies may also lead to injury to peripheral nerves and damage to blood vessels which may lead to the formation of false arterial aneurysms. An aneurysm is a localised ballooning of the wall of an artery and may be classified into true and false aneurysms. In a true aneurysm the localised ballooning or dilation of the vessel wall includes all layers of the wall of the vessel even though the internal elastic lamina may be partly deficient. The wall of a false arterial aneurysm is formed by fibrous scar tissue bridging a defect in the vessel wall and forming a weak area which dilates. Another complication is synovitis which may occur if foreign bodies gain access to a synovial joint.

FIRST AID MANAGEMENT OF WOUNDS CONTAINING LARGER FOREIGN BODIES

The first aid management of wounds which are complicated by larger penetrating foreign bodies is to control bleeding by the application of pressure on the surrounding tissue, but not on the foreign body itself. Removal of penetrating foreign bodies should not be attempted as this may lead to damage to tissue in the vicinity, or to deeper tissue, and result in bleeding. Padding should be placed around the foreign body and the area bandaged. A ring pad may be used with bandages over and around the wound. Small loose foreign bodies such as gravel lying on the surface of a wound are lightly brushed off before the wound is bandaged and the patient transported to definitive medical care.

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CONCLUSION

The presence of foreign bodies, therefore, poses several problems for the management of wounds. There is an increased risk of wound infection, delayed healing and complications related to the presence of epidermis implanted into the wound. Foreign body granulomata may occur and these can sometimes lead to localised nodules in the skin or elsewhere. There may be damage to nerves and vessels with retained foreign bodies. These complications are in addition to the problems posed by large foreign bodies which may not necessarily result in immediate death but which may require first aid prior to evacuation to definitive medical care.

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CHAPTER TWENTY-EIGHT

CROSS-INFECTION AND ANTISEPTICS

Geoffrey Newman-Martin, Joan Faoagali and John Pearn

All who work in the pre-hospital setting must protect their patients from infections. All who care for casualties and all who train students in the techniques of first aid must protect themselves from infection.

An understanding of cross-infection, and of the way in which it can be prevented, is one of the fundamentals of all first aid work. The daily business of the first aider and the paramedic is to keep wounds clean, to protect them from infection and at the same time not become infected from the casualty (Figure 28.1).

The first aider also must be familiar with the principles and practice of equipment cleaning and sterilisation, and of the disinfection of training aids, especially of manikins used in CPR teaching.

TERMINOLOGY

The terms "antiseptic" and "disinfectant" are here used interchangeably. Any reference to trade names is purely for illustrative purposes and does not constitute a recommendation.

An antiseptic or disinfectant is a chemical agent which destroys microorganisms or inhibits their growth on living tissues (Figures 28.2A and B). Different antiseptics and disinfectants do this to different degrees. Disinfection is a process which kills or inactivates organisms but not necessarily spores. (Spores are metabolically inert forms of bacteria resistant to stress such as heating, drying or mild disinfection.) Disinfection does not necessarily kill all microorganisms, but reduces them to a level which is not harmful to health. The term is applicable to agents used to treat inanimate objects and may also be applied to agents used to treat the skin and other body membranes and cavities.

Cleaning involves the removal of organisms and soil. Washing with soap or detergent and water will remove most of the organisms and protein or other organic material which may provide a source of nutrients to organisms, as well as protecting them against drying and the killing power of disinfectants.

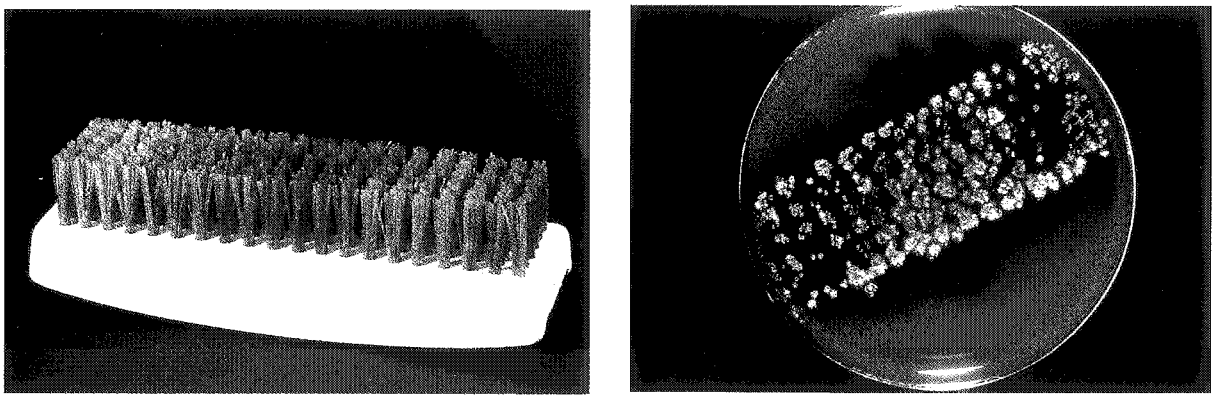
The proportion of microbial flora killed by a disinfectant depends on the intrinsic power of the disinfectant and the innate resistance of the microorganism. Chemical disinfectants may be ranked according to their biocidal capabilities as high-, intermediate- or low-level. High-level disinfectants act against all microbial forms and may even sterilise if the contact time is long enough. Intermediate-level disinfectants kill most microorganisms except bacterial spores and sometimes small, nonlipid viruses (eg enteroviruses). Low-level disinfectants have an even lower level of effectiveness.

It should not be overlooked that washing with soap or detergent and water will remove or kill 80 per cent of organisms on a surface. It is recommended that the use of disinfectants should be minimised as they are costly, often environmentally unfriendly, possibly toxic and generally unnecessary.

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Figure 28.1 *Ambulance officers care for motor-cycle and pillion riders, victims of a two-vehicle collision in Darwin. Note the disposable latex gloves. (Photograph courtesy of St John Ambulance Australia (Northern Territory).)*



A B

Figures 28.2 A and B *The task confronting a disinfectant.* **A** *a nail brush on a hand basin, and* **B** *culture of an imprint of the nail brush bristles on a blood agar plate (24 hours at 35°C). Heavy growth of multiple organisms including* Pseudomonas *species,* Bacillus *species and* Staphylococcus *species.*

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Table 28.1 Effective disinfectants

Generic disinfectant	Advantage	Disadvantage	Presentation
Alcohols			
Methyl alcohol Ethyl alcohol Isopropyl alcohol	Rapid action. Broad spectrum. No residue.	Expensive. Evaporates. Poor sporocide and virucide. Can dissolve some glues holding lenses, precipitates protein and may clog fine channels. "Burns" mucous membranes. No residual action.	30–70 per cent concentration required for action. Good skin disinfecctant (with detergent/disinfectant). Good for wiping clean surfaces.
Chlorhexidine	Rapid action. Cumulative effect. Broad spectrum. Good detergent.	Some toxicity on skin. Expensive. Some products clog dispensers. Limited activitiy against viruses and acid-fast bacilli. No activity against spores.	2–4 per cent concentrations. Preoperative skin preparation. Handwash. Dry handwash (with alcohol).
Halogens			
Hypochlorite	Cheap. Effective. Virucidal agent.	Rapidly inactivated by organic material. Strong fumes. No detergent action.	Various concentrations used in different circumstances.
Iodine			
Iodophor	Broad spectrum bactericidal agent.	Expensive. Toxic or allergic reactions. Occasional reports of contamination. NOT usually sterile.	Preoperative skin preparations. Handwash. Surgical scrub.
Irgasan	Good anti-staph activity. Low toxicity.	Expensive. Minimal Gram-negative action.	Handwash – liquid – bar soap
Phenols	Broad spectrum. Resistant to inactivation.	Expensive. Poisonous. Unpleasant smell.	Synthetic mixtures used for environmental cleaning.
Aldehydes			
Formaldehyde Glutaraldehyde	Broad spectrum.	Strong irritant vapour. Expensive. Toxic to mucous membranes and tissues.	Tissue fixative.

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Table 28.2 Summary of activity of disinfectants

Chemical disinfectants	Optimum activity against microorganisms						Inactivation by	
	Gram-positive bacteria	Gram-negative bacteria	T.B.	Spores	Fungi	Viruses	Hard water	Organic contamination
Alcohols								
Isopropyl alcohol 70%	Good	Good	Good	None	Good	Fair	Slight	Slight
Aldehydes								
Glutaraldehyde 2%	Good	Good	Fair	Good (Slow)	Good	Good/Fair	?	Slight
Halogens								
(a) sodium hypochlorite	Good	Good	Good	Slight/Fair	Good	Good	Slight	Serious
(b) povidone-iodine	Good	Good	Good		Good	Good	Slight	Serious
Diguanides								
Chlorhexidine	Good	Fair	Slight	None	Good	Limited	Serious	Serious
Phenolic								
Medol 1%	Good	Good	Fair	Slight	Good	Minimal	Slight	Slight

The two tables above give basic information about a range of disinfectants. Table 28.1 lists some common generic disinfectants available for use in a health-care setting and summarises their advantages, disadvantages and presentation. Table 28.2 summarises the activity of various disinfectant groups.

CRITERIA FOR THE USE OF DISINFECTANTS

Disinfectants are general protoplasmic poisons. The criteria for choosing an ideal disinfectant are set out below.

1 Broad spectrum – widest possible antimicrobial spectrum.
2 Fast acting – rapidly lethal action on all vegetative forms and spores of bacteria, fungi, protozoa and viruses.
3 Not affected by physical factors – active in the presence of organic matter such as blood, sputum or faeces.
4 Compatible with soaps, detergents and other chemicals.
5 Non-toxic to humans and animals.
6 Surface compatibility – does not corrode instruments and other metallic surfaces. Does not disintegrate cloth, rubber, plastics or other materials (Figure 28.3).
7 Leaves a residue on treated surfaces.
8 Easy to use.
9 Odourless.
10 Economical.

In particular, since we are looking at products which may come in contact with the skin or mucous membranes, it is important to know the characteristics of an ideal skin disinfectant.

1 Freedom from adverse skin reactions, including removal of lipids, irritation, or toxic or allergic dermatitis.
2 Absence of dermal absorption resulting in systemic toxicity.
3 Ability to penetrate crevices, skin folds or other skin structures.
4 Rapid broad-spectrum action against Gram-positive and Gram-negative bacteria; activity against viruses or bacterial spores may also be desired.
5 Retention of activity in the presence of skin secretions, blood, mucus or faeces.

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6 Residual or cumulative action due to retention of the active agent on the skin.

All of these issues need to be assessed prior to selection, to avoid the costly exercise of replacing an unsuitable product or ending up with a random selection of personal choice products. Examples of important disinfectants follow.

TYPES OF DISINFECTANT

Alcohols

Alcohols exert their microbicidal action by means of protein denaturation, but require to be diluted with water in order to do so.

Ethanol 70–80 per cent v/v and isopropanol 70–90 per cent v/v are two commonly-used items which rapidly kill many organisms. They have no efficacy against bacterial spores. They do not exert any residual effect.

Chlorhexidine

Chlorhexidine is active against Gram-positive bacteria and to a slightly lesser extent against Gram-negative bacteria. It may rapidly inactivate some viruses.

When used as handwash, chlorhexidine exhibits low skin irritation and low skin sensitisation, provided the hands are rinsed after application. Dilute solutions of chlorhexidine (0.05 per cent or less) may sometimes be contaminated, usually with Gram-negative bacilli, such as *Pseudomonas aeruginosa, Klebsiella* species and Enterobacteriaceae.

Lipophilic viruses (enveloped viruses) such as herpes viruses may be susceptible to chlorhexidine. Hydrophilic viruses are non-enveloped; an example is the Norwalk agent virus which causes outbreaks of diarrhoea and is resistant to the effects of chlorhexidine.

Soaps and anionic detergents will inactivate chlorhexidine.

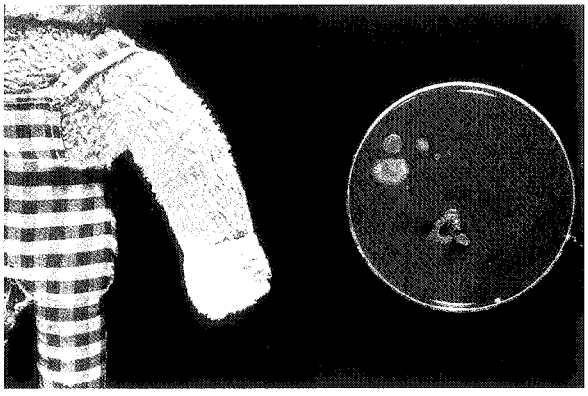


Figure 28.3 *A potential source of infection. The arm of a teddy bear and, beside it, a culture plate showing growth of staphylococcal and* Bacillus *species from the arm.*

Irgasan

Irgasan is a good antistaphylococcal antiseptic, with limited activity against Gram-negative bacteria.

Povidone-iodine

Povidone-iodine exerts its antimicrobial action (on Gram-positive and Gram-negative bacteria, bacterial spores, lipophilic and hydrophilic viruses and yeasts) by release of iodine, which is able to penetrate the cell wall of microorganisms quickly; its lethal effects are the result of disruption of protein and nucleic acid structure and synthesis inside the microorganisms themselves. Like other halogenated disinfectants, it is an oxidising agent, which may partially explain its antimicrobial action, together with direct halogenation of cell contents. It has no cumulative action from regular use.

Povidone-iodine is less irritant to the skin, and less likely to cause hypersensitivity reactions, than iodine. Adverse reactions associated with manikin disinfection appear to be unlikely.

Betadine povidone-iodine at concentrations of greater than or equal to 5 per cent completely inactivates HIV *in vitro*; inactivation is immediate.

Aldehydes

Formaldehyde and glutaraldehyde are highly effective against Gram-negative and Gram-positive bacteria, moderately effective against acid-fast bacteria and hydrophilic viruses, effective against lipophilic viruses, and effective at a slow rate against bacterial spores.

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Glutaraldehyde is a high-level disinfectant which is not corrosive to equipment. Alkaline glutaraldehyde can be used for inactivating hepatitis B virus and HIV in blood and serum.

Glutaraldehyde causes contact dermatitis and severe irritation of the eye, skin, nose and throat, even at very low levels, and its use requires the use of protective clothing, using Worksafe Australia guidelines.

Glutaraldehyde is the only chemical recommended by Australian Standard 4187(6) for high-level disinfection of reusable medical equipment items that cannot be heat sterilised. The Australian National Council on AIDS recommends that all health-care workers should know the risks involved with using glutaraldehyde and how to use this chemical safely and correctly.

On the grounds of toxicity, the aldehydes must be considered unsuitable for use in the disinfection of manikins for CPR training.

Chlorine disinfectants

Free chlorine is a fast-acting high-level disinfectant. 100 ppm of free chlorine will kill 10^6 to 10^7 *Staphylococcus aureus, Salmonella choleraesuis* and *Pseudomonas aeruginosa* organisms in less than 10 minutes.

Chlorine disinfectants include hypochlorites (eg sodium hypochlorite, NaOCl), chlorinated isocyanurates (eg sodium dichloroisocyanurate, $C_3N_3O_3Cl_2Na$), trichloro-s-triazinetrione, and sodium oxychlorosene.

Sodium dichloroisocyanurate is a powder which exerts its action after being dissolved in water. One tablet of Puritabs, containing 17 mg of sodium dichloroisocyanurate, dissolved in 1 litre of water liberates 10 ppm of available chlorine. This is used to produce water fit for drinking. It will kill viruses, bacteria and encysted protozoa at temperatures around 20°C at neutral pH after a contact time of 30 minutes; organic matter will reduce available chlorine concentrations. Lower temperatures (below 10°C) and increasing pH both reduce the cysticidal activity of halogens.

Hypochlorite solutions are usually prepared by chlorination of strong sodium hydroxide solution until the alkalinity is neutralised, and then diluted for use by consumers. The solution is unstable, particularly if it is acidic, so manufacturers make a product with excess alkalinity. Hypochlorite solutions which are strongly alkaline will be corrosive to skin, eyes, mucous membrane and clothing – which reduces their usefulness in disinfection of manikins. It appears also that, if they are too alkaline, they may be much reduced in their antimicrobial effectiveness. Halogen-based disinfectants are most active in the pH range 6–8 (ie approximately neutral), and sporicidal action is greatly accelerated by buffering the solution to pH 7.6; hypochlorite anions, which predominate in alkaline solutions, are virtually devoid of antimicrobial activity. Sodium hypochlorite can be buffered to pH 9.0 but at this pH shows significant loss of bactericidal activity compared with sodium hypochlorite at pH 7.0, particularly in the presence of organic material. Quality control in manufacturing is thus crucial.

Chlorine disinfectants exert their action by the release of non-ionised hypochlorous acid, although the hypochlorite ion OCl^- may be a contributory factor.

Hypochlorite solutions used as germicides contain from 0.5 per cent to 2 per cent active ingredient, although concentrations of chlorine disinfectants are often expressed as available chlorine.

The Australian National Council on AIDS Bulletin No. 16 (1994) recommends the use of a hypochlorite solution containing 10 000 ppm available chlorine (defined in their report as a 1 in 10 dilution of household bleach) for treatment of grossly contaminated surfaces.

Chlorinated disinfectants react with all types of organic matter, and should be used with a sufficiently high chlorine content to leave an effective chlorine residue after this occurs.

Halogenated disinfectants, such as chlorinated disinfectants, can be used against Gram-negative bacteria and bacterial spores (if used in high concentrations), and 0.5 per cent sodium hypochlorite will inactivate hepatitis B virus. Chlorine disinfectants can inactivate hepatitis B virus and HIV in blood, and indeed hypochlorite bleach solutions have been promoted as the agent of choice for disinfection of needles for personal use by intravenous drug users.

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In view of the range of quality available, it is important to use bleaches of known concentration.

Chlorine compounds have relatively low residual toxicity, but are harmful to the eyes and skin. There are other disadvantages in using chlorine disinfectants, including the corrosion of metal and potential damage to clothing and soft furnishings.

Peroxides

Peroxides are oxidising agents with limited antibacterial activity. The antibacterial potency of certain peroxides depends primarily upon the reactivity of free hydroxyl (OH) radicals; different microorganisms vary considerably in their susceptibility to oxidising agents.

The disinfectant effect of hydrogen peroxide lasts only as long as the oxygen is being released, and is of short duration; in addition the antimicrobial effect of the liberated oxygen is reduced in the presence of organic matter.

Hydrogen peroxide is used as an antiseptic in 3 per cent solution (eg in contact lens sterilising solutions). Dilute solutions are prepared by diluting concentrated solutions (up to 90 per cent) which are stable.

Hydrogen peroxide is active against most microorganisms but enteric viruses and bacterial spores require a high concentration for lethal action. Solutions containing 3 per cent or more hydrogen peroxide are irritant to tissues, the degree of irritation being more severe with increasing concentration, becoming corrosive at higher concentrations. Activity against HIV and hepatitis B is unproven.

Phenolics

Phenolic compounds are low or intermediate-level disinfectants. This group includes compounds, such as *p*-chloro-*m*-xylenol, which are soluble in water when surfactants or emulsifiers are added. The correct balance of active and surfactant, and the type of surfactant, are important to enable release of the phenolics into the aqueous phase; they are most effective at neutral or slightly acidic pH, when the molecule is non-ionised. The concentration of active is critical to efficacy; solutions which are too dilute may be ineffective.

Some phenolic disinfectants (but not chloroxylenols) may be used against some Gram-negative bacteria. Some phenolic disinfectants can be used against acid-fast bacilli *(Myobacterium tuberculosis)*. Some others are effective against lipophilic viruses, but they are less effective against acid-fast bacteria and Gram-negative bacilli. Phenolics are toxic if ingested and can cause chemical burns. Skin sensitisation has been reported. Their taste and odour are objectionable (although odour may be masked with pine oil). They can decontaminate biological material other than blood.

Dilute solutions of synthetic phenolic disinfectants may sometimes be contaminated, usually with Gram-negative bacilli, such as *Pseudomonas aeruginosa, Klebsiella* species and Enterobacteriaceae.

It is critical that the correct pH and concentration be used, so that the limited spectrum of action is optimised. These disinfectants are not suitable for use with manikins used for CPR training.

Medol is a mixture of synthetic phenols with a broad antibacterial spectrum but poor activity against viruses.

Quaternary ammonium compounds

Quaternary ammonium compounds (QACs) are low-level disinfectants. They are cationic surfactants, usually marketed as 10–15 per cent solutions (although household disinfectants can contain as little as 0.5 per cent QACs). They comprise molecules in which one hydrogen atom of an ammonium ion (NH_4^+) has been replaced by long hydrocarbon chains. They include such compounds as benzalkonium chloride. Their mode of microbicidal action appears to involve the characteristics of surface tension reduction and positive charge, thus causing uptake by microorganisms. They are most efficacious in alkaline solution, that is, in the pH range 9–10.

Gram-positive bacteria are highly susceptible, while Gram-negative bacteria are moderately susceptible to the effects of QACs. Lipophilic viruses are susceptible. Acid-fast bacteria, bacterial spores and hydrophilic viruses are, however, resistant. Quaternary ammonium compounds inactivate HIV. Dilute solutions of

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quaternary ammonium compounds may sometimes be contaminated, usually with Gram-negative bacilli, such as *P. aeruginosa, Klebsiella* species and Enterobacteriaceae.

They are inactivated by various proteins, including those in blood, and by anionic soaps and nonionic surfactants. They are also inactivated by adsorption onto cotton and some plastics. They exhibit relatively low acute oral toxicity, although ingestion of a concentrated solution may cause a variety of toxic symptoms and can be fatal. Skin sensitisation reactions sometimes occur.

QACs are not recommended for use for manikin cleaning.

DISINFECTION OF EQUIPMENT

First aid equipment should be: heat resistant and able to be autoclaved and sterilised – such items should be used in preference if available; or capable of chemical sterilisation by hypochlorite solution (such as manikins); or disposable.

Wet sterilisation or chemical disinfection requires special care. The antiseptic solution may become contaminated, diluted, soiled or inactivated, so that in the worst case, instead of cleaning the item which one is attempting to sterilise, one may find that the item is coated with a culture of resistant, harmful microorganisms. Wet sterilisation is expensive and staff may be affected by toxic fumes given off from the product. Equipment to be wet sterilised must be scrupulously clean *before* it is placed in the disinfectant, otherwise the disinfectant may not be able to get to the surface it is supposed to clean. An impermeable surface coagulum may be formed which actually protects the microorganisms in its depths.

Equipment must be stored dry and protected from dust after cleaning. Dilute disinfectants often harbour water-loving microorganisms, such as *Pseudomonas* species, which can contaminate stored equipment. Microorganisms do not survive drying and a dust-free environment prevents airborne transmission of microorganisms.

Careful use of aseptic techniques and appropriate disinfectants and antiseptics as well as an understanding of how microorganisms spread will ensure that wounded patients are not exposed unnecessarily to micro-organisms. Establishment of a disinfectant policy with regular review will ensure the cost-effective use of these products.

A disinfectant policy which sets out clearly which disinfectant should be used and where it should be used will ensure the appropriate and cost-effective use of

Table 28.3 Devices, processes and germicidal products for use in equipment sterilisation

Device classification	Process classification
Critical Any agent which enters sterile tissue or vascular systems (eg implants, scalpels, needles, other surgical instruments).	*Sterilisation* Sporocidal chemical: prolonged contact.
Semi-critical Equipment which touches mucous membranes (eg flexible endoscopes, laryngoscopes, endotracheal tubes, other similar instruments).	*High-level disinfection* Sporocidal chemical short contact.
Non-critical Equipment which touches intact skin (eg stethoscopes, table-tops, floors, beds).	*Intermediate-level disinfection* Hospital disinfectant with label claim for tuberculocidal activity. *Low-level disinfection* Hospital disinfectant without label claim for tuberculocidal activity.

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these products. The use of disinfectants for equipment sterilisation is summarised in Table 28.3.

ENVIRONMENTAL DISINFECTION

The routine disinfection of environmental surfaces is wasteful and unnecessary.

It is quite adequate to remove soil, litter, mud or vegetable matter with an efficient (and cheap) detergent and hot, clean water and a clean mop or disposable cloth.

If there is significant organic environmental soiling with blood, vomitus, urine or faeces, it may be appropriate to decontaminate with hypochlorite (ie powder or liquid bleach) prior to removal.



Figure 28.4 *Victim of a landmine blast being resuscitated in the UN Hospital in Kigali, Rwanda, in the aftermath of the 1994 civil war. Note the use of disposable gloves, washable aprons and overboots as protection against blood-borne cross-infection. (Photograph courtesy of J. Pearn.)*

Disposable gloves must be worn when cleaning contamination or spills of human organic material, especially blood (Figure 28.4). Paper or disposable cloths should be used to enclose the "soil" and to enable it to be carried or placed in a plastic bag for disposal. Concentrated hypochlorite should not be used in an enclosed area or on urine as the fumes may be unpleasant and are sometimes irritating to mucous membranes and the respiratory tract.

Impermeable gloves should be used not only for protection from microbiological contamination but also for protection from the potential corrosive, irritating or toxicological effects of those cleaning agents which are not intended to come into contact with the skin.

Skin disinfection should be carried out by effective, non-toxic and broad-spectrum agents.

One product is sufficient for routine use as there is no good evidence that the sequential use of different products offers any advantage over a single product. Indeed, there is some data to suggest that sequential use of soaps followed by detergents may be antagonistic as sometimes one product may be inactivated by the other.

There is no ideal skin disinfectant. The product chosen should be selected for its proven efficacy, lack of toxicity and side-effects, proven sterility, ease of use and presentation, appearance and cost. The most commonly used "pre-packed" skin antiseptics are sachet-contained, individually wrapped preparations of isopropyl alcohol.

The Australian Council on AIDS recommends that skin be disinfected with any of the following preparations: 70 per cent w/w ethyl alcohol, 80 per cent v/v ethyl alcohol, 60 per cent v/v isopropyl alcohol, alcoholic (isopropyl and ethyl) formulations of from 0.5 to 5 per cent w/v chlorhexidine, or aqueous or alcoholic povidone-iodine (1 per cent w/v available iodine).

MANIKIN DISINFECTION

CPR has brought about significant improvement in the survival rates of patients who might otherwise have been given up as dead, and has enabled first aid practitioners to intervene effectively. However, the success of CPR

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is greatly dependent upon technique, and those who teach CPR have long recognised the necessity for "hands-on" practice. Unfortunately, practising CPR on those who do not need it carries with it a risk of serious injury, so it has been necessary to develop manikins on which to practise. These manikins have some of the basic mechanical characteristics of the human cardio-respiratory system. They are expensive items and in a classroom it is neither practicable nor economic to have one manikin per student.

This means that manikins must be shared, and since the students have to apply their mouths to the face of the manikin, there is a theoretical possibility of disease transmission via saliva or droplet contamination from respired air, although the potential for salivary transmission of HIV is remote. There are a number of diseases which potentially might be transmitted in this way, such as HIV, viral hepatitis, tuberculosis, rubella and respiratory tract infections. To date, there are no proven reported cases of transmission of HIV in this manner. There is no evidence for an environmentally mediated (casual) mode of transmission of HIV; although the virus has been cultured from tears, urine, saliva and cerebrospinal fluid, these fluids are not implicated in disease transmission, with the exception of needle-stick injuries. The risk to health-care workers is thus negligible.

Hepatitis B virus occurs in numerous body secretions, but only blood, saliva and semen have shown infectivity.

Traditionally the way the problem has been tackled is to provide each student with a face- or mouth–nose-piece and, sometimes, disposable lung bags or airway tubes. As heat sterilisation is inappropriate because heat will damage the manikin, the face- or mouth-piece is decontaminated after use by soaking in antiseptic solution.

The Australian Resuscitation Council (ARC) recommends (1993) the use of 70 per cent alcoholic chlorhexidine for two minute immersion of face- or mouth–nose-pieces. This solution comprises 0.5 per cent w/v chlorhexidine in either ethanol 70 per cent or industrial (methylated) spirit 70 per cent v/v. ARC considered that isopropanol does not match ethanol until its strength reaches 95 per cent; it is not interchangeable with 70 per cent ethanol or industrial (methylated) spirit.

If alcohols are used as wipes, there must be at least 30 seconds contact time to permit disinfection of the manikin. Being toxic to tissues, and by virtue of their lipid-removing ability, ethanol and isopropanol are unsuitable for direct application to tissues, but this would not prevent their use in disinfection of manikins.

There are some practical disadvantages of using alcohols for disinfection of CPR manikins, including the fact that rubber swells and plastics may be hardened by prolonged contact with alcohols.

A combination of a suitable concentration of an alcohol, such as ethanol, and chlorhexidine offers the advantage of a wide range of action, and effectiveness against a number of viruses of particular concern, such as hepatitis B virus and HIV. Together with low human toxicity, low irritation and sensitisation potential, and reasonable aesthetic appeal in terms of odour, this makes it the agent of choice.

The Centers for Disease Control and Prevention, Atlanta, Georgia, in the United States, recommend that manikins be decontaminated between students by wiping the face and inside of the mouth of the manikin with a clean absorbent material with either diluted hypochlorite solution (one cup liquid household bleach per gallon (ie per 4.5 litres) of tap water) or 70 per cent isopropyl alcohol. The surfaces should remain wet for at least 30 seconds then wiped dry with another clean absorbent material.

The antiseptic solution recommended by the Australian Resuscitation Council is 0.5 per cent w/v chlorhexidine in 70 per cent ethanol. In an emergency, in the absence of this agent, we suggest that a suitable dilution of a freshly-made high-quality hypochlorite bleach is an effective substitute.

RISKS TO FIRST AIDERS

There are many microorganisms which are of potential risk to first aiders – indeed to all members of the health team. Influenza, herpes sores, tuberculosis, chicken pox and measles can all be transmitted by close body contact.

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Diseases which can be fatal if contracted by contamination with body fluids are hepatitis B (referred to as HBV); hepatitis C and E; and human immunodeficiency virus (HIV). The prevention of hepatitis and HIV cross-infection is crucial to the hands-on practice of first aid and paramedic work.

Both HIV and HBV viruses are killed by a range of disinfectants. In routine situations the most useful of these are ethanol, isopropanol and hypochlorites. Hypochlorite is less effective in the presence of organic matter.

Blood spills and needle-stick injuries

All who are involved in the pre-hospital phase of the fight for survival come into contact with blood. Several diseases can be transmitted by blood contamination, particularly hepatitis B and C and the human immunodeficiency virus (HIV). The chance of a first aider contracting one of these diseases during his or her work with the victims of trauma or acute illness is virtually zero. At the time of writing, there have been no confirmed reports of a first aider anywhere in the world contracting infection with HIV from a victim whom they were helping. Nevertheless, it is essential to know what to do if one has a contamination accident, that is if one sustains a penetrating injury or a mucosal splash from a victim's blood or body fluids.

The algorithm in Table 28.4 lists in summary form the procedures which should be followed as soon as possible after suffering an accident involving human blood and/or body fluids. Putting off the reporting of such an incident increases the risk of subsequent infection and decreases the success of any intervention.

Table 28.4 Algorithm for needle-stick and body fluid accidents

1 FLUSH needle-stick injury and mucosal splashes with running water.

2 SAVE body fluid source. (May be tested for HIV, hepatitis B or hepatitis C.)

3 SOURCE
- **a** *Unknown*
  (Major blood or body fluid splash or penetrating sharp injury.)
  Is health-care worker immune to hepatitis B?
  NO: Give I.M. (deltoid) 2 ml HBIG (hepatitis B immunoglobulin), available from Red Cross.
  YES: Give I.M. (deltoid) HIG, 2 ml.
- **b** *Known*
  Check HIV, hepatitis B and hepatitis C status.
  If NEGATIVE, no immunoglobulins needed.

4 ALWAYS
- **a** *Take blood* (clotted) from source; test or store if status known (may retest).
- **b** *Take blood from accident victim for HIV, hepatitis B and hepatitis C status.* Clotted sample required.
- **c** *Send all specimens to appropriate laboratory.*
- **d** *Inform appropriate medical adviser.* (Tests will usually occur next day, *not* overnight.)
- **e** *Contact infectious diseases physician or medical microbiologist if problems arise or source is a known HIV-positive patient.*

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Universal blood and body secretions precautions (BABS) for the first aider

It is not possible to identify on initial contact casualties possibly infected with transmissible agents. In order to prevent transmission of potentially infectious agents, all health-care workers should, therefore, use appropriate barrier precautions to avoid direct contact with blood or body fluids from any casualty.

Universal precautions through body substance isolation (BSI) and blood and body secretions (BABS) constitute a system of infection control designed to

- reduce the possibility of transmission of micro-organisms from one casualty to another primarily via the hands of first aiders, and
- protect first aiders from organisms carried by casualties.

There are four main elements to BABS. They are very relevant for all who care for patients.

1 *Handwashing* is a critical element in infection control. Ideally, hands should be washed before and after giving first aid, after removal of gloves, and as soon as possible after contamination with blood or body fluids.

2 *Gloves* should be worn for touching blood, body fluids, mucous membranes or non-intact skin of all casualties or when handling items or surfaces soiled with blood or body fluids. Gloves should be changed for different patient care procedures (eg emptying a urinary catheter bag, or inserting an endotracheal catheter or I.V. line).

Gloves should be changed if they become damaged. Gloves should not be washed, as they easily develop holes.

Vinyl and latex gloves are of equal efficacy for non-sterile procedures.

3 *A water-repellent barrier* in the form of a plastic disposable gown or apron should be used when it is likely that clothing will be soiled with body substances.

4 *Mask and eye protection* should be used if body fluids are likely to be splashed or projected onto the face and mucous membranes. People who wear glasses normally do not need to use extra eye protection. Lightweight clear plastic glasses should be available and used by any person who performs first aid procedures and does not normally wear glasses.

Other elements inherent in BABS for patient care and, where relevant, first aid include the following.

- Safe and responsible disposal of "sharps".
- Use of a resuscitation barrier if mouth-to-mouth or mouth-to-nose resuscitation is planned.
- Containment of used linen in an impermeable waterproof or closely woven bag.
- Routine disposal of paper and other waste not contaminated with blood or body secretions.

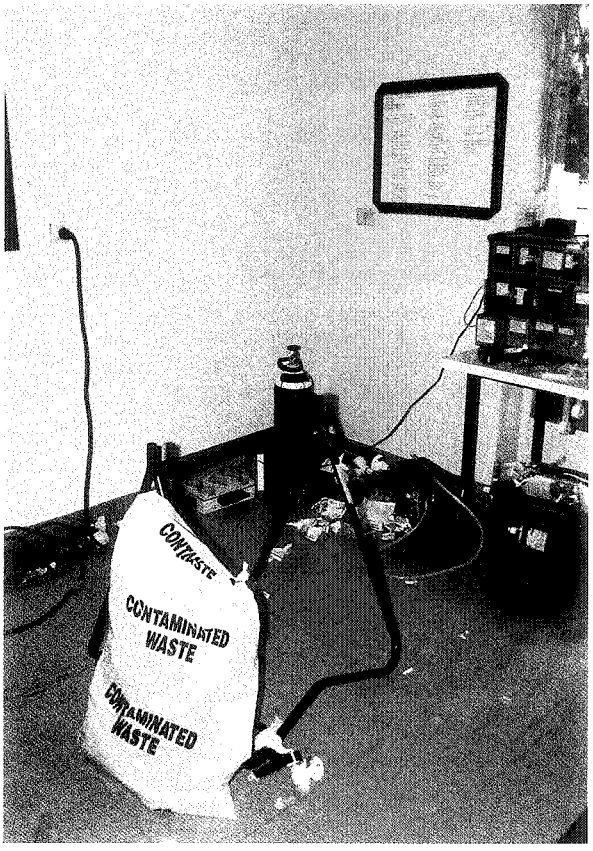


Figure 28.5 *The scene in a resuscitation bay at the UN Hospital in Kigali, Rwanda, after the resuscitation of a severely injured patient under desperate circumstances. All blood-soaked items (swabs, clothing etc.) have been treated as infectious, and sharps have been disposed of in special containers. (Photograph courtesy of J. Pearn.)*

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- It is unnecessary to use specific infectious or biohazard labels as all specimens are handled as if presumed infectious.
- Cleaning of spills requires the use of disposable gloves and the use of an appropriate decontaminant. The local disinfectant policy should detail the product and how it is to be stored, used and discarded.
- Housekeeping should be aimed at keeping area and equipment dust-free and free from organic material. Expensive disinfectants are not required. A housekeeping policy which sets out the simple methods and equipment required should be available for the cleaning staff.

Careful adherence to the above procedures will minimise the risk of infection to all health-care workers (Figure 28.5), for whom first aiders and paramedics comprise the forward troops. These procedures are the "fence at the top of the cliff" preventing exposure to microorganisms, rather than "the ambulance at the bottom of the cliff" trying to put together the pieces after the event.

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CHAPTER TWENTY-NINE

IMMUNISATION FOR FIRST AIDERS, PARAMEDICS AND OTHER HEALTH PROFESSIONALS

Joan Faoagali and John Pearn

The profession of first aid carries with it the potential of increased risk of infectious disease. All who work with the sick and injured are aware of this risk; but with meticulous attention to the doctrines of training, the potential for risk need never be translated into realised disease. A large number of infectious diseases are preventable by immunisation, surely one of the greatest discoveries in the history of humankind.

Immunisation is important not only in the context of protecting first aiders. Another, and a higher, ethic is the protection of patients or victims themselves from iatrogenic transmission of disease. There are many examples of this phenomenon in the recent history of medicine, not the least important instance being when unimmunised doctors spread rubella to pregnant women who were under their care.

Many vaccine-preventable diseases are of sudden onset. Those at greatest risk are the immediate family of the victim and, in the case of emergency care situations, the first aider or paramedic. Particular examples are those who care for victims of rabies, cholera, poliomyelitis, some types of encephalitis and diphtheria. First aiders and paramedics are thus part of the wider group of health-care workers who not only require the routine vaccines recommended for all citizens but also, because of their exposure to the sick and injured, require extra protection against diseases spread by excreta, blood and body fluids.

Immunisation may be either active or passive. Active immunisation consists of the administration of a modified product (antigen or toxoid or killed microorganism or part of an organism, or modified live microorganism) so that the body develops circulating antibodies to it. These antibodies persist in the blood stream for many years and cross-react with and attack the "wild-type" organism from which the original vaccine was made. Active immunisation takes time to protect the subject at risk, and usually up to three months or more are required for complete protection. Often a series of injections (or oral vaccines) is required to build up full immunity. In some cases of accidental exposure to infectious situations, urgent immunisation "after the event" can help protect a health worker or other contact. This is possible when the incubation period of the wild-type infection is longer than the time it takes to generate antibodies to the modified vaccine used for such an emergency immunisation. Those "caught short" and who have been in contact with some types of encephalitis and even milder diseases such as measles and rubella can be helped in this way.

Passive immunisation is a different phenomenon. Here, short-term "instant" protection can be given in some emergency situations if an unvaccinated and unprotected health worker is exposed. The antibodies are made in the lymphoid systems of humans or animals, extracted and purified, and then kept in refrigeration until needed. This passive immunisation is used to treat victims of snakebite or spiderbite, or unprotected workers exposed to rabies or hepatitis A and B. Passive immunisation is not always effective, does not last for more than a few weeks and can have significant side-effects.

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The terms "vaccination" and "immunisation" are often used interchangeably with reference to active immunisation.

All involved in pre-hospital patient care should be fully immunised, as a pre-planned condition of work in their profession. The specific diseases will vary from country to country, but it is essential to be protected from the diseases to which one's community is subject.

> *Benefits and risks are associated with the use of all vaccines. No vaccine is completely safe or completely effective. The benefits range from partial to complete protection from the consequences of disease. The risks range from common, trivial and inconvenient side-effects to rare, severe and life-threatening conditions. Recommendations on vaccination balance the scientific evidence of benefits as against costs and risks; to achieve optimal levels of protection against infectious or communicable diseases.* (Commonwealth of Australia NHMRC Australian Immigration Handbook, *5th ed. (1994) 1986.)*

Some of the key terms involved are defined below.

Vaccine A suspension of attenuated live or killed microorganisms (bacteria, viruses or rickettsiae), or fractions thereof, administered to induce immunity and prevent disease.

Toxoid A modified bacterial toxin.

Immunoglobulin Purified human antibody (immunoglobulin G, IgG).

Specific immunoglobulin Purified pool of human antibody containing a high titre of specific IgG, for example, rabies (RIG), zoster (ZIG), hepatitis B (HBIG) or tetanus (TIG).

Antiserum, antitoxin, antivenom, antivenene A preparation of antibodies derived from the serum of immunised animals. Not used widely nowadays, except when treating cases of snake, spider and stonefish envenomation.

VACCINES AND THE FIRST AIDER

All health-care workers, be they paramedics or physicians, first aiders or surgeons, should themselves be fully immunised and protected from diphtheria, tetanus, whooping cough, poliomyelitis, measles, mumps, rubella, hepatitis and tuberculosis. Those working in countries where rabies, cholera, typhoid, plague, yellow fever and Japanese encephalitis are endemic, should also be protected by prior immunisation.

Adults who have not received their childhood immunisations should discuss this with their local doctor. It is never too late to be protected, but different doses and varying timing of the repeated doses of vaccines may be necessary.

Remember

1. Immunisation should not be carried out during the course of an acute illness.
2. Mild local reactions are common after tetanus and the combined tetanus–diphtheria vaccine.
3. A history of allergic symptoms is *not,* in itself, a contraindication to vaccination, but special precautions may be necessary.
4. If the recommended intervals between doses are prolonged, immunisation *can* be continued with subsequent doses. There is no need to recommence the schedule or give additional doses. The immune response is *not* impaired by such delays.
5. The antibody response to specific immunisations can now be measured in the laboratory. Health-care workers should consider documenting their immunity to infectious diseases so that they know whether they are susceptible or immune (eg to chicken pox). If a vaccine is available and they are non-immune (eg to rubella, hepatitis B, tetanus), it is essential to obtain a booster dose.

Childhood vaccination schedules

Many first aiders will have received the recommended childhood vaccinations as set out in various publications and summarised in Table 29.1. These vaccinations will most likely have provided first aiders with childhood immunity against the contagious diseases listed.

Adults who have not undergone full childhood immunisations should carefully discuss how they can rectify this situation with their preferred medical adviser, to ensure that they are protected against these infections.

Some vaccines such as those to prevent *Haemophilus influenzae* associated with meningitis and pertussis may

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Table 29.1 NHMRC (Australia) Standard Childhood Vaccination Schedule August 1994

Age	Disease	Vaccine
2 months	Diphtheria, tetanus and pertussis Poliomyelitis	DTP – "Triple antigen" OPV – Sabin vaccine
	Haemophilus influenzae type b (Hib) (Schedule 1 or 2)**	Hib vaccine (a or b or c)*
4 months	Diphtheria, tetanus and pertussis Poliomyelitis Hib (Schedule 1 or 2)**	DTP – "Triple antigen" OPV – Sabin vaccine Hib vaccine (a or b or c)*
6 months	Diphtheria, tetanus and pertussis Poliomyelitis Hib (Schedule 1 only)**	DTP – "Triple antigen" OPV – Sabin vaccine Hib vaccine (a or b)*
12 months	Measles, mumps, rubella Hib (Schedule 2 only)**	MMR Hib vaccine (c)*
18 months	Diphtheria, tetanus and pertussis Hib (Schedule 1 only)**	DTP – "Triple antigen" Hib vaccine (a or b)*
Prior to school entry (4–5 years)	Diphtheria, tetanus and pertussis Poliomyelitis	DTP – "Triple antigen" OPV – Sabin vaccine
10–16 years	Measles, mumps, rubella	MMR
Prior to leaving school (15–19 years)	Diphtheria and tetanus Poliomyelitis	ADT – Adult diphtheria and tetanus OPV – Sabin vaccine

* *Abbreviations for Hib vaccines: **a** is HbOc ("HibTITER"); **b** is PRP-T ("Act-HIB"); **c** is PRP-OMP ("PedvaxHIB").*

** *There are two different schedules for Hib vaccines. Schedule 1 Hib vaccination applies to the use of HbOC and PRP-T. The selected vaccine is given at 2, 4, 6 and 18 months. Schedule 2 Hib vaccination refers to the use of PRPOMP. This vaccine is given at 2, 4, and 12 months.*

Note that a fourth Hib vaccine (PRP-D; "ProHIBit") is approved for use as a single injection for children over 18 months of age.

All of the vaccines in the standard schedule, except OPV (Oral Polio Virus Vaccine), are given by deep subcutaneous or intramuscular injection. Oral Polio Virus Vaccine (OPV) is given orally. OPV should never be injected.

not be appropriate for adults. Primary vaccination of adults with oral polio vaccine is also not usually recommended in developed countries due to the very occasional and rare development of paralysis in adults.

Vaccines recommended for adults

Each year, adults develop vaccine-preventable diseases because they have not received the recommended vaccines. In North America, these include hepatitis B, influenza, measles, rubella and pneumococcal vaccine. In Australia, there is no promotion of adult immunisations to improve the prevention and control of vaccine-preventable diseases.

The number and percentage of certain vaccine-preventable diseases reported in United States patients over 20 years of age is shown in Table 29.2. This information is not available for Australian adults.

Table 29.3 lists the estimated vaccine coverage amongst United States adults. These rates confirm the wide variation in coverage from 2 to 95 per cent for different vaccines. Similar rates are thought to occur in all developed countries.

In each of several recent epidemics in the United States, more than 40 000 influenza-related deaths occurred. Approximately 40 000 deaths related to pneumococcal infections and an estimated 300 000 new hepatitis B cases occur annually. Among young adults, the incidence of measles and mumps has increased.

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Table 29.2 Number and percentage of selected vaccine-preventable diseases reported in persons over 20 years of age (United States 1985–89)

Disease	Total cases	Cases in adults No.	(%)
Diphtheria	11	7	(63.6)
Hepatitis B	125 137	109 422	(87.4)
Measles	34 348	5 128	(14.9)
Mumps	34 138	3 632	(10.6)
Rubella	2 108	954	(45.3)
Tetanus	301	278	(92.4)

Table 29.3 Estimated vaccine coverage among adults (United States)

Vaccine	Age (years)	Coverage (%)
Diphtheria	>60	34–51
Hepatitis B	NA	2–90
Influenza	>20	17
Measles	18–29	85–95
Pneumococcal	>20	9
Rubella	15–44	80–90
Tetanus	>60	16–59

NA = not applicable

Ten to fifteen per cent of childbearing-aged women are not immune to rubella, and most Americans over 60 years of age lack adequate protection against tetanus and diphtheria. Safe and effective vaccines are available for all these diseases, and the cost of vaccination is substantially less than the cost of treating the disease.

Health-care workers are at an increased risk of infectious diseases because of their concentrated exposure to the sick and because they may have unprotected exposure to blood and body fluids in an emergency situation. Such persons, especially those without documentation of previous vaccinations, should be considered susceptible and should be started on an age-appropriate vaccination schedule which should include:

Tetanus and adult diphtheria toxoid
Measles
Mumps
Rubella
Oral (or inactivated) polio vaccine
Hepatitis B

Influenza may be included if presentation occurs in autumn.

BCG vaccination for protection against tuberculosis may need to be initiated if it is considered that there is a risk of contact with open tuberculosis.

First aiders fully immunised as children

For these the following regimen is recommended.

- *Tetanus booster* doses every 10 years.
- *OPV* (oral polio virus vaccine – Sabin vaccine) revaccination between 15 and 19 years of age.
- *Measles, mumps and rubella* (MMR) vaccine booster between 10 and 16 years of age.
- *A course of hepatitis B* vaccine if not undertaken as a child. The need for booster doses of hepatitis B vaccine is generally suggested after 5 years for individuals at high risk. Booster doses are not recommended for individuals less than 10 years of age who were vaccinated as infants or young children.

  Hepatitis B antibody detection in serum confirms the development of protective antibodies. Some recipients of hepatitis B vaccine do not respond with the production of detectable antibodies. Current advice is that a fourth injection can be given, but it is not appropriate to continue revaccinating *ad infinitum*. Persistent non-responders should be informed of the need for hepatitis B immunoglobulin within 48 hours of inoculation with hepatitis B surface antigen.
- *Influenza* First aiders should consider obtaining an annual influenza immunisation. They are often

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exposed to the elements and unprotected respiratory secretions and are at risk on both counts from exposure to influenza virus. First aiders are expected to work under adverse conditions and when not feeling well may be incubating influenza which they then pass on to their patient who may be less robust than the first aider. Annual influenza vaccinations have been shown to decrease hospitalisation of the elderly with respiratory infections and deaths from pneumonia from elderly and compromised patients. Vaccination of health-care workers is increasingly advocated as a method of ensuring that these important community members are not removed from the work force by preventable diseases. First aiders are important first line health-care providers and should be similarly protected.

Contagious diseases and microbes for which there is currently no vaccine available

The first aider may be exposed to any of the following diseases and microbes.

- *Chicken pox* is a highly contagious infection caused by the *Varicella zoster* virus. It usually produces a mild infection in children which can be fatal in 7–10 per cent of immunocompromised individuals.
- *Cytomegalovirus* (CMV) causes a form of infectious mononucleosis. Infection generally results in a mild infection in immunocompetent individuals. CMV is spread by saliva (kissing), genital secretions (sexual intercourse) and as transfused white cells.
- *Infectious mononucleosis* is caused by the Epstein–Barr virus and like CMV this is generally mild in immunocompetent individuals, spread by sexual activity, saliva and as transfused white cells.
- *Herpes simplex viruses* cause cold sores and genital herpes. These viruses are spread by direct contact with the active lesion, for example, through kissing, touching or genital contact. Wear gloves, and wash hands or mucous membranes after contact with active lesions.
- *Respiratory viruses other than influenza A and B* There is a large number of different viruses which are capable of causing respiratory infections. They are spread by fingers and fomites (mutually handled objects such as handkerchiefs), so handwashing is an important protective measure for individuals and prevents cross-infection with these organisms.
- *Rotavirus* is a virus which mainly causes childhood diarrhoea. Careful handwashing after handling fomites, or after contact with children who have rotavirus infection, will interrupt its transmission.
- *Human immunodeficiency virus* (HIV) causes AIDS. If an inoculation incident occurs, discuss this urgently with an appropriate infectious diseases specialist.
- *Hepatitis C virus* is spread by blood via inoculation. If an inoculation incident occurs, discuss this urgently with an appropriate infectious diseases specialist.

WHEN NOT TO VACCINATE

There are some circumstances in which vaccination is not advisable. These are set out in Table 29.4. However, there are also many false beliefs about the risks of immunisation; these beliefs (Table 29.5) should never deter first aiders from gaining the necessary protection.

SPECIAL CIRCUMSTANCES

In certain circumstances not covered elsewhere in this chapter, particularly those facing health-care workers in developing countries, specific vaccination action may be desirable to ensure that the first aider is adequately protected. See Table 29.6.

All adults travelling to developing countries should be immune to diphtheria and tetanus and, if not previously immunised, should be given a primary immunising course (three doses of ADT administered at time zero, 4–8 weeks, and 12 months) with boosters administered every 10 years thereafter. All individuals should be immune to measles, mumps and rubella. For practical purposes, those born before 1957 can generally be considered to be immune to measles and mumps. All other individuals should be vaccinated unless it can be documented that they have either received vaccine on

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Table 29.4 Contraindications to vaccination for first aiders

Indication	Comment
Pregnancy.	Live vaccines should wait until after parturition except in exceptional circumstances, such as a polio epidemic.
Immunocompromised (eg HIV infection or malignant disease).	Live vaccines are usually contraindicated. Must be assessed by a specialist.
Fever over 38°C.	Vaccination should be postponed until recovery.
Use of corticosteroids in high dose, oral, topical or injectable forms.	Precludes vaccination with live vaccines.
Allergy to egg products.	Inability to eat, or to like eating, eggs does not constitute allergy.
Encephalopathy within 7 days following a previous vaccination.	
Immediate allergic or anaphylactic reaction after a previous vaccination.	
Previous proven infection with the organism for which the vaccine is being administered.	Except for tetanus toxin which is a poor antigen. Infection should be followed by a toxoid vaccination course.
Recent previous organ transplantation, such as bone marrow, heart, kidney, liver.	
Sharing a household with an immunocompromised individual.	MMR is safe as it is not transmitted. Discuss with a specialist.
Recent receipt of normal or hyperimmune human immunoglobulin.	In general, vaccines should be given 3 weeks before or 3 months after the dose of immunoglobulin. Seek specialist advice for details in specific circumstances.
Recent blood transfusion (within 3 months).	
Proven allergy to vaccine components such as neomycin or streptomycin.	Seek specialist advice.

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Table 29.5 False contraindications for vaccination for first aiders

Indication	Comment
Breast feeding.	There is no evidence of risk to the breast feeding baby if the mother is vaccinated with the vaccines described here. There is also no contraindication to vaccination of the baby and no risk to successful immunisation.
Redness and swelling at the injection site.	
Fainting, nausea or mild fever after vaccination.	
Family history of reaction following vaccination.	Should be carefully assessed to ensure that immunodeficiency does not exist.
Family history of convulsions.	
Prematurity.	
Asthma, eczema, hay fever or snuffles.	Vaccination contraindicated only if high dose oral, topical or injectable steroids are being used. Inhaled steroids are not a contraindication.
Mild illness with low-grade fever or mild diarrhoeal illness in an otherwise well person at the time of a scheduled immunisation.	
Current antimicrobial therapy or the convalescent phase of an illness.	
Pregnancy of the first aider's mother or other household contact, except in the case of live virus vaccines such as rubella, measles and poliomyelitis.	
Recent exposure to an infectious disease.	
Allergies to penicillin or other antibiotic, except anaphylactic reactions to neomycin (eg MMR-containing vaccines) or streptomycin (eg OPV).	None of the vaccines licensed in the United States or Australia contain penicillin.
Allergies to duck meat or feathers.	No vaccine available in the United States or Australia is produced in substrates containing duck antigens.
Family history of an adverse event unrelated to immunosuppression following vaccination.	

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Table 29.6 Vaccinations which may be advised for first aiders in special circumstances

Vaccine	Reason
Meningococcal vaccine	May be indicated in an epidemic situation as advised by public health officials.
Pneumococcal vaccine	Persons who have had a splenectomy or who are over 65 years of age or who have had a head injury resulting in leakage of cerebrospinal fluid.
Typhoid vaccine	May be recommended by health officials in certain conditions such as flooding or other disasters where clean water supplies are compromised.
Hepatitis A vaccine	May be indicated in situations where clean water supplies are compromised or in an outbreak. Specialist advice should be sought.
Q fever	Q fever vaccine is recommended for those at risk of infection with *Rickettsia burnetii*, ie first aiders working in abattoirs, as veterinarians and laboratory personnel handling potentially infected tissue should be considered for vaccination.
Cholera	Helpful in certain refugee situations.
Plague	Essential in some countries where plague is endemic.
Yellow fever	Necessary for health, and essential for the passage of national frontiers from countries in South America and Africa where yellow fever is endemic.

or after their first birthday or have had physician-diagnosed disease.

Rubella vaccine should be administered to all individuals, particularly women of childbearing age, unless they have documentary proof of having received rubella vaccine on or after their first birthday or laboratory evidence of immunity. A history of prior rubella disease is unreliable and should not be accepted. Influenza vaccine is recommended for routine annual administration to adults 65 years of age and older and to individuals at any age who have chronic illness. Pneumococcal polysaccharide vaccine is recommended for administration to the elderly and the chronically ill.

Hepatitis B virus vaccine is recommended for individuals at high risk of exposure to hepatitis B virus. These include primarily health-care personnel at high risk of contact with blood that might be infected with hepatitis B virus, homosexuals, users of illicit injectable drugs, and individuals living and working in institutions for the mentally retarded, and household contacts of carriers of HBsAg.

Smallpox has now been eliminated from the world.

CONCLUSION

Health-care workers, whether first aiders, paramedics or nurses, face the risk of infectious diseases when performing their duties; knowledge, coupled with the use of appropriate vaccines and safe practices, will minimise these risks.

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CHAPTER THIRTY

PREVENTIVE FIRST AID

John Pearn

Long gone are the days when paramedics and professional first aiders regarded their role simply as a passive one – eternally responding to the victims of trauma but doing little to prevent the injuries in the first place. The new millennium will see a switch to empowerment of first aiders to take a proactive role in the profession of first aid; and in this context to join, belatedly, the ranks of other health professionals such as dentists, doctors and sports physiotherapists who include a preventive component in the daily practice of their profession.

To this end, the term "preventive first aid" embodies the positive concepts of proactive education, example, advocacy to increase safety, and the exertion of some control so that both the rate and severity of injuries might be reduced. So important is the theme of preventive first aid that the era is now upon us when its absence makes any first aid intervention or teaching incomplete.

ADVOCATES FOR SAFETY

Because of their knowledge and experience, first aiders and all who are involved in pre-hospital care find themselves in an ideal position to achieve reduction of illness and injury. This is possible locally, "on-site", at such sporting events as football, swimming carnivals and cycle races. In all codes of football, it is known that proportionately more injuries occur in the early part of each game and in the early weeks of each new football season. Proper pre-season training, effective pre-match warm ups and the use of mouth guards and protective headgear are all known to reduce injury. The doctor, paramedic or volunteer first aider has a professional duty to express a strong opinion about the desirability, indeed necessity, of such practices. In fun runs, and long distance foot and cycle races of all types, the provision of adequate fluid-stations will reduce the incidence of heat stress. Again, the first aider has an inescapable preventive role to play at pre-race conferences, by expressing an opinion about the reduction of preventable illness simply by the provision of frequent stations dispensing adequate volumes of fluids of the correct osmolality.

Many head injuries involving cyclists can be prevented, and their severity reduced, by the use of helmets. Advocacy for their widespread purchase and, even more importantly, their universal use will make the first aider's life more pleasant at amateur cycling carnivals. At motor racing venues, first aiders can be effective in lifting compliance rates for the use, by speedway drivers, of safety devices and heat-resistant clothing. To do this, the racetrack first aider should become known to the regular drivers, should mix with them in the pits and gain their professional respect, and, by precept and example, lift the level of safety compliance, especially with respect to the provision of safety equipment and its use.

As well as these roles in the context of sports, the first aider has an inescapable responsibility in the area of industrial preventive medicine and health. If a high rate of injuries is occurring in a local workforce, the on-site first aider must bear some of the responsibility for this

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unsatisfactory state of affairs. On the factory floor first aiders treat lacerations, contusions, foreign bodies in eyes, burns and the victims of electric shock. They see in greater realism and with a starker perspective than do those at higher levels on the managerial ladder the effects of these accidents, and thus are often the most qualified to offer preventive solutions. Continued advocacy for a safer workplace, for safer homes and gardens and for safer areas of public recreation is thus a major function of all who care for victims in the first links of the "chain of survival".

Basic Life Support (BLS) is that phase of emergency care which supports the ventilation and circulation of victims of respiratory and/or cardiovascular collapse. The American Heart Association, over the past two decades, has included in the concepts of training for CPR the philosophy that prevention is an integral component.

> *Basic Life Support includes the teaching of primary and secondary prevention. The basic concept, presented by the American Heart Association (AHA), that it is possible to prevent and control coronary heart disease, should be reinforced during the teaching of BLS, with an emphasis on prudent heart living and risk factor modification. The earlier this information is transmitted to the community, the stronger the impact on mortality and morbidity.*

FIRST AID TRAINING – WORKSHOPS, OUTDOORS AND EXPEDITIONS

Does first aid training increase the motivation to avoid injury? There is a widely held belief that if a sportsperson, a tradesperson or a parent has received some first aid training, then they and their workmates and children will be less likely to be injured by preventable trauma. There is a widespread belief that if one is educated about injuries and their management, then intelligent and informed individuals will modify their behaviour to reduce risk. Relatively little research has been undertaken to test these assumptions, but what little there is tends to support the apparent logic of this belief.

Major studies to test the hypothesis that first aid training is an effective means of accident prevention began in Canada in 1969. Research suggested that first aid training was associated with safer driving behaviour and a reduction in road traffic accident rates. A universal difficulty with this type of question is that those who attend first aid classes, are more likely, *a priori*, to be caring and concerned individuals and, therefore, less likely to sustain accidents. However, prospective studies of factory workers have shown some heartening trends. One large study, involving 1 200 subjects who took a four-hour St John Ambulance emergency first aid course in the town of Leek in Staffordshire, has shown that community-wide accidental injury rates subsequently fell in the general society. This and other research has indicated that there are substantial indirect effects which flow on from first aid training. It is further believed that there is a "trickle-down" effect, particularly of benefit to the children of trained first aiders. If this is so, this bonus provides an inter-generation, quasi-genetic, beneficial effect also on the safety attitudes of the next generation.

A retrospective but controlled study of the effects of first aid training on injury rates to both staff and employees of the Forestry Commission of Tasmania showed a post-course reduction of trauma. There were different effects on different groups of first aid students. After receiving first aid instruction, there was a 43.7 per cent reduction of reportable injuries (those the subject of potential compensation claims) to high-risk employees (loggers, axemen, mechanics and labourers) over the ensuing 5 years; and a 36 per cent reduction to a medium-risk group (such as storemen, graduate foresters and supervisors). The results from this study have paralleled other similar findings from the Mt Lyall Copper Mine in Queenstown (also in Tasmania).

There are certain high-risk groups in which the principles of preventive first aid are especially important. These include those whose recreational or employment pursuits place them in high-risk situations where, if an accident or sudden illness occurs, then individuals have to act as the first aiders themselves. Such individuals thus have a particularly strong motivation to prevent injuries in the first place. These groups include yacht crews and wilderness adventure

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teams. The latter include mountain climbers, cavers, wilderness theme parties, backpackers, kayaking and adventure rafting groups. In all these cases, their route takes them far from the roadhead, and often, even far from practical aircraft access or rapid rescue. Such groups have a special need for pre-expedition training in the principles of preventive first aid.

One other group which has particular need of the principles of preventive first aid are servicemen and women in many of the armed services. In these instances, accidental injury or preventable illness not only renders them dysfunctional from the point of view of their primary fighting role, but may compromise the primary aim of the military operation itself.

MODUS OPERANDI OF INJURY PREVENTION

There are two types of injury prevention – primary and secondary. Primary prevention aims to prevent the accident or illness itself – to ensure, for example, that people are not burnt, do not sustain broken bones, or do not become bitten by snakes. Secondary prevention, by contrast, aims at the prevention of death in those who are already injured; it aims to prevent complications in non-fatal trauma, and to promote convalescence in the shortest possible time span. Thus secondary prevention aims at things such as the prevention of inhalation of vomitus in unconscious head-injury victims, the prevention of vascular injury by splinting fractured limbs before evacuation by transport, and the prevention of secondary infection by adequate protection and cleaning of wounds and burns.

Secondary prevention has been one of the core tenets of first aid since its identification by von Esmark in Germany and by Surgeon-Major Peter Shepherd in the United Kingdom, in the 1870s.

It is, however, in the neglected area of primary prevention that the first aider needs to take a more professional role. There are three ways in which injuries can be prevented.

1 Education
2 Design improvements
3 Safety legislation

Each of these approaches is quite distinct, albeit complementary to the others. None is mutually exclusive.

Education as a preventive tool is logical and easy, but so often therein lies its weakness. Research studies show that simply telling people about risks does not, by itself, modify risky behaviour – especially in those at highest risk. "Preaching to the converted" is a very common sin of long-standing tradition amongst all the disciplines of the health professions. To be effective, preventive messages in both the print and electronic media must be specific, must be focused and must emphasise positive aspects. Promoting healthy lifestyles can best be undertaken by the use of high-profile role models. Reducing both recreational and occupational trauma can also be most effectively undertaken, in the educational sense, by the use of appropriate sporting and other heroes. Research has shown that if a magazine article, a poster or other graphic display, or a television programme, is too horrific, then the target viewer simply brings ego-defence mechanisms into play, and will either not identify as a target subject ("it can't happen to me"), or will simply switch programmes or "turn off". First aiders and paramedics can easily become inured to the sight of blood, bones and bodies, and forget that many potential students will simply not enrol in basic first aid classes because they "faint at the sight of blood".

There are many types of trauma in which it is not possible to increase safety by better equipment and design or by improving safety legislation. Bathtub drownings in infants, suffocation in cubby-house and tunnel collapses and snakebite accidents are such examples. None of these types of injury are amenable to any legislative or regulatory approach, and very few to designing better equipment or modifying the built environment. In so many injuries, education, albeit inefficient, is the only approach that the first aider has. Maintaining the point, focus and target of the preventive safety message – with the audited result of improving safety motivation and practice – is one of the great challenges for all health-care professionals today.

Trauma can also be reduced by improved equipment design and the invention of better safety hardware. The

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safety-catch on a rifle or hand-gun is one such example. The first aider has a major duty to promote the use of the safest equipment. Almost all industrial eye injuries can be prevented by the use of safety goggles. Most facial burns from acid in school chemistry classes can be prevented by face-shields. Virtually all toddler drownings can be prevented by protecting this vulnerable group from water hazards (principally home swimming pools), by the use of safety fencing with self-closing, self-latching gates, with high, hidden locks. The first aider has to promote the concept that the hardware of safety is not an "optional extra" but an integral part of the basic equipment itself. Educating both the public and specific groups of potential victims is a long-term process, and success is never immediate. Whereas car seat restraints were once considered a luxury, they are now regarded as standard equipment, and rightly so. In the next decade they will become standard equipment in buses as well. Mountain climbers are exposed to great potential risks but, with proper equipment and adherence to safety drills, realised injuries are relatively uncommon. The difference between potential risks on the one hand and realised risks on the other is a very important distinction. Professional first aiders and ambulance officers must be strong advocates for the reduction of realised risks by the use of safety equipment. In this context, the Royal Society for the Prevention of Accidents in the United Kingdom has adopted the slogan "Safety is not the foe of adventure but an inescapable ally".

The third portal for injury prevention encompasses the broad field of safety legislation and recreational regulation. Historically, and in one sense unfortunately, the use of safety legislation has always been brought in as a last resort. It took a century of advocacy and often bitter acrimony to protect young children against the dangers of chimney sweeping in Britain. It took some four decades of bitter political debate to achieve safety legislation to paint the Plimsoll Line on the sides of ships to prevent overloading. It took 20 years of vigorous and sometimes bitter advocacy in Brisbane, and more than 500 needless toddler drowning accidents in Queensland, to achieve safety legislation to ensure that domestic water hazards were contained. Safety legislation promoting seat restraints in cars, the use of helmets by motor cyclists and the protection of construction labourers by appropriate footwear, all required prolonged advocacy to become a reality. Today, signs on industrial construction sites which read "No boots, no job" are the practical embodiment of an ethic of safety. This approach, ultimately carried through to legislative protection, again enshrines the ethic of the Good Samaritan where one is seen as one's brother's keeper. The first aider has a key role to play in this advocacy process.

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PART

V

THE AFTERMATH *of* FIRST AID

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CHAPTER THIRTY-ONE

TISSUE HEALING AND SCARRING

Robert Pearce and Gael Phillips

If a trauma victim survives, tissue damage repairs itself. One aim of first aid is to promote this healing and to remove factors which inhibit it. After a laceration, burn or fracture, inflammation begins immediately; and inflammation is the first step in tissue repair (Figures 31.1 and 31.2). This effective process of repair is inhibited if movement, lack of oxygen or infection occur. Thus wounds are kept at rest and fractures are splinted to prevent the movement of healing tissues, blood supply is maximised by positioning limbs properly, and wounds are kept clean to prevent infection. An understanding of wound healing and tissue repair is the universal facet of the science of first aid.

REGENERATION AND REPAIR

Regeneration and repair are fundamental facts of life and they occur to varying degrees in virtually every organism. The process of wound healing leads to reconstruction of its component parts – the connective tissue, the blood vessels and the covering epithelium.

Not all tissues are capable of regeneration. Those whose structure is determined by specialised function (eg nerve tissue) are largely replaced by fibrous or scar tissue in the process of healing. Limited regeneration of liver and gland cells can occur in humans. Bone injury is repaired by regeneration of bone cells (osteogenesis). All tissues heal better in the young.

There are two types of repair in open soft tissue wounds (ie wounds involving a break in the continuity of the skin):

1 primary wound healing in incised sutured wounds, and
2 secondary wound healing in open excised wounds.

The basic aspects of the healing process are similar in both; the wounds heal by the synthesis of granulation tissue and new connective tissue, and the subsequent formation of a fibrous scar.

The injury and the response

Both cells and blood vessels are damaged at the moment of injury. Blood extravasates into the wound. If too much blood extravasates into the tissues, the resulting haematoma can impede tissue repair. Two systems of coagulation – the "intrinsic" and the "extrinsic" – act to promote rapid blood clotting. Within a few moments of blood platelets touching any tissue or substance not a sound component within blood vessels, they undergo a change leading to clot formation. In this intrinsic system, platelets release phospholipids, and coagulation is, therefore, partly controlled by a combination of vasospasm and intravascular thrombosis. The extrinsic coagulation mechanism is activated by thromboplastin released from injured cells, normally in the damaged tissues outside the blood vessels.

Nearby vessels, especially capillaries and venules, subsequently dilate. Platelets and white cells aggregate and begin to stick to the endothelial lining. In doing so they release proteolytic enzymes which initiate an enzymatic cascade attracting inflammatory cells. These cells, the leucocytes, migrate between the endothelial cells into the area of injury. Within a few hours the edge of the wound is infiltrated with granulocytes and

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Figure 31.1 *The first phase of wound healing. Unsutured wound 24 hours after injury, showing gaping defect in the epidermis, dermis and subcutaneous fat, lined by an acute inflammatory reaction, fibrinous exudate and blood clot. H & E magnification × 49.*

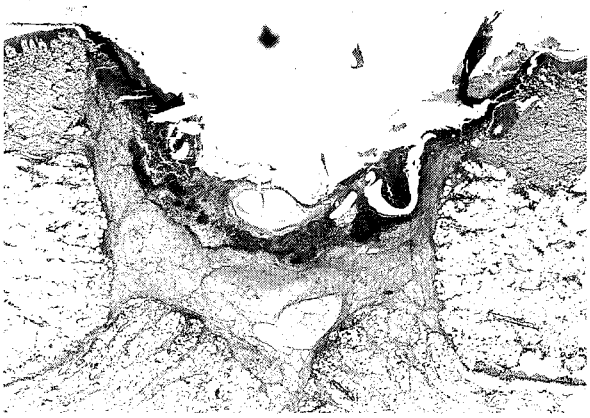


Figure 31.2 *First phase of healing. Unsutured incised wound 48 hours after injury, showing a more advanced acute inflammatory response with extension of inflammation into the subcutaneous tissue. H & E magnification × 49.*

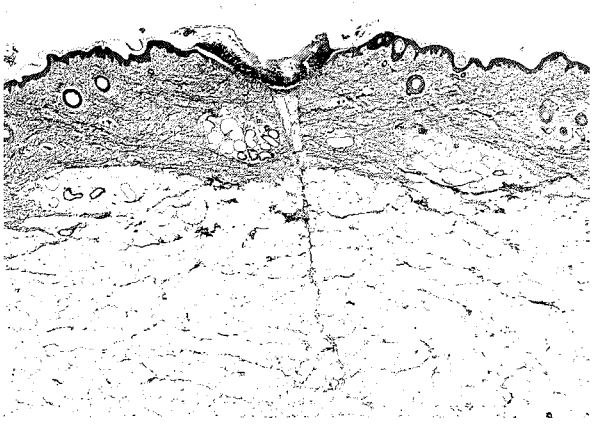


Figure 31.3 *Primary healing in an incised sutured wound 48 hours after injury. The gap is bridged by fibrin and there is minimal acute inflammation. The incised edges of the wound are closely opposed. H & E magnification × 49.*

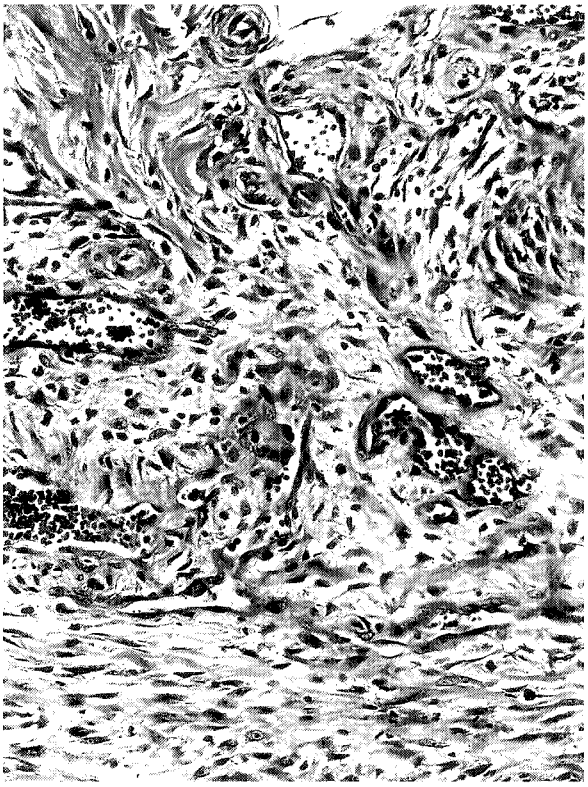


Figure 31.4 *Second phase of wound healing. Granulation tissue in a healing surgical wound showing proliferation of fibroblasts and capillary blood vessels with a scattering of acute and chronic inflammatory cells. H & E magnification × 294.*

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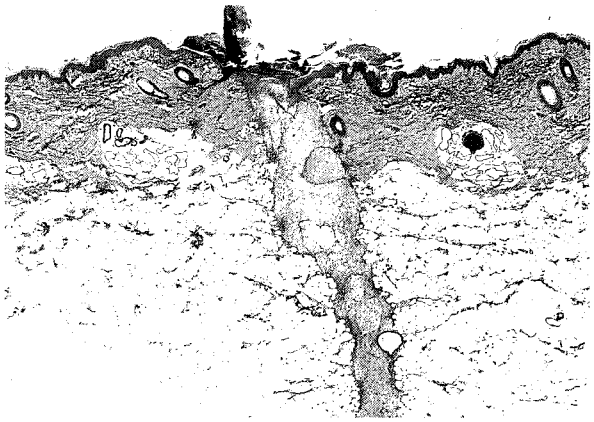


Figure 31.5 *Secondary healing in unsutured incised wound 4 days after injury. The gap is bridged by fibrin and there is a proliferation of fibroblasts and capillary sprouts along the cut margins of the wound, forming a small amount of granulation tissue. H & E magnification × 49.*

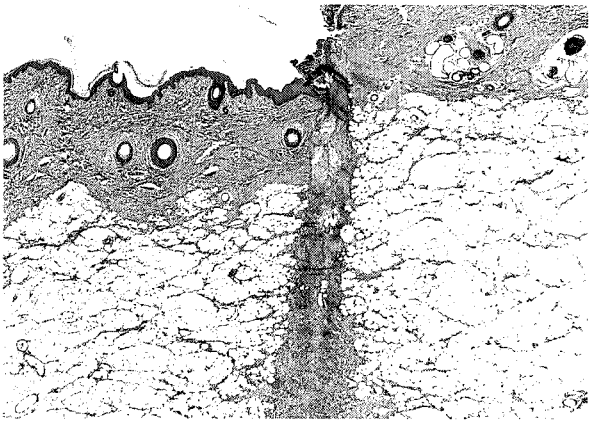


Figure 31.6 *Primary healing in incised surgical wound 4 days after injury. The defect in the dermis contains residual fibrin blood clot and there is also well developed granulation tissue bridging the wound in the subcutaneous fat. H & E magnification × 49.*

macrophages, highly metabolic white cells. These are soon replaced by active fibroblasts which produce scar tissue. This then represents completion of the inflammatory or first phase of repair:

- initiation of coagulation mechanism,
- dilatation of capillaries in wound margins, and
- migration of plasma, leucocytes and macrophages into the wound.

The second phase of wound repair is dominated by the presence of granulation tissue – a vascular and cellular tissue containing fibroblasts, macrophages and mast cells (Figure 31.4). The fibroblasts synthesise collagen and other essential elements of new connective tissue. There is little regeneration of elastic tissue during wound repair. In skin wounds, connective tissue is necessary to provide support for the new epidermis. In a skin wound, as soon as the granulation tissue is formed, epithelial cells from vascular endothelium and residual hair follicles spread over the healing surface. They later multiply and mature to reconstruct a functional epidermis. The process of neovascularisation is essential to wound healing. It is inhibited by inappropriate creams and lotions applied to the wound and by lack of an adequate blood supply.

Collagen is synthesised by fibroblasts which require some form of chemical mediator to become activated. Collagen formation starts on the third day after injury and rapidly increases from the fifth to the tenth day of repair. Synthesis of collagen is accompanied by a marked increase in the mechanical strength of the wound.

The third phase of repair consists of a period of remodelling. During this phase, which is of indefinite duration, the wound assumes the appearance of a relatively avascular scar. In a matter of weeks contraction of the wound, due to the activity of contractile fibroblasts, occurs. Healing bones remodel themselves depending on their proper alignment, initial immobility and subsequent lines of normal force and stress.

Connective tissue formation and remodelling during wound healing represent a highly ordered sequence of biochemical and cellular events finally leading to restoration of the tissue integrity. The chemical contents of neurophils are important in the first phase of wound

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repair. Such chemicals include prostaglandins, collagenase, oxygen-free radicals and proteolytic enzymes.

Other chemical mediators, such as histamine released from platelets and mast cells, are responsible for the early phase of vasodilatation. Similarly the capillary engorgement and increased vascular permeability which follow are determined by serotonin, kinins and prostaglandins. Some chemicals which are released have specific chemotactic properties. Chemotoxins attract cells and result in the early migration of neutrophils and other leucocytes to the site of the wound.

Biosynthesis *de novo* of collagen, the major structural component of the skin, plays a central role in repair of soft tissue wounds. Collagen metabolism is itself regulated by the interaction of fibroblasts with the surrounding extracellular connective tissue matrix, and also by cytokines released from the endothelium, platelets and inflammatory cells. Ten to thirteen days after a wound has been sustained, only those cells located beneath the newly formed basement membrane scar are still actively synthesising collagen.

Excessive collagen production during the repair phase will result in the formation of an hypertrophic scar called a "keloid". Such raised, thickened fibrous scar is commonly seen in healed, deep partial thickness burns, and in other wounds where healing and repair have been prolonged or disrupted by movement, infection or by a foreign body reaction. Keloid scar formation also involves abnormal collagen synthesis where fibroblastic activity progresses in an accelerated way. True keloid tends to occur most commonly in certain skin types (eg negroid) and should be distinguished from hypertrophic scar formation, which is self-limiting and to some extent reversible.

PRINCIPLES OF WOUND CARE

The principles of first aid apply in the primary care of an open soft tissue wound – reassurance, rest, immobilisation, elevation, control of haemorrhage and the application of clean dressings. These steps are particularly important before transport or movement are considered. In practice, carry out the following procedures.

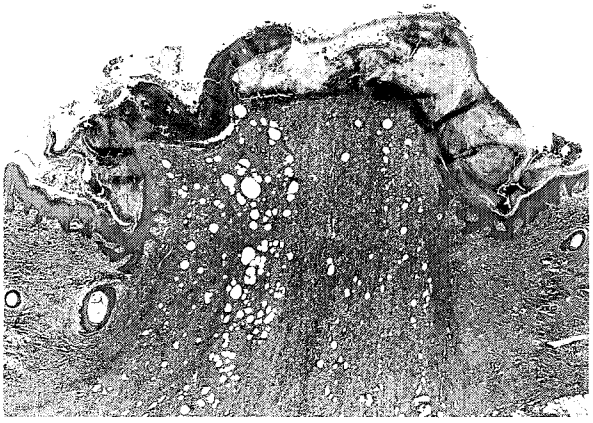


Figure 31.7 *Secondary healing in unsutured incised wound 6 days after injury. There is a wide defect in the dermis and subcutaneous fat which is filled with necrotic cell debris and blood clot with only partial replacement by granulation tissue, growing in from the edges of the wound. Regeneration of the surface epithelium is not complete and the surface of the defect is covered by a scale crust of desiccated blood clot and necrotic inflammatory cells. H & E magnification × 49.*

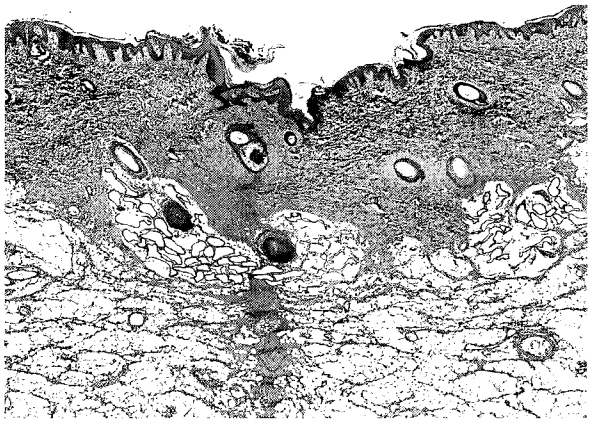


Figure 31.8 *Primary healing in sutured incised wound 8 days after injury. The defect is now completely bridged by fibroblastic granulation tissue and the surface epithelium has completely regenerated. H & E magnification × 49.*

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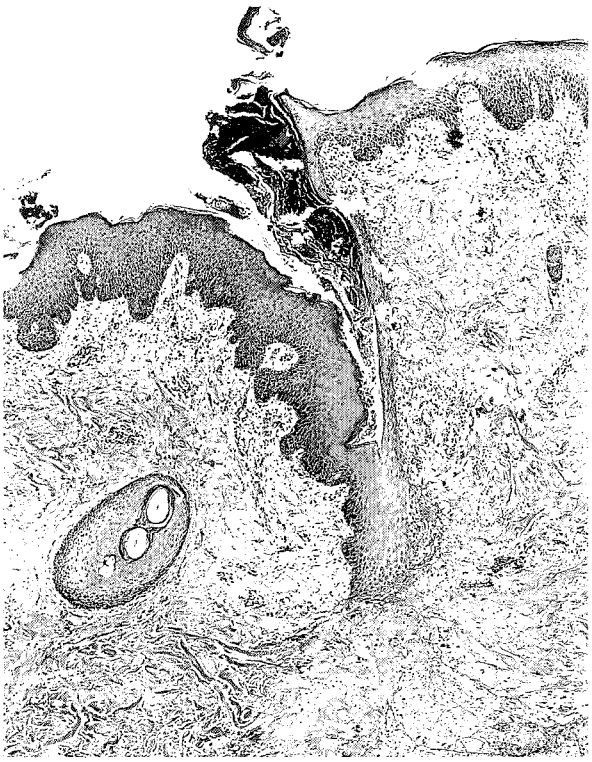


Figure 31.9 *Partial dehiscence of sutured surgical wound. Regeneration of the epidermis extending down to line the incised defect within the dermis. The wound shows acute inflammation, oedema and granulation tissue in the surrounding dermis. H & E magnification × 98.*

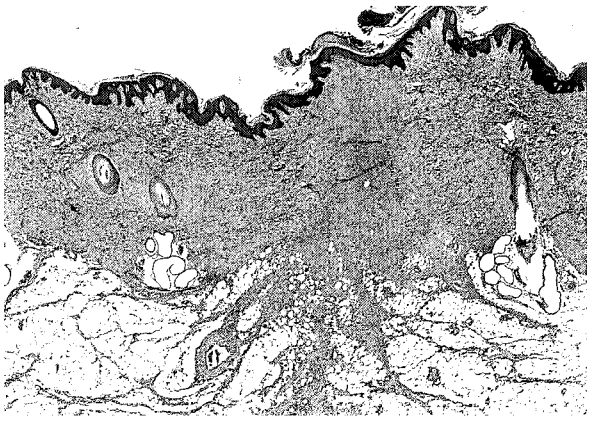


Figure 31.10 *Third phase of healing in a sutured incised wound 12 days after injury. The defect in the dermis is now replaced by a young fibrous connective tissue scar which is undergoing further organisation and remodelling. Trabeculae of fibroblastic granulation tissue extend into the subcutaneous fat. H & E magnification × 49.*

- Clean a dirty wound. Foreign body contaminants must be removed mechanically or washed out with clean water or with antiseptic solutions to reduce sepsis and infection during the healing phase.
- Make an untidy wound tidy. Wound repair and healing may be facilitated by judicious mechanical (surgical) debridement of ragged and potentially non-viable tissue in all layers before the wound is closed. In all but trivial wounds, this is not the task of the first aider.
- Adequate haemostasis is essential to avoid haematoma formation and subsequent infection.
- Primary anatomical repair. It is reasonable to expect a clean wound which has been repaired surgically within 6–12 hours to heal without complication. All anatomical layers and structures must be explored and repaired at this stage as a primary procedure to ensure optimal functional and cosmetic results.
- Drainage of any potential dead-space should be considered to prevent haematoma formation.
- Supportive dressings, immobilisation, elevation and regular post-operative surveillance are essential for all but trivial wounds.

Dressings for closed (repaired) wounds provide support to the wound, thus facilitating repair.

A wound which is likely to produce exudate or require drainage (eg a blistered burn) should be covered with absorbent gauze which must be changed at regular intervals to prevent maceration of the skin and wound infection.

Small, dry wounds can be covered with an antibiotic ointment (not powder) or antiseptic solution.

Complications

1 *Scar formation* The best cosmetic result will be obtained by primary closure of a clean, tidy wound and early, primary healing in which complications have not occurred. Scar contracture as a late phenomenon can interfere with joint mobility or produce an unsightly cosmetic result. This may require surgical revision. Unstable scars, usually in burn wounds, have thin epithelium and are sometimes best replaced with a skin graft.

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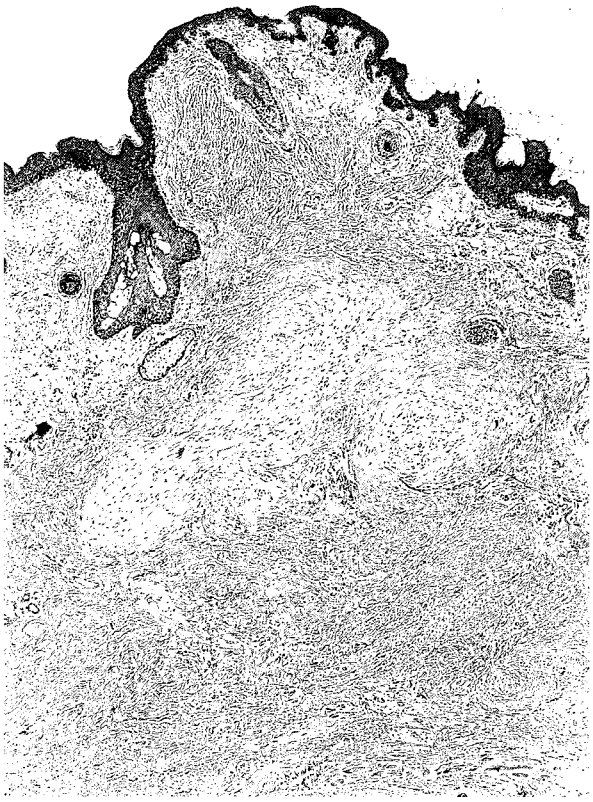


Figure 31.11 *Fully healed incised sutured wound showing a mildly hyperplastic organised scar after 6 weeks. H & E magnification × 49.*

2 *Delayed healing* This usually results in unsightly, unfavourable scar formation. Excessive fibrous tissue in hypertrophic scars not uncommonly causes pain and itching. If these occur, a doctor may reduce symptoms by an injection of triamcinolone.

3 *Wound infection,* chronic ulcer formation, granuloma and unstable scars are all likely to result from interference with the mechanism of repair. Mechanical contaminants, for example foreign bodies such as dirt, clothing and suture material, and infective microorganisms are commonly involved. Metabolic disturbances within the patient, such as malnutrition, anaemia or diabetes mellitus, may prolong healing and vascular damage will slow the normal mechanisms of repair.

4 *Involvement of other structures,* for example irritation and pressure on nerves, may cause pain. The tethering of a scar to muscles or tendons will interfere with normal function and the normal range of movement, and may be painful.

Wound healing of burnt tissues is promoted by

- rapid and complete restoration of fluid balance and blood volume,
- exclusion of bacteria,
- early debridement and closure, and
- maintenance of nutrition and resistance to infection.

Some dressings and some surgical procedures used in the care of wounds are specialised. In particular, debridement and grafting may be required not just once but several times to achieve optimum wound healing.

Nevertheless, even the most sophisticated plastic surgery depends to some extent on the state of the wounds with which the surgeon starts. Expert first aid and expert care in the field will ensure that infection is reduced to a minimum, that secondary damage (before healing starts) is prevented and that the wonder of wound repair is promoted from the moment of injury.

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CHAPTER THIRTY-TWO

POST-TRAUMATIC STRESS SYNDROME

Beverley Raphael and Lenore Meldrum

The after-effects of a frightening event may affect both the victims of the incident and those who treat them. One of the aftermaths of distressing situations may be a psychological condition known as the Post-traumatic Stress Disorder (PTSD). It may affect victims of traumatic events long after the physical wounds have healed. It may be an unfortunate legacy in those who confront danger or disaster but who otherwise escape physical injury. It is a subject which is as inescapable for the first aider or paramedic – who so often is at the scene of frightening or traumatic events – as it is for the doctor or nurse.

In today's society anxiety and anxiety-related syndromes affect a significant proportion of the population. These syndromes may be transient or prolonged and can be associated with great disability and costs. The disorders grouped in the spectrum of anxiety-related syndromes include simple phobias, social phobia, obsessive compulsive disorder, generalised anxiety disorder, panic disorder, agoraphobia and Post-traumatic Stress Disorder. Anxiety frequently follows exposure to a traumatic event and is now often recognised as closely linked with Post-traumatic Stress Disorder syndromes.

To help with consistency and communication the American Psychiatric Association has designated five key criteria for the identification of Post-traumatic Stress Disorder. These are:

1 the experience of an extreme or abnormal event;
2 phenomena of re-experiencing that event, such as intrusive thoughts or nightmares;
3 phenomena of numbing, detachment and denial such as shutting off feelings and withdrawal;
4 phenomena of persistent increased arousal including sleeping difficulties, irritability and hypervigilance; and
5 the persistence of symptoms.

Some degree of these symptoms may be experienced transiently after experiencing any frightening event. It is only when the symptoms persist that victims can truly be said to be suffering from Post-traumatic Stress Disorder. These five criteria for the diagnosis of Post-traumatic Stress Disorder are elaborated further in Table 32.1.

Experiences which are outside the range of usual human experiences are the necessary, but not necessarily sufficient cause, for the development of this disorder. Such extreme stressors are usually the key factors prior to the onset of this disorder and are strongly influential in the shape of its phenomenology. It is important to understand that most traumatic events are sudden, untimely and unexpected and may have a violent and shocking aspect as well.

WHAT IS A TRAUMATIC EVENT?

A psychologically traumatic event is a very serious and very stressful event which affects an individual or those very close to them. It may be a very serious accident; the death of someone, especially a child; violent deaths, such as a murder or a suicide; many deaths at the one time; rape; personal disasters such as vehicle or air crashes; disasters such as fires and floods; shooting and community violence; being robbed, mugged or assaulted; and many other such episodes.

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Table 32.1 Diagnostic criteria for Post-traumatic Stress Disorder

A The person has been exposed to a traumatic event in which both of the following were present:

1. the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; and
2. the person's response involved intense fear, helplessness or horror. Note: in children, this may be expressed instead by disorganised or agitated behaviour.

B The traumatic event is persistently re-experienced in one (or more) of the following ways:

1. recurrent and intrusive distressing recollections of the event, including images, thoughts or perceptions. Note: in young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
2. recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognisable content.
3. acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: in young children, trauma-specific re-enactment may occur.
4. intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
5. physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

C Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

1. efforts to avoid thoughts, feelings or conversations associated with the trauma;
2. efforts to avoid activities, places or people that arouse recollections of the trauma;
3. inability to recall an important aspect of the trauma;
4. markedly diminished interest or participation in significant activities;
5. feeling of detachment or estrangement from others;
6. restricted range of affect (eg unable to have loving feelings); and
7. sense of a foreshortened future (eg does not expect to have a career, marriage, children or a normal life span).

D Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

1. difficulty in falling or staying asleep;
2. irritability or outbursts of anger;
3. difficulty concentrating;
4. hypervigilance; and
5. exaggerated startle response.

E Duration of the disturbance (ie symptoms in criteria B, C and D) is more than one month.

F The disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning.

Specify as:
Acute: if duration of symptoms is less than 3 months.
Chronic: if duration of symptoms is 3 months or more.
Specify as:
With delayed onset: if onset of symptoms is at least 6 months after the stressor (*The Diagnostic and Statistical Manual,* American Psychiatric Association, 1994, pp. 427–429.)

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Traumatic events are so called because they generate psychological trauma, injury or wounding. To the victim this injury is just as real as physical injury and may have long-lasting severe effects. Psychological first aid is directed towards mitigating these effects as well as promoting treatment and recovery.

People who experience traumatic events may be living their lives as usual when the episode occurs, so there may be an element of great shock. The person may feel frightened, stressed and unable to act; they may be unable to respond, or may feel frozen. Sometimes the event threatens the person's life or the life of loved ones, and that may bring extreme horror and distress, both at the time it occurs and afterwards. "Traumatic encounter with death" and "traumatic loss" are two of the most prevalent subcategories of stress or traumatic experiences that can occur.

Traumatic loss refers to the loss of close attachment figures (eg bereavements) which occurs in association with sudden, unexpected, untimely or otherwise traumatic circumstances of death. In such instances, people suffer grief as well as traumatic stress and are very much in need of help, support and comfort.

Traumatic encounter with death refers to the stresses experienced in sudden or violent confrontation with death which may lead to Post-traumatic Stress Disorder. This may lead to the type of reaction that people have after severe exposure to combat or violence – whether they be direct victims or be incidentally exposed as, for example, bank customers are in a hold-up. The encounter may be sudden (violence, abuse, rape, sudden unexpected and horrifying events), protracted (torture), or of a chronic nature such as ongoing physical and sexual abuse in childhood. All may lead on to Post-traumatic Stress Disorder. When emergency workers experience someone else's death in a traumatic or violent manner or suffer a threat to their own lives they may also experience these reactions. Disasters like the Ash Wednesday bushfires, the Granville rail disaster, mass shootings of urban bystanders, the Piper Alpha oil rig fire, and the Hillsborough disaster have all caused many people to suffer traumatic stress reactions. Certain variables lead to more severe traumatic stress reactions:

- the severity of a stressor,
- the perceived stress associated with it,
- the appraisal of an event as threatening,
- the helplessness to act,
- the shocking or inescapable nature of the horror, or
- being witness to violence.

The greater the traumatic stress, the greater is the need to anticipate that care will be necessary.

REACTIONS TO TRAUMATIC EVENTS

It is normal to experience reactions to stressful events. The event itself is abnormal and to react to it is normal. Reactions may come on straightaway, or after a period of shock and numbness. Sometimes they may be delayed for days, weeks or even longer. Reactions vary a great deal for each person, for there is always some personal perspective of what this experience means to this person at this time.

Common reactions

Each person's experience is a unique personal event. Past experiences and individual perceptions all contribute to differing behaviours. However, there are some reactions that commonly appear amongst people caught up in an abnormal event. It is usually reassuring to people to know how others have reacted in similar situations, even if individual reactions also occur. Stress reactions may cause physical symptoms, emotional reactions or thinking disturbances.

Normal feelings experienced

Any person exposed to a traumatic incident may experience numbness or disbelief at what has happened for some time after the incident. In such cases, the event seems unreal or like a dream. This is part of psychological reaction by which those so stressed by a traumatic experience attempt to "close off" their emotions to protect themselves, and sometimes those close to them, from being hurt. It is an ego-defence mechanism. Often, partners find it extremely difficult to cope with such numbing and perceive it as rejection.

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There is often an inability to comprehend the enormity of what has happened.

Many people who have experienced a traumatic event are left with an awareness of personal vulnerability as well as great fears of death, further damage to oneself or loved ones and panicky or irrational feelings.

Fearfulness may generalise or lead to extreme anxiety in the face of any reminder. Immense anger often surfaces at whoever is seen as having caused the incident or "allowed it to happen". People may also express anger at the injustice and senselessness of what has happened, as has been seen in a number of community scenes around the world when children have been abused or murdered. There is also often a generalised anger and irritability for some time after being traumatised.

People frequently feel helpless in times of such crises and this may constitute a special problem because these feelings may be mistakenly interpreted as personal weakness. Recognition of this aspect of trauma reactions is very important because this may evoke a sense of shame which may have a detrimental effect on the recovery process. It is also important that health professionals see their own feelings in the context of this situation and realise that such an experience would have been likely to have induced helplessness in most people. People are often melancholy after trauma. They feel sadness about human destruction, and about losses of every kind. Sometimes the biggest loss is their belief that the world is safe and predictable. These common feelings usually last for short periods only and gradually diminish over ensuing weeks. Different feelings may be dominant as time goes by. There is much evidence to suggest that it is helpful to people who have been stressed to be able to express these feelings and to share their experience with others who understand.

Additional traumatic stress reactions may be expressed in many ways and in various combinations. Tension is often displayed in exaggerated startle reactions or general agitation – physical or mental. Sleep disturbances are common post-trauma reactions. People experience an inability to sleep, or have thoughts that prevent sleep, often replaying the incident over and over. Dreams and nightmares of the incident or other vivid and frightening events are often reported by traumatised people. Fearfulness of the place, or reminders of the incident, are very common responses among victims of trauma. Intrusive memories and feelings that interfere with concentration and daily life are frequent or they may be in the form of "flashbacks" when the experience is relived. When people attempt to shut out such distressing reminders it often leads to a deadening of feelings and thoughts which may extend to other areas of life. There may be a loss of interest in and involvement with life, and a withdrawal in interpersonal relationships.

Irritability is a common reaction experienced by traumatised people and may be accompanied by frequent mood swings. Depression about the event or other past events, or non-specific depression or guilt about how one behaved, may all be associated with exposure to a traumatic incident. Social needs differ from person to person – some experience a great need to be alone while others may need constant companionship. There may be a powerful need to share the experience with others, especially those who have been through the same thing. Or there may be a wish to avoid all talk and reminders of the horror, at least for a time. Such feelings of preoccupation and avoidance may alternate and predominate. Wherever possible, carers – whether these be fellow first aiders or paramedics or other health professionals – should attempt to accommodate these needs and be sensitive to the victim's individual experience. Physical sensations of tiredness, palpitations, tremors, breathing difficulties, headaches, tense muscles, aches and pains, loss of appetite, loss of sexual interest, nausea, diarrhoea and constipation are but some of the numerous physical reactions recorded by people suffering a traumatic stress reaction to an acute event. Mental reactions such as poor concentration, difficulty in thinking, self-doubts, or loss of confidence may concern the traumatised person and these are often under-reported because those affected may fear for their sanity. Self-medication is one of the dangerous aspects of trauma reactions. Taking pills and drinking excessively to deal with the distress of

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intrusive memories may lead to ongoing physical or emotional problems, or dependence on alcohol or other drugs.

INCIDENCE, PREVALENCE AND ILLNESS BURDEN ASSOCIATED WITH POST-TRAUMA SYNDROMES

Experiencing traumatic events is normal. A recent community study showed that nearly 40 per cent of young adults report such experiences. The traumatic events most referred to included: sudden injury or serious accident, physical assault, seeing someone seriously hurt or killed, news of sudden death or injury of close relative or friend, narrow escape, threat to one's life, rape, and natural disaster including fires. The rate of Post-traumatic Stress Disorder after exposure was 23.6 per cent and the lifetime prevalence of Post-traumatic Stress Disorder was 9.2 per cent making it a common disorder among young adults. Epidemiologic research has found that the prevalence of Post-traumatic Stress Disorder in the community at large is about 1 per cent (0.5 per cent men and 1.2 per cent women). It is higher in populations who are exposed to traumatic events such as combat or mugging, and in such groups the rate lies between 3.5 per cent and 20 per cent. The more severe the trauma, the greater is the risk that this disorder will become chronic.

Studies of rape victims have shown high levels of traumatic stress reactions; 94 per cent of women presented with symptoms meeting the criteria of Post-traumatic Stress Disorder shortly after the rape. Three months afterwards, 47 per cent of women were so affected; and as long as 17 years after a rape, 16.5 per cent had Post-traumatic Stress Disorder. Childhood sexual abuse is also very likely to lead to Post-traumatic Stress Disorder, although good systematic studies are lacking. Reports suggest at least 50 per cent of children so abused develop Post-traumatic Stress Disorder, with adverse effects on development and with vulnerability to the problems of later life.

People in community and workplace settings where they may be exposed to traumatic events may also be very vulnerable to adverse traumatic stress reactions. There is evidence of heightened response to traumatic stressors for some workers as part of their work roles, for example, first aiders and paramedics, police, ambulance and officers of other emergency services. They are at heightened risk of exposure. The social environments of the workplace are very important in this context. In the best organisations – ones that are sensitive to the occupational hazard of Post-traumatic Stress Disorder – attention to training, education and preparatory briefing, and effective past experience, will diminish the risk; while in other organisations lack of these may heighten it. After exposure, the best systems recognise trauma and acknowledge the workers' stress and contribution, provide support and debrief those at risk. If organisations and senior individuals fail in this regard, negative consequences follow, with a heightened risk of the disorder for the workers who are involved.

Perhaps the most tragic impetus to the understanding of trauma has come from war, particularly studies on Vietnam veterans. Studies in the United States, where systematic data is available, suggest that at least 500 000 Vietnam veterans are suffering from Post-traumatic Stress Disorder related to the severity of the stressful experience they had during combat in Vietnam. Kinzie reviewed other studies of epidemiology, including studies of survivor groups. He found that after a disaster, such as the Mount St Helens volcano eruption, there was a lifetime prevalence of Post-traumatic Stress Disorder of 2–3 per cent. The commonest stressor for men is combat and, for women, assault.

Refugees are a group that have often been exposed to severe trauma, deprivation and violence. Kinzie, in a subsequent study of Cambodian people in the community, showed that about 90 per cent suffered Post-traumatic Stress Disorder, and most were not receiving care. Holocaust survivors are another vulnerable group with 87 per cent having been described as suffering the Concentration Camp Syndrome. Prisoners of war also show ongoing levels of Post-traumatic Stress Disorder; over 30 per cent in a group reported by Kinzie had Post-traumatic Stress Disorder some 40 years after their imprisonment.

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Co-morbidity or secondary morbidity

It has been well recognised that co-morbidity is common with Post-traumatic Stress Disorder, with other anxiety disorders, depression and substance abuse being common concurrent diagnoses. In a study conducted by Davidson and his colleagues it was found that there was greater psychiatric co-morbidity for those with Post-traumatic Stress Disorder, increased risk for suicide attempts, increased frequency of bronchial asthma, hypertension, peptic ulcers and social phobias.

PSYCHOLOGICAL FIRST AID

Psychological support is extremely important for traumatised people in the aftermath of a traumatic event and there are a number of practical ways this can be provided. First, the helper should always offer human comfort and support as the initial first aid measure. Just being with an affected person, protecting them from further harm and conveying recognition of their suffering in a compassionate and respectful manner is very important.

Many people feel uncomfortable with trauma survivors because the latter may have a great need to talk about what has happened. Alternatively, some are numb. The need for empathy and identification with the horror of the victim's experience may overwhelm the would-be helper. It is important for the affected person to be able to tell the "trauma story" in a supportive and caring environment, as and when they are ready. This may well be the beginning of their first tentative steps towards psychological recovery. The terror that has been experienced by the person may require ventilation in the time just after the event or in other circumstances later when they have distanced themselves from the danger. By being able to talk about a perceived close encounter with death they are likely to be more able to process the emotional impact this has had on their lives and to move on to acceptance and adjustment. However, people should not be forced to talk of what has happened – just gently supported and encouraged. There must, however, be a recognition that they may need to express their feelings and discuss what has happened bit by bit so that they can cope with their distress in manageable doses.

The ability to be an attentive and supportive listener is important for anyone who wishes to assist a survivor of trauma. Many survivors express an almost irrational anger at God for natural disasters, at the Government for the state of the roadway that may have contributed to a motor-vehicle accident, or at the helpers at the scene of an incident who may have unwittingly irritated the victim, or been seen not to have done enough. The recognition by helpers that such anger is natural and not personal may assist those concerned in dealing with this particular aspect of the stress reaction.

Survivors of trauma have sometimes been observed to regress to earlier stages of development when suffering from acute stress – for instance curling up in the foetal position, wishing to be cuddled, whimpering or even wetting themselves. Helpers must be careful not to be judgemental regarding such behaviours. The sufferer must not be made to feel ashamed about them and may need support from others in order to be able to accept that these responses were part of their reaction to trauma.

It is also very important that the normal networks of support be kept intact at any time of crisis. For instance, whenever possible, families should be kept together and children kept with their parents. It is necessary that, as much as possible, the normal communication channels be kept open and that no member of the kinship group be left isolated and alone.

The key elements of the task confronting the first aider in giving psychological assistance to the traumatised are set out in Table 32.2.

It is necessary that any helpers involved in the support of traumatised people carefully monitor their own levels of distress. Working with trauma victims may lead to a high level of empathic response. In extreme cases the helper may develop many of the stress reactions of the victim. A higher level of identification with the victims may lead to the helper's feeling a need to withdraw or, alternatively, they may become far too enmeshed in the problems to be effective in their task.

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Table 32.2 Psychological first aid

1	Offer human comfort and support.	Tell the "trauma story".
2	Allow the person to talk freely.	Express a range of feelings: • helplessness, • anger (natural and acceptable), • pain. Don't be judgemental.
3	Behaviour varies enormously.	Support and explain "regressions".
4	Keep family units together.	
5	Retain human dignity.	
6	Identify those stressed or at risk.	
7	Assist those decompensated.	

This could almost be referred to as "there, but for the grace of God, go I" reaction.

It is important that both the stressed and the helpers work towards a goal of active mastery of the problems being encountered. The helpers should facilitate this in a non-intrusive manner, whilst assisting the person to confront and integrate their experience. It is absolutely essential that the helpers set accomplishable limits on the task of assisting recovery after trauma. All helpers in trauma-related incidents need to be able to "unload" their own reactions – whether on an informal basis with work colleagues, family or friends, or in a more formal review, supervisory or counselling arrangement. There is thus a need for a hierarchy of care and support.

Pamphlets and other educational materials outlining normal reactions to abnormal and stressful events and how to deal with them should also be provided. Such concrete items give people something to turn to afterwards and may include general guidelines about post-traumatic stress and advice about when, how and where to get further help, including telephone numbers of appropriate contacts.

First aiders and other health professionals who are unable to handle these intense feelings or physical sensations should seek professional help from a psychiatrist, psychologist, social worker or appropriately qualified counsellor. For both victim and paramedic, seeking assistance should also be encouraged if they feel their emotions are not returning to normal over a period of time, if they continue to have physical symptoms, or if they continue to have nightmares and disturbed sleep. If the traumatised person has no person or group with whom to share their feelings, they may need specific support or professional counselling assistance. If their relationships and work seem to be suffering, or if they are having accidents or making uncharacteristic mistakes, or increasing the use of alcohol or drugs, professional advice will be of some assistance.

DEBRIEFING

Many emergency and related services provide stress debriefing programs for their workers. These are usually associated with peer support programs and sometimes backed up by counselling and employee assistance programs.

Psychological debriefing is usually held shortly after the traumatic incident. Workers may initially be provided with a brief opportunity to deal with their

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feelings or stress as they come off shift – this is known as "defusing". Many of the debriefing processes follow a model known as Critical Incident Stress Debriefing (CISD) where workers meet in a group and recount their experience and reactions and are provided with educational and supportive input by a mental health professional and trained peer supporters. Such debriefing usually takes place 24–72 hours after the event. It may be followed up, if necessary, by further debriefs. While most people experience these programs as helpful, many of those most in need may not be reached. Some need further assistance including one-to-one counselling. Nevertheless, this is a form of early intervention, almost a "group first aid", and may be helpful to many people in dealing with some aspects of their psychological trauma, even if not guaranteed to prevent subsequent difficulties. Such debriefings are sometimes provided to community groups, for example, after a major accident or disaster.

COUNSELLING AND PSYCHOTHERAPY

Many people exposed to severe trauma may need individual counselling and psychotherapy to help them work through and recover from their traumatic experience. This may vary from a few sessions to more prolonged and in-depth therapy. Such counselling should be provided by mental health professionals who are knowledgeable and skilled with respect to trauma therapies. Trauma psychotherapy and cognitive behavioural therapies have been shown to be effective. If traumatic loss has occurred, it may be necessary for the traumatic experiences to be worked through before grieving can occur and effective bereavement counselling be implemented. It is essential that the importance, for the affected person, of being able to tell their "trauma story" is recognised by first aiders and paramedics, and that this is supported with recognition of the need to "dose" their emotional response in manageable amounts. It should also be noted that earlier traumatic experiences of childhood abuse, for instance, may lead to vulnerability and confound the recovery process. Therapists working in this field need to maintain their empathy, but also be aware of counter-transference reactions which may lead to distancing or enmeshment and much lessen their ability to help those affected.

Some people may become so highly aroused, distressed, depressed or in other ways disturbed that specialist psychiatric assistance is essential. People who develop Post-traumatic Stress Disorder should be supportively referred for assessment and therapy including medication if this is necessary. If this does not occur, undue and prolonged disability may result.

Education about stress reactions and how to manage them and promote recovery is essential and should be used alongside the interventions outlined above. Education in emotional or psychological first aid is essential, not only for those at risk but also, as with other forms of first aid, for the wider community.

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NOTES AND REFERENCES

These annotated references have been chosen to provide a source of further help and reading on selected topics of importance to all who are part of the pre-hospital care team.

CHAPTER 1 – ACCIDENT SITE CONTROL

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Leonard, R.B. (1993). Initial scene assessment and patient care. *Aviat Space Environ Med* **64**: 546–551. (Hazardous materials, ie chemicals that are toxic, corrosive, flammable, or explosive, are a ubiquitous aspect of modern life. After manufacture, they are shipped by truck, train, barge, and pipeline, and stored at a wide variety of locations, including factories, military bases, and warehouses. Accidents involving hazardous materials present an added dimension of

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danger to emergency personnel arriving first at the scene, and have the potential to produce chemically contaminated patients who require special medical treatment. Personnel arriving first at the scene must understand how to evaluate the scene for fast and safe mitigation without endangering themselves. Chemically contaminated patients require prompt treatment, which, for optimal outcome, must begin at the scene. Although frequently the identification of the hazardous materials involved is not known initially, emergency personnel may safely provide medical care to the victims by understanding and following the principles of hazardous materials accidents and the pathophysiology of chemical injuries which can result.)

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CHAPTER 2 – TRIAGE

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CHAPTER 4 – FIRST AID IN THE AIR

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A dictionary of terms used in aeromedical evacuation

Ambient pressure This is the pressure in the aircraft cabin at any one time. If a pressurised aircraft is cruising at 35 000 feet, the cabin will be pressurised so that it is at an equivalent of 6000–8000 feet. This will be the cabin ambient pressure as it will be in an unpressurised aircraft actually flying at 6000–8000 feet.

Bull nose adaptor This is the valve used on large oxygen cylinders in many English-speaking countries. It is a female connection that has a thread on its inner surface that is specific for that gas. It will mate only with a male fitting that is gas specific.

Deep vein thrombosis (DVT) Blood clot in the leg or thigh that has the potential to become life-threatening if it breaks off and embolises to the lungs. There is an increased incidence of DVT in relatively immobile patients. It is one of the most common causes of unexpected death during flight in passengers. This is called by some people the "Economy Class Syndrome". The organisation for which the author works (Access 24) routinely uses prophylaxis against DVT in all transported patients unless there is a specific contraindication.

End tidal CO_2 monitor A device attached to the patient's breathing circuit that measures the partial pressure of carbon dioxide at end expiration. This gives an estimate of, among other things, the adequacy of the patient's ventilation.

Endotracheal tube cuff The endotracheal tube is the tube taking gases from the ventilator tubing into the patient's trachea. The cuff is that part of the tube which may be expanded to form an airtight seal with the tracheal wall by adding air. When the ambient pressure drops this air will expand, thus increasing the pressure in the seal and potentially damaging the tracheal wall.

Heparin This anticoagulant is used in the prevention of deep vein thrombosis (see above). Unless there are specific reasons why it should not be used, Access 24 routinely gives 5000 units of heparin subcutaneously every 8 hours during an air transport of a patient.

"Hilift" This is a truck whose rear compartment is fitted with hydraulic lifters that allow it to be elevated to the level of the doors on large jets. These trucks are used mainly for loading food containers onto aircraft. Many airports have modified highlifters available to load stretcher patients directly from the tarmac onto the aircraft.

Invasive blood pressure monitor A mechanism for measuring the patient's blood pressure by inserting a cannula directly into an artery and then via a transducer displaying the blood pressure on a screen. Its main advantages are that it is very accurate, it gives a continuous display of blood pressure as opposed to other techniques that will only measure the pressure intermittently, and it allows easy sampling of blood for analysis.

Minute volume This is the volume of gas breathed by the patient in one minute. It may be calculated by multiplying the respiratory rate by the tidal volume.

Pin index A system used in the United States and many other countries including Australasia to ensure that the correct gas tubing can only be attached to cylinders containing the correct gas. The yoke that fits over the cylinder head contains two pins in unique locations which match up with two holes on the cylinder head.

Pulse oximeter A device for the non-invasive measurement of oxygen saturation of arterial blood. The patient–equipment interface is usually a sleeve that fits over one of the patient's fingers.

Thomas Pack A commercially available back-pack for storing and transporting disposable items that may be required for the medical management of critically ill patients.

Transducer Any piece of equipment that can transform energy from one form into another. In the context used in this chapter, the transducer converts blood pressure into a signal that may be read on a visual display.

Transmeridian A journey that, in going east to west or vice versa, crosses time zones. It is only transmeridian flights that produce jetlag

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Vacuum mattress A mattress composed of many beads enclosed in an airtight bag. When all of the air is sucked out of the bag, the mattress becomes rigid and maintains the shape it was in at the time the vacuum was created. Vacuum mattresses are used by many retrieval organisations as the main surface on which the patient lies for the duration of the transport. The fact that it maintains its shape for long periods makes it ideal for transporting patients in whom movement may be dangerous (eg patients with acute spinal injury). Its main disadvantage is its bulkiness.

Ventilator A device for artificial ventilation of the lungs. All modern ventilators produce positive pressure to drive gas into the lungs. They all need a power source of some type. This is usually provided either by electricity or by high pressure gas.

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CHAPTER 10 – DEFIBRILLATION

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CHAPTER 11 – DEVICES FOR EXPIRED AIR RESUSCITATION

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CHAPTER 12 – ASSISTED VENTILATION

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CHAPTER 13 – BURNS

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CHAPTER 14 – CRUSH INJURY

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CHAPTER 15 – DIVING ACCIDENTS, PULMONARY BAROTRAUMA AND GAS EMBOLISM

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CHAPTER 16 – DROWNING

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Kemp, A.M. and Sibert, J.R. (1991). Outcome in children who nearly drown: a British Isles study. *Brit Med J* **302**: 931–933. (This paper reports the outcome, in terms of the degree of brain damage, of 330 children who survived near-drowning episodes in Great Britain. All of the children who had regained consciousness by the time they reached the Accident and Emergency Department survived. Of 33 children who still had fixed dilated pupils at the time of admission to hospital, one-third died, one-third recovered and one-third survived with a severe neurological defect.)

Orlowski, J.P., Abulleil, M.M. and Phillips, J.M. (1989). The haemodynamic and cardiovascular effects of near-drowning in hypotonic, isotonic or hypertonic solutions. *Ann Emerg Med* **28**: 1044–1049. (Whether a victim drowns in salt water or fresh water has little effect on the haemodynamic effects. The cardiovascular changes that occur with near-drowning and aspiration of water are not dependent on the toxicity of the aspirated fluid but on the direct result of anoxia.)

Pearn, J. (1985). Drowning. In *The Critically Ill Child: Diagnosis and Medical Management.* (J.D. Dickerman, J.F. Lucey eds). Third Edition. W.B. Saunders Co, Philadelphia. Chapter 7: pp. 129–156. (A major overview of all aspects of drowning including epidemiology, pathophysiology, rescue, first aid management, medical management, prognosis and prevention. Includes 176 references.)

Pearn, J. (1985). Pathophysiology of drowning. *Med J Aust* **142**: 586–588. (A major view of the changes in the heart and lungs in the drowning victim. This overview includes detailed discussion of the drowning episode itself, changes in the lungs in the submerged victim, the progress of hypoxia and the onset of brain death. Includes 40 references.)

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This publication is out-of-print and is for historical information and interest only.
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CHAPTER 17 – ELECTRIC SHOCK AND LIGHTNING INJURIES

a Historical

Any of the works of C.F. Dalziel and Theodore Bernstein make fascinating reading and give insight into the parallel development of the science and engineering of electricity with medical aspects of injury from electricity. Many of their works centre around legal execution by electricity and, unfortunate though this may be, penal electrocution has contributed to our knowledge of the effects of electricity on the body. Bernstein's work is more specifically historical, and Dalziel's work, while partly historical, forms the pioneering background of much present thought especially on fibrillation thresholds. See, for example, the following references.

Bernstein, T.

Effects of Electricity and Lightning on Man and Animals. *J For Sc* **18**(1). (1973).

Theories of the Causes of Death from Electricity in the Late Nineteenth Century. *Med Instrum* **9**(6): 267–273 (1975).

(with T.S. Reynolds) The Damnable Alternating Current, *Proc IEEE,* **64**(9): 1339–1343 (1976).

(with T.S. Reynolds) Edison and "The Chair", *IEEE Tech Soc Mag,* **8**(1): 19–28 (1989).

Dalziel, C.F.

A Study of the Hazards of Impulse Currents. *Proc AIEE,* **Oct 1953**: 1032–40.

Electric Shock Hazard. *IEEE Spectrum.* **Feb 1972**: 41–50.

(with W.R. Lee) Lethal Electric Currents. *IEEE Spectrum.* **Feb 1969**: 44–50.

(with W.R. Lee) Re-evaluation of Lethal Electric Currents. *IEEE Trans Ind Gen Applic,* **IGA-4**(5): 467–476. (1968).

b Basic Electrical Theory

The underlying electrical theory necessary to understand simple circuits and electrical quantities is well covered in the following references.

Carlson, A.B. and Gisser, D.G. (1981 *et seq*). *Electrical Engineering: Concepts and Applications.* First Edition. Addison Wesley.

Smith, J. (1984 *et seq*). *Circuits, Devices and Systems.* Fourth Edition. John Wiley and Sons.

c Electrical injuries

Electrical injuries are covered in the following references. Andrews *et al.* provide a comprehensive overview of the topic, and Lee provides specific information on electroporation and its implications. Some of the latter work is quite mathematical especially in *Electrical Trauma.* This work is fairly brief on other pathophysiology and management. *Electrical Injury* is more broad and accessible. It is the better work for a comprehensive overview. Bridges' work is an excellent reference on theoretical matters relating to underlying mechanisms in shock physiology. AS3859 contains a most useful compendium of curves and equations for a wide variety of effects and thresholds for current on the human body.

Andrews, C.J., Cooper, M. and ten Duis, H.J. (1995). The Pathology of Electrical and Lightning Injuries. In *Forensic Sciences.* (Wecht, C.). Bender, NY. Chapter 23A.

Bridges, J.E., Ford, G.L., Sherman, I.A. and Vainberg, M. (1985). *Electric Shock Safety Criteria.* Pergamon Press. (Presently out of print, but library copies will repay perusal.)

Fish, R. (1993). Electric Shock, Part III. *J Emergency Medicine* **11**: 599–603. (Shock is sometimes deliberately applied during electroshock therapy, and with stun guns, cattle prods and shock batons. Regardless of the reason for the shock, electric injuries should be treated to preserve cardiac and respiratory function, and to prevent further tissue damage. Safe extrication at the scene, rapid triage, and emergency medical treatment are discussed.)

Lee, R.C., Cravalho, E.G. and Burke, J.F. (1992). *Electrical Trauma: The Pathophysiology, Manifestations, and Clinical Management.* Cambridge University Press.

Lee, R.C., Capelli-Schellpfeffer, M. and Kelley, K.M. (eds). (1974). Electrical Injury: A Multidisciplinary Approach to Therapy, Prevention, and Rehabilitation. *Ann NY Ac Sc,* Vol 270.

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Standards Australia. (1991). *Effects of Current Passing Through the Human Body, AS3859-1991.* Standards Australia.

d. Lightning injuries

Lightning injuries are covered by the following references. Andrews *et al.* is perhaps the only comprehensive text on the subject, and reviews are provided by Cooper and Andrews, and Andrews *et al.* A useful compendium of statistical material is provided by Lopez *et al.*

Andrews, C.J., Cooper, M., Darveniza, M. and Mackerras, D. (1992). *Lightning Injuries: Electrical Medical and Legal Aspects.* CRC Press, Florida.

Andrews, C.J., Darveniza, M. and Mackerras, D. (1989). Lightning Injuries: A Review of Clinical Aspects, Pathophysiology, and Treatment. *Advances in Trauma.* **4**: 241–289.

Cooper, M.A. and Andrews, C.J. (1994). Lightning and Electrical Injuries. In *Management of Wilderness and Environmental Emergencies.* (Auerbach, P. *et al.).* Third Edition.

Lopez, R.E., Holle, R.L. and Heitkamp, T.A. (1994). *Deaths, Injuries and Property Damage due to Lightning in Colorado from 1950 to 1991 based on 'Storm Data', NOAA Tech. Memdm ERL NSSL-103, NOAA Nat Sev Storms Lab., Norman Oklahoma.*

CHAPTER 18 – EYE INJURIES

Colvin, J., Langford, S., Emonson, D. and Kister, M. (1995). Initial Management and Transport of Patients with Perforating Eye Injuries. *Aust Fam Physician* **24**: 1017–1020.

Fong, L.P. (1995). Eye Injuries in Victoria. *Med J Aust* **162**: 64–68.

Fong, L.P. (1995). Sports-related Eye Injuries. *Med J Aust* **160**: 743–747.

Gregory-Roberts, J. (1992). Pitfalls in Penetrating Eye Injuries. *Med J Aust* **157**: 398–399.

Hammerton, M.E. (1995). Burns to the Eye: an Overview. *Aust Fam Physician* **24**: 998–1,001.

Hammerton, M.E. (1995). Management of Ocular Burns. *Aust Fam Physician* **24**: 1006–1010.

Hirst, L.W., Michels, R.G. and Ilif, W.J. (1984). *Ocular and Periocular Trauma.* (Sights and Sounds in Ophthalmology; Vol 5.) Mosby, St Louis.

Royal Victorian Eye and Ear Hospital. (1995). Lecture Notes on Ophthalmology. East Melbourne.

Shingleton, B.J. (1991). Current Concepts: Eye Injuries (review article). *New Eng J Med* **325**: 408–13.

CHAPTER 19 – FRACTURES

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Frankel, V.H. and Burstein, A.H. (1970). *Orthopaedic Biomechanics.* Lea and Febiger, Philadelphia.

Hatch, R.L. and Rosenbaum, C.I. (1994). Fracture care by family physicians. A Review of 295 cases. *J Family Practice* **38**: 238–244. (This excellent study shows that first aiders and family doctors can expect to encounter fractures of whom (a) 34 per cent will need reduction, (b) 60 per cent will recover completely without any after effects, and (c) 80 per cent will recover with full, good or excellent range of motion after healing. Ten out of 295 (0.3 per cent) had persistent symptoms. Fractures which cause long-term problems are those involving the corist, and those whose fracture causes malalignment and requires reduction.)

Harkess, J.W., Ramsey, W.C. and Bijan Ahmadi (1984). Principles of fractures and dislocations. In *Fractures in Adults* (Rockwood, C.A. and Creen, D.P. eds). J.B. Lippincott Company. pp. 141–146

Watson, J.T. (1994). Current Concepts Review. Treatment of unstable fractures of the shaft of the tibia. *J Bone Joint Surgery* **76A**: 1575–1584. (Using the example of fractures of the shin bone (tibia), a detailed literature review shows the importance of degree and severity of the initial displacement of bones following fracture, the severity of damage to surrounding soft tissues, the degree of comminution at the site of the fracture, the amount of energy

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causing the fracture and the presence of infection at the fracture site. The average time for healing in adults with tibial fractures that were stable and were caused by low-energy mechanisms was twenty weeks; and thirty weeks for unstable fractures caused by high-energy (eg gunshot wounds) mechanisms.)

CHAPTER 20 – HEAT STRESS, DEHYDRATION AND THE HEAT DISORDERS

Editorial comment. (1993). Heat Related Deaths – United States, 1993. *Morbidity and Mortality Weekly (MMWR).* CDC **42**: No. 28

Hales, J.R.S. and Richards, D.A.B. (1987). Principles for the prevention of death from heat stress. In *Heat Stress, Physical Exertion and Environment* (Hales, J.R.S. and Richards, D.A.B. eds). Excerpta Medica, Elsevier Science Publishers, Amsterdam International Congress Series No 773. p. vii.

Hubbard, R., Szlyk, P. and Armstrong, L. (1990). Influence of Thirst and Fluid Palatability on Fluid Ingestion during Exercise. In *Perspectives in Exercise Science and Sports Medicine – Vol 3. Fluid homeostasis during exercise* (Gisolfi, C.V. and Lamb, D.R. eds). Benchmark Press.

Khogali, M. and Mustafa, M.K. (1987). Clinical management of heat stroke patients. In *Heat Stress: Physical Exertion and Environment* (Hales, J.R.S. and Richards, D.A.B. eds). Elsevier Science Publishers, Amsterdam. pp. 499–511.

Khogali, M. (1987). Heat Stroke: An overview with particular reference to the Makkah pilgrimage. In *Heat Stress Physical Exertion and Environment* (Hales, J.R.S. and Richards, D.A.B. eds). Excerpta Medica, Elsevier Science Publishers, Amsterdam International Congress Series No. 773. pp. 21–36.

Lind, A.R. (1983). Pathophysiology of heat exhaustion and heat stroke. In *Heat Stroke and Temperature Regulation.* (Khogali, M. and Hales, J.R.S. eds). Academic Press, Sydney. pp. 179–188.

Ramsay, C., Watson, W. and Robinson, W. (1993). Effect of cooling time on survival in classical heat stroke. *Journal of Wilderness Medicine* **4**: 27–31.

Richards, D.A.B. and Richards, C.R.B. (1987). In *Heat Stress Physical Exertion and Environment.* (Hales, J.R.S. and Richards, D.A.B. eds). Exerpta Medica, Elsevier Science Publishers, Amsterdam No. 773. pp. 419–426.

Richards, R., Richards, D. and Sutton, J. (1992). Exertion-induced heat exhaustion. An often overlooked diagnosis. *Aust Fam Physician.* **21**: 18–24.

Sweeney, W.B. and Krafts-Jacobs, B. (1992). A method for shipboard treatment of multiple heat casualties. *Military Medicine.* **157**(3): 145–147.

CHAPTER 21 – COLD INJURIES: HYPOTHERMIA AND FROSTBITE

Auerbach, P.S. (ed.) (1995). *Management of Wilderness and Environmental Emergencies.* Third Edition. C.V. Mosby. (A massive tome which is virtually the bible of wilderness medicine. Has extensive and very thoroughly referenced chapters on accidental hypothermia, cold water immersion and frostbite and other cold-induced injuries.)

Doyle, F., Zehner, W.J. and Terndrup, T.E. (1992). The effect of ambient temperature extremes on tympanic and oral temperatures. *American Journal of Emergency Medicine* **10**(4): 285–289. (This study demonstrated that tympanic and oral temperatures taken within the first 20 minutes after exposure to outdoor temperature extremes may fail to reflect accurately the patient's true temperature.)

—— (1992). Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care. *JAMA,* **268**(16): 2171–2302. (Contains a section on special resuscitation situations including aspects of both basic and advanced life support of the hypothermic victim. Includes an excellent algorithm for the treatment of hypothermia.)

—— (1995). Hypothermia and Cold Weather Injuries Seminar, Princeton University Outdoor Action Program. (Web page address: http://www.princeton.edu/~rcurtis/hypocold.html.) (This 1995 Internet document is a useful summary of current thinking on the topic, although its use of the Fahrenheit scale with no conversion to Celsius somewhat limits its usefulness.)

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The first aid protocols in this publication may not reflect current practices.

Lloyd, E. (1986). *Hypothermia and Cold Stress.* Croom Helm. (Still one of the definitive texts on the subject and an invaluable source of information which is otherwise difficult to find.)

Rees, W.G. (1993). A new wind-chill nomogram. *Polar Record* **29**(170): 229–234. (Outlines the theoretical work of Siple, Steadman and others on the estimation of wind-chill and the calculation of equivalent temperatures. Describes an improved nomogram for estimating wind-chill and hence assessing the danger of hypothermia and frostbite.)

Ward, M.P., Milledge, J.S. and West, J.B. (1995). *High Altitude Medicine and Physiology.* Chapman and Hall Medical. (Covers the entire range of medical problems associated with mountaineering, with excellent chapters on thermal balance and regulation, reaction to cold, hypothermia and local cold injury.)

CHAPTER 22 – POISONING

Bolgiano, E.B. and Barish, R.A. (1994). Use of new and established antidotes. *Emergency Clinics of North America* **12**: 317–333. (A comprehensive review of the current use of antidotes in the treatment of cyanide poisoning, digoxin overdose, organophosphate and methanol poisoning.)

Henderson, A., Wright, M. and Pond, S.M. (1993). Experience with 732 acute overdose patients admitted to an intensive care unit over six years. *Med J Aust* **158**: 28–30. (This excellent paper gives an overview of the types of drugs and poisons ingested by 1000 consecutive patients who presented to the Emergency Department of a major Brisbane hospital following suicidal overdose, and details of 732 who were admitted for life-support monitoring to the Intensive Care Unit. The mortality of acute overdose is 2.0 per cent. 46.8 per cent of poisoned suicidal patients ingest more than one drug. Factors associated with an increased risk of death are male sex, pre-existing disease, delay in presentation and major complications such as aspiration of vomitus, the toxicity of the drug or chemical ingested and the quantity swallowed.)

Henretig, F.M. (1994). Special considerations in the poisoned pediatric patient. *Emergency Clinics of North America* **12**: 549–566. (A state-of-the-art, comprehensive review of all aspects of the management of poisoned children.)

Kulig, K. (1992). Initial management of ingestions of toxic substances. *New England J Med* **326**: 1677–1681. (An authoritative and definitive account of best current practice in the field of toxicology that are relevant to the crucial first hour after the patient reaches the emergency department.)

Micromedex Inc. (1994). Poisindex® *Toxicologic Management* p. 80 (A comprehensive syndicated, copyright, computerised compendium of all types of human and animal poisoning, giving overviews, life support management, clinical effects, laboratory tests, antidotes and treatment details for many thousands of toxic chemicals and drugs. Address: Poisindex®, Denver, Colorado 80203, USA.)

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CHAPTER 23 – SHOCK

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