**Environmental hazards**

**Clinical problems**

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| ***LEARNING OBJECTIVES*** |
| After studying this module on Environmental hazards, you should be able to:1. Initiate early burns management and identify patients to refer to a specialised unit
2. Recognise and manage associated burn complications such as inhalational injury, cyanide and carbon monoxide exposure
3. Describe principles of management and approach to patients with hypothermia and heat stroke
4. Prioritise and initiate timely treatment of near-drowning/drowning victims
5. Recognise the broad spectrum of pesticide toxicity and implement specific and supportive treatment
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| ***FACULTY DISCLOSURES*** |
| The authors of this module have not reported any disclosures. |
| ***DURATION*** |
| 7 hours |
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**Introduction**

From time to time patients are admitted to the general Intensive Care Unit (ICU) suffering from life-threatening complications of exposure to a variety of environmental hazards. The number of such patients in an individual ICU may be small, but timely and effective treatment may be life-saving. This module deals with management of the individual patient; managing major disaster scenarios is beyond its scope.

**Burns** Most centres will, from time to time, need to deal with the immediate management of burns patients and then organise the safe transport of these patients to a specialised unit. Management of these various conditions can be a major challenge to the ICU team but skilful care is generally rewarded with a good outcome. Complications include cyanide and carbon monoxide poisoning.

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| **Temperature-related injuries** Accidental hypothermia may occur anywhere and during any season. Hyperthermia occurs mainly in hot environments, particularly when humidity is high or when heavy exercise is undertaken by people who are not acclimatised.**Near-drowning/drowning** is a leading cause of accidental death all over the world.**Pesticides** include agents such as insecticides and rodenticides. The production of these compounds has the aim of destroying organisms; so all are potentially harmful to humans. | Human exposure to environmental hazards is frequently accidental, occurs often in the workplace or residence, involves adults and also children, and has variable morbidity and mortality |

# 1/ Burns, including electrical injury

Burns continue to be a leading cause of death world-wide. Thermal injury should not be viewed as an isolated event but rather has effects on multiple organ systems.

**Acute situation**

Burn victims are multiple trauma patients and should be initially evaluated as per standard resuscitative or ATLS guidelines; see reference below. Also published as a series of 12 articles in BMJ, June to August 2004. PMID [15178618](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15178618%5Buid%5D), [15191982](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15191982%5Buid%5D), [15205294](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15205294%5Buid%5D), [15217876](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15217876%5Buid%5D), [15242917](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15242917%5Buid%5D), [15258073](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15258073%5Buid%5D), [15271835](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15271835%5Buid%5D),[15284153](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15284153%5Buid%5D), [15297346](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15297346%5Buid%5D), [15310609](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15310609%5Buid%5D), [15321905](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15321905%5Buid%5D), [15331482](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=DetailsSearch&Term=15331482%5Buid%5D).
See also the website of the American Burn Association
<http://www.ameriburn.org/ABLS/ABLS.htm>

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In addition to their obvious burn injury, patients may have other injuries which contribute to, or complicate the primary burn. There is significant morbidity associated with these injuries if missed at the time of initial assessment. Burns resuscitation and treatment of associated life-threatening injuries such as pneumothorax, haemorrhage and severe head trauma should proceed concurrently. It is important to make an estimation of burn size at this stage as this will help guide immediate fluid resuscitation and prognostication.

## Immediate actions

### *Airway and inhalational injury*

Immediate assessment of the airway is vital when examining any burn patient. All burn patients are potentially at risk of inhalational or upper airway injury, but those at particular risk include patients who were burned in an enclosed space, those who were asleep or under the influence of drugs or alcohol and those who have, for example, associated head injuries.

Inhalation of hot gases can produce burns as far as the terminal bronchi. Smoke injury is common, can extend more distally, and is a result of corrosive damage by chemical products produced by the fire.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s103.jpg | **History, signs and symptoms of an inhalational injury** |

If the history or examination is strongly suggestive of inhalational injury, prompt tracheal intubation may be life-saving. A delay in securing the airway can lead to a difficult or impossible intubation at a later stage when massive facial and upper airway oedema can develop. Suxamethonium can be used safely to secure the airway only on initial presentation. Use of suxamethonium at a later stage can lead to life-threatening hyperkalaemia. For further information on the pathophysiology and specific management of inhalational injury see the references below and the PACT module on Airway management .

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| **Think** | It is a lot easier to extubate a patient who subsequently demonstrates only minor or no airway injury, than it is to intubate a patient with previously unrecognised inhalational burns who now has a compromised and difficult airway. |

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| **Note** | **While all endotracheal tubes should be well secured, it is especially important in these patients as, if accidentally dislodged, it may be impossible to reinsert.** |

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| **Caution** | **Do not cut the endotracheal tube to an 'oral length'. Patients with facial and inhalational burns commonly develop marked oedema and if the tube is cut, it may ultimately become too short and be difficult to secure and/or be dislodged.** |

### *Circulatory resuscitation*

The next step is to insert two large bore cannulae, preferably through non-burned skin. In practice, it may be necessary to place them through burned skin or centrally. Delay in intravenous resuscitation increases mortality. Two litres of normal saline should be commenced immediately, pending an initial estimate of burn size. In moderate and severe burns, once the patient is stable, a central venous catheter should be inserted before the development of oedema makes the procedure much more difficult, preferably before the patient is transported to a specialised centre.

### Tailoring initial fluid therapy

Resuscitation volumes in the first 24 hours are based on clinical assessment and on analysis of the circulatory response to fluid therapy. Formulae incorporating the total body surface area (TBSA) provide a guide to expected fluid resuscitation requirements. There are numerous formulae available, some of which use crystalloid only and some of which use a combination of crystalloid and colloid. To date there have been no definitive trials demonstrating a survival benefit of one formula over another. The most commonly used formulae include the Parkland and the Modified Brooke regimens. The presence of inhalational burns increases fluid requirements by at least 25%.

### *Assessment of burn injury*

Burn injuries are classified by extent, depth, circumferential components and the presence or absence of inhalational injury. Several charts are available to estimate the extent of TBSA burned. In adults, the 'Rule of Nines' can be used to quickly estimate the size of burn. However use of a Lund-Browder diagram, which is an age-specific chart that accounts for changing body proportions with age, will give a more accurate estimation in all patients, but is particularly important in children. Small or irregular areas of burn can be estimated using the palm of the patient's hand as a guide. The area of the palm, including the digits is approximately 1% of TBSA (0.5% of TBSA, if the digits are excluded).

You will find diagrams for the Rule of Nines and Lund-Browder on the following website.

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In addition to the extent of burn, the burn depth is carefully assessed. Depending on the structures involved, depth is classified as partial or full thickness, or as in the table, below.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s109.jpg | **Classification of burn depth** |

Circumferential or near circumferential burns are noted. At best they need close monitoring, but frequently urgent escharotomy or fasciotomy is required.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s110.jpg | **Example of 1st and 2nd degree burns**Picture courtesy of Anne Berit Guttormsen |

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| **Note** | **Burn depth and size may not be initially clear and are often underestimated even by experienced clinicians. Regular reassessment is vital. The full extent and depth are often not clear for up to 48 hours post injury.** |

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s111.jpg | **Fluid resuscitation formulae** |

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| **Activity** | **Find out the adult and paediatric burns resuscitation protocols used in your hospital.** |

### Continuing fluid management

These formulae are guidelines only. Burns patients must be carefully and repeatedly reassessed to ensure adequacy of resuscitation. The amount of fluid required will vary between patients depending on pre-existing conditions, co-existing injuries and the severity of the inflammatory response. A central venous pressure trend, if available, will be helpful, as will serial lactate measurements, and regular urea, creatinine and haematocrit estimations. Adequate resuscitation can only be ascertained by regular monitoring of many physiological variables. No single variable is a reliable indicator.

Goals include:

* Warm peripheries
* Heart rate and blood pressure appropriate for age and for the patient
* Urine output 1.0 ml/kg/hr
* Absence of metabolic acidosis
* Normal level of consciousness

The benefit of pulmonary artery catheters and pulse contour analysis techniques in the management of patients with severe thermal injuries remains unproven. The initial problem is largely related to hypovolaemia, and careful monitoring of physiological variables, including CVP if necessary, is usually sufficient. Patients who have other major injuries, or those who have significant co-morbidities such as cardiac disease, and those who develop multi-organ complications could potentially benefit from more intensive monitors such as pulse contour analysis or pulmonary artery catheter.

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| **Note** | **Ongoing unexplained fluid requirements or persistent hypotension should raise the suspicion of unrecognised associated injuries, missed inhalational burn, associated poisoning, or other complications such as myocardial infarction or sepsis.** |

## Treatment

### *Specific treatment*

Superficial burns usually heal within one to two weeks. Burns of intermediate depth require careful monitoring and dressing by experienced personnel. A proportion will fail to adequately heal and will need excision and grafting.

The morbidity and mortality associated with deep burn injury has been significantly reduced in recent times by early surgical intervention. Removing dead burned tissue removes a source of ongoing inflammation and sepsis, and exposes a clean and viable wound bed. Ideally the burn wound is closed as soon as possible using split-skin grafts harvested from non-burned areas of the patient's body. However, in massive thermal injury there may be insufficient non-burned skin available, and the use of homografts or artificial skin substitutes may be necessary. Successful closure may require multiple operations over a prolonged period of time.

Treatment of burn injuries requires a coordinated multidisciplinary team approach. The type of personnel required varies depending on the severity of the burn, associated injuries, and complications. Further management of the burn patient is supportive and active. If the patient is not in a centre incorporating a major burns unit, a decision on transfer to a burns facility is made as soon as the patient has been assessed, resuscitated and immediate life-threatening issues addressed. This is done in conjunction with a specialist burns team.

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| **Note** | **Prior to transfer the question of airway security should be readdressed. Intravascular fluid resuscitation must continue throughout.** |

You will find the American Burn Association criteria for referral to a burns centre on the following website [Verification/Guidelines].

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It is vital that a full and accurate trauma and resuscitation history is communicated to the accepting team, and that they are kept up to date with the patient's progress. Competent critical care staff must accompany the patient. See the PACT module on Transportation for further information on transfer of critically ill patients .

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| **Note** | **Significant intra-operative fluid loss is not uncommon in this group of patients. Haemorrhage may be complicated by coagulopathy, requiring transfusion of blood and blood products. Care must be taken to limit the potential for hypothermia.** |

Several surgical precautions such as soaking debrided areas with epinephrine-soaked pads, infiltrating skin graft harvest sites with epinephrine solution may help to reduce blood loss.

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| **Activity** | **Find out your hospital guidelines for the management of massive haemorrhage.** |

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| Question Why might hypothermia occur? What measures are indicated to prevent intra-operative hypothermia? |

### *General treatment*

### Supportive – comparable with any inflammatory, multi-organ insult

Multi-organ failure remains a common complication of severe burn injury. This summary emphasises burn-related considerations.

### Cardiovascular system

Complications include dysrhythmias, myocardial ischaemia and cardiac failure. The systemic inflammatory response seen with severe burn injury often necessitates the use of vasopressors after adequate volume resuscitation, even in patients with normal hearts.

### Respiratory system

Pulmonary complications are frequently encountered in burn injury patients, and include immediate problems associated with direct injury at the time of initial exposure and later complications such as nosocomial pneumonia. Acute lung injury and ARDS require ventilatory support. Although a minority may overcome respiratory difficulties with non-invasive ventilation alone, the majority will require intubation and invasive ventilation. Tracheostomy is not uncommonly needed in severely burned patients on prolonged ventilatory support, particularly in those with facial burns or severe inhalational injury. However the role of early tracheostomy is controversial especially if the burn involves the anterior neck.

### Renal

Early and vigorous fluid resuscitation is vital. Timely escharotomies reduce the risk of limb ischaemia and rhabdomyolysis.

### Gastrointestinal / Nutritional

Institute early enteral nutrition via the nasogastric, or if necessary, nasojejunal route. Occasionally, total parenteral nutrition (TPN) is necessary to maintain nutritional requirements, however it is generally avoided in favour of enteral routes where possible.

Energy requirements are high due to the hypermetabolic response depending on the extent of burn injury (up to 200% of normal metabolic rate). There are formulae to calculate caloric requirements, e.g. 25 kcal/kg/day + 40 kcal/%TBSA burned/day (Currieri). At least 1-2 g/kg/day of protein should be given.

Supplementation with glutamine, arginine and omega 3 fatty acids may be helpful. Anabolic agents such as growth hormone, insulin, and oxandrolone and propranolol have been used in attempts to decrease loss of lean body mass and to accelerate wound healing and shorten hospital stay. To date there are few large clinical trials demonstrating benefit, however some smaller studies have supported their use in moderate and severely burned patients.

Although growth hormone may increase mortality in the majority of critically ill patients, (Takala reference, below), administration may be beneficial in severely burned patients, particularly paediatric patients. For more information see the following references and the PACT module on Nutrition .

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### Infection / Sepsis

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| **Note** | **Sepsis is the major cause of death in patients who survive the initial burn injury.** |

This is a major cause of morbidity and mortality in those who survive the initial burn insult. Pulmonary infections are the most common, especially in patients with inhalational injury or those on mechanical ventilation. Burn site infection may occur due to breakdown of the skin or mucosal barrier and the presence of exudate and necrotic tissue. The presence of invasive monitoring devices and immune suppression also predispose to infection. Gram-negative organisms such as pseudomonas are common pathogens as are resistant Gram-positive organisms such as MRSA and VRE. Fungal infection may become a difficult problem later.

Interpretation of surface cultures may be confounded by extensive colonisation, and blood cultures are frequently positive after dressing changes. Detection of sepsis may be made difficult as the common signs of infection such as pyrexia, leukocytosis, and elevated markers of inflammation are frequently seen in burn patients with no infected tissues. Accurate diagnosis depends on a combination of the clinical picture, multiple surface and tissue cultures and blood cultures. It is important not to give prophylactic antibiotics but to give short courses of narrow-spectrum antibiotics targeted to clinical sepsis and based on microbiological results.

Peri-operative antibiotic use for wound debridement and grafting remains controversial, but is commonly administered for 24 hours post-procedure. A longer therapeutic course may be indicated if the burn wounds are purulent; the agent choice is guided by recent (surveillance) culture results. For more information see the PACT modules on Sepsis and MODS  and Severe infection 

### Neuropsychological

Analgesia requirements in the acutely burned patient may be very high, particularly when frequent dressing changes are necessary. Chronic pain issues may become a difficult problem, especially if treatment involves amputation surgery. Addiction to analgesics, usually opiates, may develop.

Many burned patients are left with significant scarring and deformities. In addition they frequently have post-traumatic stress disorders and phobias and will need intensive psychological and possibly psychiatric support with input from appropriate allied health professionals. Self-inflicted burn injury is not uncommon and the underlying psychological or psychiatric illness will need to be addressed.

### Musculoskeletal

Contractures and amputations will require psychological support as above, also intensive long-term physiotherapy and occupational therapy.

## Specific presentations/complications

### *Electrical injuries*

Electrical injury can occur in isolation, or in association with thermal injury. Burn casualties with associated electrical injuries should be referred to a major burns unit.

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Severe electrical injuries, excluding lightning strikes, tend to occur in the workplace. Injuries are as a direct result of the current passing through the body and secondarily as a result of prolonged muscle contractions stimulated by the current. An electrically injured patient should be managed as a multiple trauma patient. For more information see the PACT module on Multiple trauma 

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| **Caution** | **Rescuer safety, always a priority, is especially important when treating these patients. Always ensure the electricity source has been made safe.** |

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| **Think** | Prolonged muscle contractions in these patients can result in secondary fractures and dislocation injuries, including injuries to the vertebral column. Full spinal precautions are indicated. |

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| **Note** | **Immediate death from electrical injury occurs as a result of ventricular fibrillation, asystole, or because of respiratory arrest secondary to central nervous system damage or sustained respiratory muscle contraction.** |

The severity of the injury sustained depends on the following:

* Voltage encountered
	+ >1000 V: high voltage injury
	+ 220-1000 V: intermediate voltage injury
	+ <220 V: low voltage injury
* Resistance of the patient (e.g. moisture will reduce the resistance)
* Route taken through the body
* Duration of contact

Low and intermediate voltage injuries may be complicated by local tissue destruction but are not commonly associated with multisystem damage. High voltage injuries can be complicated by severe burns, myocardial necrosis, cardiac dysrhythmias, central and peripheral nervous system damage, visceral injuries, limb ischaemia, compartment syndromes, rhabdomyolysis, and ultimately multiple organ failure. The extent of injury may not be initially evident, as the injury at the entry and exit points may not appear very severe. There should be a low threshold for exploring muscular compartments.

Serial estimations of creatine kinase and frequent measurement of potassium levels may help in detecting rhabdomyolysis.

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| **Note** | **Symptomatic patients (including those with loss of consciousness), those with abnormal ECG or a history of cardiac disease, and all patients exposed to intermediate and high voltages should have continuous cardiac monitoring for a minimum of 24 hours post injury.** |

There is no specific therapy for electrical injuries. Management is symptomatic and supportive only.

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| *Inhalational injury*Inhalational burn is primarily a clinical diagnosis based on history, symptoms and signs. Chest X-ray and pulse oximetry may be entirely normal in the presence of significant airway injury. Bronchoscopy can confirm the diagnosis but a normal examination does not exclude a more distal injury. However thorough and repeated bronchoscopy and lavage can help remove the soot, debris, casts and mucous plugs that complicate airway burns. These patients can rapidly develop respiratory failure and persistent hypoxaemia despite optimal conventional ventilatory strategies. High frequency oscillatory ventilation has been used with success in difficult patients. There has been renewed interest in using nebulised heparin and fibrinolytics in this group of patients, but there are no large human trials demonstrating benefit to date.

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The pathophysiology of inhalational injury is complex. In addition to direct thermal injury, the patient may inhale particulate products of combustion and be exposed to various toxins, depending on the circumstance of the fire. Two common toxins – cyanide and carbon monoxide, require specific management.*Carbon monoxide poisoning*Immediate assessmentAlthough not always associated with burn injury, a high index of suspicion should always be maintained for carbon monoxide (CO) poisoning, particularly in high-risk injuries such as burns suffered in enclosed spaces, in patients who had burns in association with alcohol, drug use or any injury which resulted in a reduced level of consciousness. CO poisoning is responsible for many early deaths in burn victims. It is also seen in isolation, for example as a result of faulty boilers, poor ventilation, and in patients who have attempted suicide. Immediate treatmentThe management of any suspected CO poisoning must include the administration of supplemental oxygen at as high a concentration as possible. CO has an affinity for haemoglobin 250 times that of oxygen. The administration of 100% oxygen reduces the half-life of carboxyhaemoglobin from 4-6 hours to 60-90 minutes.

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| **Note** | **All burn patients should be given supplemental oxygen pending availability of carboxyhaemoglobin levels.** |

Definitive diagnosisCarbon monoxide poisoning requires blood gas analysis by CO-oximeter, which will give accurate measurements of oxyhaemoglobin, carboxyhaemoglobin and methaemoglobin. Carboxyhaemoglobin levels can be measured on arterial or venous blood. Arterial blood gas analysis using the usual blood gas machines may only demonstrate a metabolic acidosis.

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| **Think** | Oxygen saturation by pulse oximetry will be normal, even when there is tissue hypoxia due to severe CO poisoning. |

 Ongoing treatment100% oxygen continued pending carboxyhaemoglobin levels. Oxygen can be administered to a spontaneously breathing patient via a non-rebreathing mask.However, patients who have moderate or severe CO poisoning frequently require intubation to maintain their airway because of the neurological effects of CO poisoning. In addition these patients may need aggressive cardiovascular support. While the administration of 100% O2 to CO poisoned patients is mandatory, whether it should be given via a hyperbaric oxygen chamber or in normobaric conditions remains controversial. Hyperbaric oxygen in this setting may help prevent permanent neurological damage, but is not without risks, and is not universally and easily available.As CO binds even more avidly to fetal than adult haemoglobin, infants and pregnant patients present further management difficulties. On balance, most clinicians would consider hyperbaric oxygen therapy in the following circumstances:* Altered mental state
* Loss of consciousness or history of loss of consciousness
* Focal neurological deficits
* Seizures
* Pregnancy with carboxyhaemoglobin levels >15%

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| **Caution** | **The patient must be given 100% O2 while a treatment plan is formulated and throughout transfer to a hyperbaric oxygen therapy (HBOT) facility, if this is deemed necessary.** |

The following references review studies of hyperbaric oxygen use in carbon monoxide poisoning.

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Understanding CO poisoningCarbon monoxide acts by binding tightly to haemoglobin, displacing oxygen, and ultimately resulting in cellular hypoxia and acidosis. It may also have a direct cellular toxic effect. Symptoms vary depending on the severity of the poisoning.OutcomeSurvivors of severe CO poisoning are at risk of residual neurological deficit ranging from persistent vegetative state to subtle deficits detectible only on specific neurological testing. The onset of neurological dysfunction may be delayed and follow apparent initial recovery. Delayed neurological sequelae (DNS) have reportedly occurred many days or weeks after an apparent full recovery.*Cyanide poisoning*Although normally present in the body at low concentration, cyanide is a deadly poison which causes tissue hypoxia. It is found in some plants, household and industrial fires, and in reagents used in numerous industrial chemical processes. Iatrogenic intoxication may rarely occur after nitroprusside administration. Cyanide poisoning is discussed here because the most common cause of intoxication is exposure to the combustion of domestic or industrial products (wool, silk, plastic and other synthetic products). It should be considered in all burn casualties.Secondary acute respiratory failure and circulatory collapse are the leading causes of ICU admission. A variety of CNS disturbances are almost always present.

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| **Think** | of hydrogen cyanide intoxication in all fire victims complicated by inhalation injury even if other causes of hypoxia are more likely, e.g. low ambient oxygen level, carbon monoxide poisoning or methaemoglobinaemia. |

The acute situationFeatures of acute poisoning are related to the dose: small doses cause anxiety, dizziness, headache, drowsiness, tachycardia, dyspnoea. This may progress to confusion, agitation to seizures and coma, pulmonary oedema to respiratory arrest. Larger exposure causes hypotension, bradycardia and cardiovascular collapse. Abdominal pain, nausea and vomiting ensue after ingestion.Immediate actionsEmergency measures for all cyanide intoxications include administration of high flow oxygen; if the victim is unconscious or unable to maintain the airway s/he should be intubated. After the introduction of an intravenous catheter, adequate fluid resuscitation should be given. Seizures (diazepam, lorazepam, phenytoin) and coma (dextrose, thiamine and naloxone) are treated. Vital signs and ECG are monitored. Compensation of metabolic acidosis is attempted with titrated hyperventilation, and sodium bicarbonate may be considered. See the PACT module on Altered consciousness http://pact.esicm.org/courses/TemplateV2/images/go.gif.

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| **Caution** | **Caregivers should wear chemical-protective clothes with positive-pressure breathing apparatus when they are at risk of being contaminated. Any contact with salts or solutions containing cyanide or inhaling hydrogen cyanide gas from vomitus must be avoided. Contaminated clothes are removed and bagged.** |

The victim's skin is washed with soapy water if there was skin contact. Whenever ingestion of cyanide may have occurred, a gastric tube should be placed. Gastric lavage is then performed if ingestion occurred within one hour of presentation. Activated charcoal is administered at a dose of 1 g/kg.Clinical diagnosis1-Symptoms of cyanide poisoning are non-specific and vary greatly in onset. 2-The characteristic bitter almond smell may not be detectable, and the history of possible exposure to cyanide is fundamental for the diagnosis.3- Sources of cyanide include reagents used in industrial chemical processes (photography, precious metal extraction, fumigation, electroplating) usually as calcium, potassium or sodium salts; plants (cyanogenic glycosides: apricot, apple, pear, plum, cherry seeds, bitter almonds, bamboo sprouts, cassava beans); acetonitrite (used in some artificial nail-glue removers) and the vasodilator sodium nitroprusside. 4-possible diagnostic clues are the absence of cyanosis and increased oxygen saturation in venous blood, due to blocked cellular oxygen consumption. Arterial blood gases show normal levels of oxyhaemoglobin and metabolic acidosis with an increased anion gap. Lactic acidosis may develop due to intoxication, but also due to hypovolaemia. A plasma lactate concentration of 72 mg/dl (8 mmol/l) was found to correlate with blood cyanide levels greater than 1 mg/l.

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Cyanide levels are not available in time to influence initial emergency management; however a sample should be taken before antidote administration. Whole blood level >0.5-1 mg/l is considered toxic; levels up to <0.2 mg/l are seen in cigarette smokers. Symptoms may correlate to serum level:1. 0.5-1 mg/l –flushing and tachycardia;
2. 1-2.5 mg/l – obtundation;
3. 2.5-3 mg/l – coma and respiratory depression;
4. >3 mg/l – death. Cyanide concentration may also be measured in gastric contents, urine and tissues.

Metabolic acidosis may be associated with a cyanide level of up to 1 mg/l after rapid infusion of nitroprusside. In addition, arterial blood gas, glucose, electrolytes, serum lactate, carboxyhaemoglobin and methaemoglobin level should be measured.Specific antidotal therapy1-Hydroxocobalamin, Drug definitionwhich is associated with very little toxicity, is in some countries the antidote of choice. The dose is 5000 mg in 200 ml given over 15-20 min. It displaces cyanide from the cytochrome oxidase and forms cyanocobalamin that is then excreted in the urine or metabolised by hepatic rhodanese.2-a cyanide antidote package consists of amyl and sodium nitrites Drug definition and sodium thiosulfate Drug definition. The amyl nitrite capsule should be crushed in a gauze sponge and inhaled intermittently. Once i.v. access is obtained, 300 mg of sodium nitrite Drug definition is given followed by 12.5 g of sodium thiosulfate Drug definition. (Paediatric dose: 0.33 ml/kg of 10% sodium nitrite and 1.65 ml/kg of 25% sodium thiosulfate; dose reduced if anaemic).Nitrites oxidize haemoglobin to methaemoglobin, which has a higher affinity for cyanide than cytochrome oxidase, forming cyan-methaemoglobin. Hepatic rhodanese metabolises this into thiocyanate, using sulphur from the thiosulfate and regenerating methaemoglobin.Understanding cyanide toxicity1-Cyanide, a very potent poison, is a chemical asphyxiant, which inhibits many enzymes, including cytochrome oxidase and causes acute profound tissue hypoxia.2-It reversibly inhibits oxidative phosphorylation, rendering energy production ineffective. 3-Metabolic acidosis is the result of anaerobic metabolism and lactic acid production. 4-Tissues do not extract oxygen from the blood and this leads to the arterialisation of venous blood. Thiocyanate is the less toxic product excreted in the urine, resulting from the metabolism of the unbound cyanide.Cyanide toxic effects depend on the dose (lethal dose is as little as 200 mg after ingestion, 150-200 ppm by inhalation), on the route of ingestion (it is also easily absorbed through the intact skin) and on duration of exposure. The onset of symptoms after skin contact may be rapid; after ingestion of cyanide salts (potassium or sodium cyanide) it may be as rapid as ten minutes or as long as two hours; inhalation of hydrogen cyanide produces symptoms and even death in a few minutes.OutcomeAlthough some patients have survived potentially lethal doses of cyanide with only non-specific supportive care, with antidote therapy survival has been reported with much higher blood concentrations of cyanide.**Understanding burn injury**1-Initial 'burn shock' is followed by a hyperdynamic catabolic state which can persist for several days. 2-Patients with extensive burns are also immunocompromised because of loss of skin tissue, allowing direct invasion of tissues, and because of altered lymphocyte, neutrophil and cytokine function. For more information see the PACT module on Immunocompromised patients http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/media/go.gif.**Goals during this time include**:* Early wound excision and closure
* Prevention, diagnosis and treatment of sepsis
* Nutritional support of the hypermetabolic state

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| **Note** | **Removal of the burned tissue and wound closure as soon as possible is vital.Burned tissue is an ideal culture medium for bacteria and fungi, but even if not infected, the presence of burn tissue is an excellent source of inflammatory mediators and will support an ongoing systemic inflammatory response.** |

**Long-term management of burn patients**Depending on the extent and location of the thermal injury, burn patients can require surgical and non-surgical treatment for many months or even years, long after the acute phase has resolved.Potential issues include:* Further non-urgent, reconstructive surgery
* Scar management
* Ongoing physiotherapy
* Occupational therapy
* Rehabilitation
* Psychological support/therapy

**Outcome**Major advances in burn care over the past several years have led to a significant improvement in morbidity, mortality and functional outcome – now patients with close to 100% burns survive. Elderly patients have a far greater mortality. Associated inhalation injury continues to be a challenge and is a major predictor of morbidity and mortality in all age groups.Factors which have contributed to this improvement include vigorous fluid resuscitation, early excision and closure of burn wounds, greater understanding of the pathophysiology and complications of burn injuries, and advances in critical care, nutrition, and in the management of sepsis.The establishment of specialised burn centres has contributed to the advances in burn management. The importance of a committed, specialised, multidisciplinary team in achieving an ultimate quality outcome cannot be overstated.

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| 2/ Temperature-related injuries**Hypothermia**Without adequate protection, people at the extremes of age, victims of trauma, the homeless and those who have ingested alcohol, barbiturates, benzodiazepines or other central nervous system depressants are more susceptible to hypothermia in cold environments.Hypothermia can range from a relatively minor complication of outdoor pursuits to an overwhelming multisystem or even fatal process. Accidental hypothermia, defined as inadvertent decrease of core temperature to below 35 °C (95 °F) is one of the commonest environmental emergencies seen in Emergency and Critical Care Units.While it is usually encountered during colder winter months and in countries with severe cold climates, it may occur in warmer temperatures, particularly in patients with underlying medical conditions or social circumstances which predispose to hypothermia. In healthy individuals, it is a leading cause of death during outdoor pursuits, commonly associated with immersion and near-drowning (see Task 3 [http://pact.esicm.org/courses/TemplateV2/images/go.gif](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT3)).**Hypothermia: predisposing factors/conditions**See the following references.

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| **Note** | **Patients with indoor hypothermia have a higher mortality than those presenting with hypothermia as a result of exposure to cold weather conditions, largely because this subset of patients often have underlying medical conditions predisposing them to hypothermia.** |

*Classification of severity*Hypothermia is classified as mild, moderate and severe, based on the degree to which the core temperature has dropped from normal. This has implications for resuscitation.

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| **Note** | **Deterioration from mild to severe hypothermia can be rapid and overwhelming.** |

Mild hypothermia is classified as a core body temperature between 32 and 35 °C (90 and 95 °F). In normal individuals shivering occurs when the core temperature is reduced by 0.7 °C (1.3 °F), which can increase the basal metabolic rate by a factor of 5. When the body temperature reaches 35 °C (95 °F) there is uncontrolled shivering and profound peripheral vasoconstriction. Catecholamine surges result in tachycardia, hypertension and increased cardiac output.

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| Question These haemodynamic changes can result in a 'cold diuresis'. What is the mechanism and what implications could this have during subsequent resuscitation efforts? |

 Moderate hypothermia occurs at temperatures of 28 to 32 °C (82 to 90 °F) and is characterised by loss of shivering due to depletion of glycogen stores, muscle joints become stiff and the patient becomes increasingly lethargic, drowsy and hyporeflexic. In contrast to mild hypothermia, the pulse, blood pressure and respiratory rate are usually depressed.Severe hypothermia is defined as a core body temperature less than 28 °C (82 °F) and is associated with severe metabolic derangement. These patients are deeply unconscious, areflexic, with rigid limbs and may have fixed dilated pupils. Profound hypotension is common, as are cardiac dysrhythmias. Bradycardia and atrial fibrillation occur at temperatures below 30 °C (86 °F), ventricular fibrillation can occur at temperatures below 28 °C (82 °F) and asystole is frequent when the core temperature drops to 20 °C (68 °F) or below.Respiratory frequency may be reduced to 1-2 breaths/min and bronchorrhoea becomes a problem. Essentially, on initial examination, life may be difficult to detect.

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| **Caution** | **Surprisingly, peripheral cyanosis may be less obvious as the patient progresses to severe hypothermia. Reliance on this sign for the diagnosis of hypothermia may lead to error.** |

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| **Note** | **Unconsciousness is uncommon at core temperatures greater than 28 °C (82 °F). Another cause of coma should be sought in an unconscious patient with only mild or moderate hypothermia.** |

*Immediate actions*Assessment and treatment must occur simultaneously. The initial primary survey is conducted as usual. Many of these patients are multiple trauma victims and must be treated as such to limit further injury. This may include application of a cervical collar and spinal board if not already in situ. Airway, breathing, circulation and neurological state need careful assessment and support as required. Minimising the potential for further heat loss is a priority. For more information see the PACT module on Multiple trauma http://pact.esicm.org/courses/TemplateV2/images/go.gif.

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| **Think** | Moving the patient out of cold conditions is an important first step. Wet clothing will add to ongoing hypothermia and should be removed as soon as the patient is in a warm environment. |

*Clinical diagnosis*Diagnosis of hypothermia depends firstly on recognising the possibility of hypothermia and secondly on the use of an appropriately placed low reading thermometer.

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| **Caution** | **Most standard thermometers only read to a low of 34 °C (93.2 °F) and can lead to a falsely high body temperature reading.** |

The commonly used tympanic membrane thermometers have not been proven to be reliable in this setting. Nasopharyngeal temperature probes are probably the most accurate but rectal thermistor temperature probes are more commonly used. Bladder temperature monitors may be another alternative. The importance of accurate diagnosis cannot be overstated. Patients with temperatures of as low as 14.2 °C (57.6 °F) (infant) and 13.7 °C (56.7 °F) (adult), in cardiopulmonary arrest have been successfully resuscitated, following more than four hours of CPR, with a return to normal neurological function.Frequently the history and physical findings obviously suggest the presence of hypothermia. However, patients regularly present with histories and examinations that do not immediately suggest hypothermia.

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| **Note** | **It is vital to recognise that hypothermia can occur in any setting in which the ambient temperature is below the core body temperature and that signs and symptoms may be vague and non-specific.** |

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| Question In cardiac arrest patients, the probability of a good neurological outcome decreases rapidly as time to return to spontaneous cardiac output increases. Why is full neurological recovery still possible in severely hypothermic patients? |

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| **Caution** | **Physical signs in hypothermic patients may be very misleading. If the possibility of hypothermia is not considered, these patients will not be optimally managed. A temperature of 34 °C (94 °F) in a patient who is about to be declared dead from unknown cause should ring alarm bells, as this is the lower limit of reading in commonly used thermometers.** |

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| **Activity** | **Find out what is available in your emergency department to measure low temperatures.** |

*Treatment*Having made the diagnosis, further management will include resuscitation, rewarming and the search for an underlying cause, if not already evident.

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| **Note** | **The method of rewarming chosen is determined by the severity of hypothermia, the haemodynamic status of the patient, available resources and the response to initial rewarming efforts.** |

Moderate to severe hypothermia is a medical emergency, requiring varying levels of support of the airway, breathing and circulation. Moderately hypothermic patients frequently, and severely hypothermic patients always, need intubation, protection, and support of their airway and oxygenation. Muscle rigidity of the jaw can make orotracheal intubation difficult. Bronchorrhoea can be a major problem, necessitating frequent suctioning. Ventilation may also be compromised by chest rigidity and reduced diaphragmatic compliance.Because of the risk of arrhythmia, patients should have continuous monitoring and be moved only with extreme caution. Sedatives, analgesics and/or local anaesthetic spray can be used to minimise the stress of intubation which may even precipitate ventricular fibrillation. Peripheral, and, or central venous access is vital, as is invasive arterial pressure monitoring.

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| **Caution** | **If not already cardiovascularly compromised, resuscitation and rewarming may precipitate haemodynamic collapse. Appropriate intravenous fluids and inotropic support must be available as rewarming commences.** |

RewarmingThere are three methods of rewarming:* Passive external rewarming
* Active external rewarming
* Active internal rewarming

**Passive external rewarming** means the patient is rewarmed by endogenous heat production and it is achieved by placing blankets and metallo-protective sheets around the patient. Temperature increases are typically of the order of 0.5-1.0 °C (0.9 to 1.8 °F)/hr. Passive rewarming is usually adequate treatment for mild hypothermia but may be insufficient if the patient is unable to shiver because of exhaustion or general debility.**Active rewarming, external and internal,** means that the patient is rewarmed by the application of external or internal heat. Active rewarming should be used if the temperature is less than 32 °C (90 °F), if the patient is haemodynamically unstable, in respiratory failure or has failed to improve with passive rewarming.**Active external rewarming** involves the use of heated blankets, forced air warmers, and radiant heaters. These techniques can achieve rises of 1-2 °C (1.8 to 3.6 °F)/hr and are suitable for moderately hypothermic patients or can be combined with active internal rewarming in severely hypothermic patients.**Active internal or core rewarming** is used in patients with severe hypothermia. These methods produce rewarming rates between 3-12 °C/hr (5.4 to 21.6 °F/hr) depending on the methods chosen:* Inhalation of warmed, humidified air (40 to 45 °C or 104 to 113 °F)
* Warmed intravenous fluids (40 to 42 °C or 104 to 108 °F)
* Warm fluid lavage of body cavities including gastric, pleural, bladder, colonic and peritoneal cavities. Lavage of the stomach, bladder or colon has limited utility because of the small surface area available to conduct heat.
* Extracorporeal shunt rewarming. Continuous arterio-venous or more commonly veno-venous haemodiafiltration using high flow rates and warm fluids has become the method of choice in many centres for active rewarming in the non-cardiopulmonary arrest, severely hypothermic patient.
* Cardiopulmonary bypass is the most effective active internal rewarming, and is the choice, if available, for the hypothermic patient who is in cardiopulmonary arrest. This method will provide warming at rates of 8-12 °C/hr (14.4 to 28.8 °F/hr) as well as oxygenation and perfusion of vital organs.
* Central venous heat exchange catheters are now available which work by circulating sterile cool or warm saline through the helically wound heat exchange balloon placed in the inferior vena cava. These can be used to warm the patient precisely at the desired rate.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s217.jpg | Complications of rewarming |

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| **Note** | **Sepsis is both a precipitant and a complication of hypothermia, therefore an extensive microbiological screen is vital. Empirical antibiotics, when clinically indicated, should include broad-spectrum Gram-positive and Gram-negative cover of the most likely pathogens, e.g. lower respiratory tract infections or aspiration pneumonia, unless another obvious source of infection is apparent. For more information see the PACT module on Sepsis and MODS *http://pact.esicm.org/courses/TemplateV2/images/go.gif*** |

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| **Note** | **A toxic screen may reveal exposure to recreational drugs or a high ethanol levelwhich may have contributed to the hypothermia or may help explain an inappropriate neurological state.** |

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| Question What haemodynamic complications may be seen with the use of active rewarming? |

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s219.jpg | **Hypothermia: physiological responses** |
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Other complicationsComplications in those who survive the initial insult include neurological damage, rhabdomyolysis, multiple organ failure, coagulopathy, compartment syndromes and local cold-induced injuries, including frost-bite which may require surgical intervention, amputation and reconstructive procedures.

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| **Think** | Always check blood glucose levels in hypothermic patients. Glycogen stores may have been profoundly depleted and hypoglycaemia may be contributing to ongoing neurological state/damage. |

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| **Note** | **Malnourished patients, especially alcohol abusers are at increased risk of hypothermia and may need aggressive vitamin and electrolyte replacement.** |

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| **Caution** | **The possibility of adrenal insufficiency or hypothyroidism should be considered, particularly in a patient who develops hypothermia in a relatively warm environment or who fails to respond to what appears to be adequate rewarming efforts.** |

*Hypothermic cardiac arrest*On initial evaluation the severely hypothermic patient may appear to be lifeless.Pupils can be unreactive, respiratory effort absent and extreme peripheral vasoconstriction in combination with profound bradycardia will make cardiac output detection difficult.

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| Question How long should you palpate for a pulse in a hypothermic patient before deciding it is absent? |

 It is important to determine if the patient is indeed in cardiac arrest. Cardiopulmonary resuscitation should be commenced if there is no cardiac output. However, instituting CPR when there is an underlying cardiac output, even if that output is much reduced, may be unnecessary and harmful.

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| **Note** | **The hypothermic heart is extremely sensitive to movement and sudden or rough movement may precipitate ventricular arrhythmias, including fibrillation.** |

Ventricular arrhythmias and asystole are frequently refractory to treatment until the patient has been rewarmed to, or above, 30 °C (86 °F). If initial resuscitation attempts fail, CPR should be continued, aggressive rewarming commenced, and defibrillation and pharmacologic therapy held until the patient has been adequately rewarmed.

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| **Caution** | **If hypothermia is the primary problem then the patient, who appears to be dead, is 'not dead until warm and dead'. Resuscitation efforts are maintained until the core temperature is greater than 32 °C (90 °F). In deciding whether to continue or cease resuscitation, precipitating events, severity of exposure and associated injuries are taken into account.** |

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| **Note** | **Atrial fibrillation and flutter usually resolve spontaneously as the core temperature rises.** |

*Understanding hypothermia*Normal body function depends on a relatively constant body temperature which is determined by a balance between heat production and heat loss. Heat is generated by cellular metabolism and is lost through four routes:* Convection
* Conduction
* Radiation
* Evaporation

Thermoregulation about a set point of approximately 37 °C (99 °F) is normally carried out by the anterior and posterior hypothalamus. The posterior hypothalamus is stimulated by the cortex when 'cold' is registered and activates mechanisms to increase heat production and reduce heat loss. Hypothermia develops when these adaptive systems are overwhelmed and core body temperature drops despite maximal heat production.As temperature drops below 30 °C (86 °F), the body loses its ability to spontaneously return to a normal temperature. That is, the body will require an exogenous heat source to survive.*Outcome*Survival in the short term depends on the degree of hypothermia, the situation in which it occurred, underlying medical conditions (if any), the age of the patient, and the resuscitation efforts undertaken. The mortality associated with hypothermia secondary to a pre-existing medical condition is far greater than that associated with primary hypothermia.

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| **Note** | **Successful resuscitation with full recovery is possible but depends on rapid diagnosis and aggressive management. The recognition and treatment of****predisposing conditions and complications is vital in this management.** |

**Environmental hyperpyrexia**Environmental heat-related illness ranges from milder forms such as heat stress or exhaustion to the most extreme form, heatstroke. Heatstroke results from failure of the body's thermoregulatory mechanisms and is a life-threatening emergency. Core body temperature commonly exceeds 42 °C (108 °F), and unless rapidly and aggressively managed is destructive to the brain and can progress to multi-organ failure and death. The pathophysiology resembles a systemic inflammatory response and is detailed in the reference below. See also the PACT module on Pyrexia [http://pact.esicm.org/courses/TemplateV2/images/go.gif](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=PYR&action=print&lp_id=1&preview=true).

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| **Note** | **Diagnosis of heatstroke requires the presence of hyperpyrexia and severe neurological dysfunction.** |

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| * Extremes of age
* Inability to increase cardiac output. e.g. cardiac failure
* Inability to recognise and/or relocate from heat source e.g. CNS disease, musculoskeletal disorders, poor social circumstance
* Lack of acclimatisation
* Fatigue-Obesity-Diabetes
* Substance abuse: alcohol, amphetamines, cocaine
* Medication, including:
	+ β-blockers
	+ Antihistamines
	+ Neuroleptics
	+ Selective serotonin reuptake inhibitors (SSRIs)
	+ Phenothiazines
	+ Anti-cholinergics
	+ Diuretics
 | Factors predisposing to heat-related illness |

*Acute situation*There are two distinct types of heatstroke:* **Classical heatstroke** can develop over several hours or even days, and primarily occurs in the elderly and those with chronic illness. This form of heatstroke occurs when heat stress is maximal for several days.
* **Exertional heatstroke** typically occurs in young, otherwise healthy individuals who engage in heavy exercise in high ambient temperatures and humidity. These patients can present as a sudden unexpected collapse and may be deeply comatose when initially examined.

*Immediate actions*Any patient suspected of heat illness, mild or severe, should be immediately placed in a cool environment. Unless the factors leading to heat exhaustion are corrected, affected patients can rapidly progress to heatstroke. Surface cooling with cold towels, application of cold water, and the provision of cool isotonic rehydration may be sufficient treatment for mild cases. See the treatment algorithm in the Glazer reference below.

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| **Note** | **Cooling should begin in the field if possible. For example pouring cold water on a collapsed athlete, e.g. with an adjacent garden hose, can effectively begin the process.** |

Heatstroke victims must be immediately and aggressively cooled. This must be initiated concurrently, with control of the airway, breathing and circulation. Morbidity and mortality are directly associated with the duration of elevated core temperature.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s231.jpg |  |

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*Treatment*Controversy continues as to the most efficient and safe method of rapid cooling but evaporative cooling and surface ice packs are most commonly used.* External cooling
	+ Evaporative cooling with room temperature mist and air flow
	+ Ice water immersion
	+ Cooling blankets
	+ Surface ice packs
* Internal cooling
	+ Cold intravenous fluids
	+ Cold lavage of stomach, bladder and rectum
	+ Intravascular cooling devices
	+ Cardiopulmonary bypass

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| **Caution** | **Cooling of the patient should begin immediately using readily available means! Don’t delay evaporative cooling while someone tries to locate the new intravascular cooling device.** |

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| **Activity** | **Find out what cooling methods are available in your Emergency Department and Intensive Care Unit.** |

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| **Note** | **Drug therapy (e.g. antipyretics, dantrolene) has nothing to offer in the management of heatstroke.** |

Heatstroke patients are also dehydrated and will require intravascular volume resuscitation. The amount of volume administered should be guided by clinical assessment using, for example, vital signs and urine output. Remember that cooling will result in abatement of vasodilatation and over-aggressive volume resuscitation may produce cardiac decompensation and pulmonary oedema particularly in the elderly. The majority of patients will respond to fluids alone, but a subset will require inotrope/pressor support. However, vasoconstriction may limit heat dissipation.Further treatment includes the search for underlying or precipitating causes, if not already obvious. Routine bloods on arrival include full blood count, electrolytes, renal profile, liver function tests, CPK and coagulation studies. Electrolyte abnormalities including hypernatraemia and hyponatraemia are common and care must be taken to correct to appropriate levels at appropriate rates. Other tests such as thyroid function tests, recreational drug screens, may be indicated depending on the clinical history. Seizure activity is not uncommon, and may be refractory to anticonvulsant therapy until temperature is controlled.*Complications*Despite aggressive cooling, the failure of one or more organs is a complication in up to 25% of patients with severe heatstroke. Acute renal failure is more common in exertional heatstroke than in classic heatstroke. Aetiology is multifactorial, but rhabdomyolysis frequently contributes. Acute severe coagulopathy and hepatic failure are not infrequent. Myocardial muscle may be damaged leading to cardiogenic shock and dysrhythmias. Neurological damage can be devastating, and carries a high mortality rate. For more information see the PACT module on Acute renal failure http://pact.esicm.org/courses/TemplateV2/images/go.gif and the following reference.

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| *Outcome*Mortality (<10%) has improved over past years with increased awareness, rapid diagnosis and aggressive management. Overall mortality is higher in the classic heatstroke group, and up to 33% have a residual neurological deficit.Poor prognostic indicators in classical heatstroke include 1-hypotension, requirement 2-for endotracheal intubation, 3-coagulopathy and 4-advanced age.The majority of exertional heatstroke victims should make a full recovery when optimally managed. Severe brain injury accounts for most of the fatalities in those who do not survive.3/ Near-drowning/drowningDrowning (death within 24 hours of submersion) and near-drowning (survival of at least 24 hours after submersion) are important causes of morbidity and mortality, mainly among children, adolescents and young adults. The availability of better prehospital care and more sophisticated technologies may improve survival but may also promote an increase in complications (ARDS and neurologic sequelae). The understanding of the pathophysiological mechanisms of near-drowning in freshwater and saltwater is important for the management of pulmonary and CNS-associated injuries.However, not all deaths on the water are due to asphyxia. The dive reflex (breath-holding, bradycardia with intense peripheral vasoconstriction and increased mean arterial pressure) elicited by the contact of the face with cold water may be responsible for a sudden death – immersion syndrome.Hyperventilation before swimming under water may permit hypoxaemia with loss of consciousness before the central respiratory drive initiates a breath; the seizure threshold may be lowered.You will find relevant information in the following references.*The acute situation*Drowning and near-drowning victims must be treated as multiple trauma patients and managed as per ATLS guidelines. Associated injuries commonly include cervical, spinal and traumatic brain injuries, and care must be taken to prevent these being exacerbated.Asphyxia after submersion produces hypoxaemia, which is responsible for many clinical manifestations seen in victims of drowning or near-drowning:* Cardiovascular effects include arrhythmias, low cardiac index, elevated right and left heart filling pressures, elevated systemic and pulmonary vascular resistance.
* Respiratory effects include acute laryngospasm (10-15%) which leads to obstructive asphyxia. Other causes of hypoxaemia include dilution of pulmonary surfactant (freshwater aspiration) or fluid movement from the capillaries to the alveoli (saltwater aspiration). Arterial oxygen content may be more significantly lowered with saltwater aspiration than with freshwater aspiration. Later, ARDS and pneumonia may worsen hypoxaemia.
* CNS effects range from minor disturbances to deep coma.
* Electrolytes and haemoglobin abnormalities are minimal in most patients who survive submersion. Haemodilution (with hyponatraemia and hypokalaemia), haemolysis and DIC may be seen with freshwater aspiration. Haemoconcentration can be observed with saltwater aspiration. However, classic patterns are not always observed.

Hypothermia is frequent in submersion victims: cerebroprotective effects of temperature below 30 °C are probably more significant in children than in adults. The diving reflex can also be a protective mechanism. See Task 2 for the effects of hypothermia http://pact.esicm.org/courses/TemplateV2/images/go.gif.*Immediate actions*Resuscitation measures (ABCs) should be started as soon as possible, at the scene if indicated. If healthcare providers are available, interventions such as tracheal intubation, defibrillation and administration of medications and fluids should be instituted as necessary.

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| **Note** | **Given the reports of successful resuscitation in several very dramatic conditions, resuscitation should be attempted in all victims of near-drowning, except when they are clearly dead.** |

Most victims do not aspirate a significant quantity of water: 10% probably do not aspirate even a small quantity. The Heimlich manoeuvre, if performed, may induce vomiting and aspiration of gastric contents.

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| **Note** | **The Heimlich manoeuvre is performed only if an airway obstruction with a solid foreign body is highly suspected.** |

On arrival at the emergency department, advanced life-support measures are started or continued, if indicated.If there is hypothermia, rewarming is a priority. Acid-base status, electrolytes and cardiovascular status (particularly cardiac rhythm) are monitored closely during the rewarming process.

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| **Caution** | **Caregivers should be aware of several possible conditions associated with drowning or near-drowning: 1-alcohol and other drugs, 2-myocardial infarction, 3-seizures, 4-air embolism (after rapid ascent during scuba diving), 5-venomous stings by aquatic animals, and 6-trauma. These may be responsible for an unsuccessful resuscitation.** |

* Neurologic assessment (including GCS, brain-stem reflexes and a more detailed examination, if possible) is performed to serve as a baseline reference and to determine the individual patient prognosis.
* In an alert patient with compromised oxygenation but with PaCO2 less than 45 mmHg (6 kPa), non-invasive ventilation (NIV) with CPAP or BiPAP may be instituted. If, however, PaCO2 rises above 50 mmHg (6.7 kPa), neurologic deterioration occurs, gastric distension or vomiting takes place or hypoxaemia persists or worsens, tracheal intubation and mechanical ventilation are indicated.
* Every patient suspected of having had a submersion injury should be observed closely for at least 12 hours to rule out the development of complications such as delayed pulmonary oedema.

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| **Note** | **Late-onset severe complications (fulminant pulmonary oedema and delayed cerebral oedema) have been reported.** |

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| Monitoring should also include:* A baseline electrocardiogram to evaluate the QTc interval
* Toxicologic studies (alcohol and other drugs)
* Levels of anticonvulsant drugs (if there is a history of epilepsy)
* CT scan of head and cervical spine if there are suspected injuries
* Laboratory diagnostic studies (blood gases, electrolytes, blood urea nitrogen, creatinine and haemoglobin)
* Chest X-ray
 | Risk factors for submersion injury include epilepsy, previously unknown long QT syndrome, alcohol and other drugs |

*Clinical diagnosis*Clinical diagnosis is based on the history of a submersion accident: in this setting all possible associated injuries should be evaluated. Clinical findings range from mild 1-respiratory, 2-cardiovascular and 3-neurologic manifestations to 4-cardiopulmonary arrest. Vomiting is common. 5-Hypothermia is likely after prolonged or cold water submersion.To determine the likelihood of benefit from further therapeutic interventions, it is important to know when, where and how the event occurred, if it was witnessed, the time between the accident and the rescue, and to rule out other possible associated injuries.

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| **Think** | If a child has suffered near-drowning in a bathtub, a non-accidental injury may be suspected. Other possible signs of abuse include injuries to the face, retinal haemorrhages, burns and foreign bodies in the lungs. |

*Treatment*Careful observation and continuous monitoring of the patient, serial physical and neurological examinations, laboratory and radiological studies are needed to guide therapy.Pulmonary dysfunction results from aspiration, atelectasis and pulmonary oedema. When endotracheal intubation and mechanical ventilation are needed, the strategy should focus on achieving adequate gas exchange and on preventing ventilator-induced lung injury. Hypercapnia and hypocapnia should be avoided; there is no evidence that moderate hyperventilation improves neurologic outcome after hypoxic-ischaemic brain injury. Bronchodilators are used if bronchospasm due to aspirated material is present; antibiotics are used only if there is evidence of infection.Standard monitoring and therapy are recommended for cardiovascular support. Cerebral injury is often established when the patient is admitted to the ICU and standard prevention of secondary injury is indicated – treatment of seizures, hypercarbia, fever, raised intracranial pressure and the maintenance of oxygenation and cerebral perfusion pressure. For more information see the PACT modules on Mechanical ventilation http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/media/go.gif, Haemodynamic monitoring http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/media/go.gif, Acute brain ischaemia http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/media/go.gif.*Prognosis and outcome*Several studies have examined the outcome of submersion accident victims and attempts have been made to define reliable predictors of outcome at the scene, the Emergency Department and the ICU. Numerous proposed predictors of outcome have been unable to discriminate intact survivors from non-survivors and severely impaired survivors, including absence of spontaneous respiration, lack of response to pain, pupillary non-reactivity and core temperature.Repeat clinical neurologic examinations in the first 24-72 hours of therapy are probably the most reliable indicators of neurologic outcome; magnetic resonance imaging, magnetic resonance spectroscopy and neurophysiological testing may help to clarify doubts.

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| **Activity** | **Examine the evidence for the use of scoring systems to discriminate intact survivors from those with poor prognosis. Review the practice in your own unit.** |

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| Question Which factors are related to better survival in near-drowning? |

**4/ Hazardous chemical exposure**You may encounter patients who have been poisoned by chemicals either as a result of an individual accident or suicide attempt, or through chemical warfare and terrorism. The clinical course may be severe and may require admission to your ICU. In this task we have identified key chemicals, principally pesticides, which you may encounter. Pesticides include insecticides, herbicides, rodenticides and other pest control products. About 10% of poisoning-related mortality is as a result of pesticide poisoning.The following references provide additional information on this topic. The Wexler reference is a useful summary of Internet resources.

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| RodenticidesThe use of the highly toxic rodenticides, such as strychnine, arsenic, phosphorus and thallium, is restricted in many countries and consequently poisoning by these products is rare. Poisoning by the less toxic anticoagulant compounds, such as indanediones and hydroxycoumarins is more common.Hydroxycoumarins alter the synthesis of coagulation factors (II, VII, IX, X) in the liver, as they interfere with vitamin K-mediated gamma carboxylation of that precursor coagulation factor proteins. As rodents developed resistance to warfarin, ‘superwarfarins’ (long-acting hydroxycoumarins) such as brodifacoum and indanedione were introduced. Most of the accidental ingestions of these compounds occur in children. Fatalities attributed to poisoning with warfarin rodenticides are unknown. The intentional ingestion of superwarfarins may be associated with significant morbidity but still with low mortality.*The acute situation*There are no characteristic features of acute poisoning because anticoagulant rodenticides have a delayed onset of action.

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| **Caution** | **Large amounts of domestic rodenticides have to be ingested to produce severe bleeding. Accidental ingestion frequently involves several grains of rodent bait which is insufficient to produce changes in coagulation. If this occurs think about multiple poisoning.** |

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| 1. Exposure to extreme cold
	* Outdoor pursuits (الرحلات (
	* Near-drowning
	* Motor vehicle breakdown
2. Extremes of age
3. Malnutrition
4. Exhaustion
5. Social environment
	* Homelessness
	* Lack of heating
6. Any medical condition which reduces ability to recognise or react to hypothermia
	* CNS disease
	* Cardiovascular disease
	* Diabetes
	* Hypothyroidism
	* Adrenal insufficiency
	* Peripheral neuropathies
	* Peripheral vascular disease
7. Trauma leading to immobility
	* Fractures
	* Head injury
	* Spinal cord injury
	* Burns
8. Some prescribed medications e.g. pethidine (mepiridine), clonidine
9. Alcohol abuse
10. Drug abuse
 | **Hypothermia: predisposing factors/conditions** |

###  *Immediate actions*

Most patients can be managed conservatively and only rarely need to be transferred to intensive care. Gastric lavage is usually unnecessary though activated charcoal may be administered if the ingestion occurred within the previous hour. Monitor prothrombin time.

### *Clinical diagnosis*

The history of possible exposure and measurement of prothrombin time (which may be normal for several hours, however) are the key to establishing the diagnosis. If the prothrombin time is normal for 48 hours after the ingestion, significant poisoning is unlikely. As in every poisoning, it is important to identify the involved toxin: a superwarfarin may need a prolonged follow-up.

### *Treatment*

Treatment decisions must be based on bleeding manifestations and may include treatment of shock, transfusions (mainly of fresh-frozen plasma) and neurosurgical treatment of intracranial bleeding. A haematologist should be consulted in severe cases. Prevention of falls and trauma, and avoiding the use of central intravenous catheters, nasogastric and endotracheal tubes, are desired.

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| **Note** | **Drug interactions with warfarin are common: as a result increased anticoagulant effect or decreased anticoagulant metabolism may occur and interfere with the treatment plan. Such drugs should be avoided.** |

Vitamin K1 (phytonadione ) should be administered (5-10 mg, slowly i.v.) if prothrombin time measured at 24 and 48 hours after ingestion is greater than twice normal. Repeated doses in severe poisonings or after superwarfarin ingestion may be required, up to 200 mg/day.

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| **Note** | **Vitamin K1 use is not recommended in patients with prosthetic heart valves or other indications for prolonged anticoagulation, as clotting may occur; should this reversal of anticoagulation be required use fresh-frozen plasma as an alternative.** |

Follow-up is recommended if vitamin K1 was given prophylatically after an acute ingestion for a minimum of five days (after the last vitamin K1) or as long as bleeding diathesis persists.

## Insecticides

Dermal exposure to, ingestion or inhalation of insecticides will result in poisoning which ranges from mild to severe depending on the quantity and toxicity of the compound. Organophosphates and carbamates inhibit acetylcholinesterase (AChE) which causes acetylcholine to accumulate and results in overstimulation of muscarinic and nicotinic receptors, producing the symptoms and signs outlined below. Carbamates result in limited toxicity because they do not enter the central nervous system and the enzyme inhibition is reversible. Organophosphates (including nerve gas agents) permanently inactivate acetylcholinesterase and penetrate the central nervous system resulting in greater toxicity and need for aggressive treatment.

### *The acute situation*

Very severe poisonings usually present with bradycardia, bronchorrhoea, sweating and miosis. Respiratory effects are the first noted after inhalation; gastrointestinal effects are usually the first noted after ingestion; sweating and muscular fasciculations are usually the first noted after dermal exposure.

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| **Caution** | **Death can occur in a few minutes as a result of muscular paralysis and sudden respiratory arrest after severe intoxication.** |

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| **Note** | **Muscarinic effects may predominate in mild organophosphate poisoning and nicotinic and CNS effects in moderate to severe poisoning. Bradycardia is present in 2/3 patients and tachycardia in 1/3.** |

### *Immediate actions*

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|  | Do not become a casualty yourself |

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| **Caution** | **Avoid contamination. Prevent direct contact with the patient's skin, clothes or vomitus.** |

### ABCs

Emergency action may be required at the scene or in the emergency department (ED). Administer oxygen. Establishment/maintenance of a patent airway (perhaps with tracheal intubation) and assisted ventilation may be required. Depolarising neuromuscular blockers should be avoided in these patients as they produce prolonged paralysis. The dose of non-depolarising blockers may need to be increased to compete with the increased amount of acetylcholine at the neuromuscular junction. Cardiopulmonary resuscitation may be needed at once. See the PACT module on Airway management for further information .

### Decontamination

Contaminated clothes must be removed and bagged. Patients poisoned through dermal exposure must have their skin decontaminated with soap or detergent and plenty of water. Hair, fingernails and umbilicus have to be well washed.

### Initial therapy

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| After, or concomitant with the correction of hypoxia, administer atropine to reverse muscarinic effects and CNS manifestations. Initial dose is 1-2 mg i.v. and may be repeated as needed to more than 1 g in 24 hours, targeted to achieve atropinisation. Atropine may be administered in bolus doses or by continuous infusion. Signs and symptoms of adequate atropinisation include drying of secretions, absence of wheezing, tachycardia (or adequate control of bradycardia), mydriasis (miosis may persist in some patients), dryness of the skin and mouth and flushing of the skin. | Signs of atropinisation |

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| **Note** | **Tachycardia is not a contraindication for the use of atropine.** |

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|  | Monitoring should be started as soon as possible as sudden deterioration may have catastrophic consequences. Even with milder poisoning, symptoms may be delayed and monitoring is recommended for at least eight hours |

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| **Caution** | **Death after exposure to organophosphate poisoning usually results from respiratory failure and may occur even before treatment starts: note however that most fatalities are a consequence of missing the diagnosis or inappropriate or insufficient therapy. Do not underdose atropine!** |

For further information on monitoring, see the PACT module on Haemodynamic monitoring .

### Prevention of toxin absorption

Gastric lavage and subsequent administration of activated charcoal are performed after ingestion. Cathartics are not recommended because patients develop diarrhoea.

Patient observation for at least eight hours is recommended as delayed onset of symptoms from skin absorption is possible.

### *Clinical diagnosis*

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| Clinical diagnosis should be initiated and take place concurrently with initial measures.History taking and physical examinationUnless the exposure has occurred as a suicide attempt, the history of exposure to the poison is usually clear from the patient or from those bringing the patient to the ED; characteristic clinical manifestations help confirm the nature of the intoxication. Improvement of signs and symptoms after initial treatment may further help to establish the diagnosis. | Organophosphate and carbamate poisoning in young children presents with CNS and hypotonic effects, muscle weakness and miosis without other cholinergic signs. In these patients, absence of classic muscarinic signs does not rule out organophosphorus or carbamate poisoning |

Symptoms and signs can be grouped into the following three categories.



Muscarinic symptoms can be enumerated with aid of the mnemonic 'SLUDGE' – **S**alivation,**L**acrimation, **U**rination, **D**iarrhoea, **G**I cramps, **E**mesis.

Some insecticides such as pyrethrins/pyrethroids rarely cause acute poisoning although anaphylactic reactions have been reported. Patients poisoned by carbamates have milder symptoms.

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| **Note** | **Depending on the severity and the route of the poisoning, symptoms may develop rapidly or may not become evident for several hours following exposure.** |

### Laboratory investigations

Measurement of red blood cell (RBC) cholinesterase and plasma cholinesterase – levels vary according to the severity of the poisoning and may aid in confirmation of the diagnosis. However there are limitations.

There is a wide inter-individual variation in cholinesterase activity: clinical parameters have to be considered when interpreting cholinesterase levels for determination of the severity and prognosis of the intoxication.

Both RBC cholinesterase and plasma cholinesterase are affected by organophosphate, but RBC cholinesterase is more specific because it correlates better than plasma cholinesterase with nervous system AChE. In most patients, the baseline level is unknown: in acute poisoning, symptoms occur usually when more than 50% of cholinesterase is inhibited and in severe poisoning less than 10% of cholinesterase is active. As a consequence, a single level of cholinesterase may not be helpful (exceptions are normal and very low levels). Patients with haemoglobinopathies and haemolytic anaemia have lower levels of RBC cholinesterase while patients with liver cirrhosis, malnutrition and pregnancy have lower levels of plasma cholinesterase.

Other helpful non-specific studies include: glucose, creatinine, urea, electrolytes, liver enzymes, arterial blood gases, oximetry, chest X-ray and ECG monitoring.

### Poison control centres

Contact your local poison control centre for advice and assistance on diagnosis and treatment. See also the PACT module on Major intoxication  which contains details of Internet resources.

### *Treatment*

### Supportive

Supportive treatment for organophosphate poisoning is continued as needed and it is usually sufficient in carbamate poisoning. A benzodiazepine may be given in small doses to relieve anxiety in both organophosphate and carbamate poisoning; it may be needed to treat seizures and twitching. The patient may be discharged from the ICU if s/he is asymptomatic with cholinesterase activity stable and specific therapy, atropine  and obidoxime/pralidoxime  (see on the next screen) has been successfully withdrawn for two days.

### Specific

##### Atropine

Atropine  may be needed for weeks. After stabilisation, atropine should be withdrawn slowly while monitoring respiratory secretions. Glycopyrrolate, which does not cross the blood–brain barrier, may be considered instead of atropine if there are no central nervous system symptoms.

##### Oximes

In organophosphate poisoning, but not carbamate poisoning, oximes should be started as soon as possible, to reverse nicotinic effects and probable CNS effects. Even if there is doubt concerning the aetiology of the poisoning but organophosphate is a possibility, pralidoxime  or another oxime such as obidoxime should be started. Oximes are not recommended for carbamate poisoning and sometimes may be detrimental. When, however, the agent is not identified and the patient is severely poisoned, oximes may be given.

The initial dose of pralidoxime  is 1-2 g in 100 ml of D5W, i.v. over 15-30 minutes; maximum infusion rate should be 500 mg/hr in severe poisoning. The initial paediatric dose is 25-50 mg/kg.

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| Obidoxime or pralidoxime Drug definition are given for 24 hours to several days (in long-acting, lipid-soluble organophosphate poisoning). You can find out more about oximes in the following article. Obidoxime may be more efficient where CNS effects are prominent. | Obidoxime is not as widely available as pralidoxime Drug definition |

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You can find further details on these and other antidotes in the [formulary](http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/druglist/drugList-envhazards.html).

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| Question Which drugs are contraindicated in acetylcholinesterase inhibitor poisoning? Why? |

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| *Managing complications*Neurological complicationsNeurologic findings associated with organophosphate poisoning may develop during the acute toxicity period or after recovery of the symptoms related to the acute intoxication. **Intermediate syndrome**starts usually one to four days after poisoning and after the patient has survived the acute cholinergic crisis.Features include weakness (bulbar, nuchal and proximal limb musculature) with acute respiratory paralysis and depressed reflexes. The mechanism is still not completely understood: it is associated with some organophosphates (such as dimethoate, parathion, methylparathion, malathion, fenthion) and it is believed that adequate treatment with oximes from the beginning may prevent its appearance. Patients often need ventilatory support for some days; resolution occurs frequently after two to four weeks with recovery.NeuropathiesOrganophosphate-induced delayed neuropathy (OPIDN) or polyneuropathy (OPIDP) occurs one to three weeks after poisoning. Symptoms start in lower limbs and progress to upper limbs with cramping, weakness, and paresthesias. Electromyograms show a denervation pattern. Frequently, sensory symptoms recover in the next one or two months; paralysis remains for several months to years. Some patients will not recover and will have persistent spasticity.Treatment is supportive. This syndrome is also associated with some organophosphates (omethoate, parathion, dichlorvos, chlorpyriphos, among others); in developed countries most of these compounds are no longer used. Psychiatric sequelae may develop later and persist for weeks to months; they include depression, personality changes, memory impairment, and confusion.

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| Question How can you differentiate organophosphate-induced delayed neuropathy (OPIDN) from Guillain-Barré syndrome? |

 However, a recent observational study of 376 patients found that the two organophosphate-related syndromes of early and late respiratory failure were not clinically distinct and overlapped considerably. Marked differences in the pattern of respiratory failure were noted between the various organophosphate compounds.

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Seizures and comaSeizures and coma should be treated aggressively.You can find further information about neurological complications in the PACT modules on Altered consciousness http://pact.esicm.org/courses/TemplateV2/images/go.gif and Neuromuscular conditions http://pact.esicm.org/courses/TemplateV2/images/go.gif.Acute pancreatitisAcute pancreatitis mainly concerns dysfunction of the exocrine pancreas; clinical manifestations may be similar to pancreatitis with a different aetiology. You can find out more on acute pancreatitis in the PACT module on Pancreatitis [http://pact.esicm.org/courses/TemplateV2/images/go.gif](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=PAN&action=print&lp_id=1|preview=print).PneumonitisPneumonitis may occur as a result of aspiration of gastric contents, but frequently is a consequence of aspiration of the petroleum distillates in which the organophosphates are carried. If there is no evidence of infection, antibiotics are not indicated.Prolonged QT intervalSome patients present with prolonged QT interval, with or without torsades de pointes; cardiotoxicity with structural lesions of the myocardial cells probably due to direct action of organophosphate poisoning may be also related to sudden cardiac arrest after symptomatology has been controlled. You can find out more about the cardiotoxicity of organophosphates in the reference below. The PACT module on Arrhythmia http://pact.esicm.org/courses/TemplateV2/images/go.gif contains further information about prolonged QT intervals.

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| *Understanding insecticide poisoning*Both organophosphates and carbamates inhibit the enzyme acetylcholinesterase, and therefore cause accumulation of acetylcholine at muscarinic and nicotinic receptors. Organophosphates have an irreversible effect on the enzyme and also cross the blood–brain barrier, and hence are more toxic than the carbamates. Oximes, such as pralidoxime Drug definition, remove the phosphoryl group from the acetylcholinesterase molecule and re-activate the enzyme at nicotinic receptors which are present in skeletal muscles and autonomic ganglia. However, they do not reverse muscarinic effects, and adequate doses of atropine are required to control secretions. For more information about the pathophysiology, see the following references.

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| **Herbicides**Although herbicides are widely used in agriculture, significant exposure (whether intentional or occupational) is not very common. Two forms of herbicides you may encounter are:* Chlorophenoxyacetic acid derivatives which include 2,4 dichlorophenoxyacetic acid (2,4-D), 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2-methyl- 4-chlorophenoxyacetic acid (MCPA). Agent Orange (a mixture of 2,4-D and 2,4,5-T) is contaminated with dioxin, which has entered the food chain in certain places, and is associated with long-term accumulation and toxicity in the exposed population.
* Bipyridyl derivatives which include paraquat and diquat.

You can find further information about these and less toxic forms such as glyphosate on the Intox website, given below:

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| *The acute situation*Ingestion of chlorophenoxyacetic acid derivatives and bipyridyl derivatives can be fatal. Death may also occur after intramuscular injection (attempted suicide) and percutaneous exposure particularly if skin is not intact. | biblio |

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### *Immediate actions*

### ABCs

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| The severity of the clinical features will determine the emergency procedures. If the patient is unconscious or unable to maintain the airway, intubation should be accomplished and assisted ventilation started if needed. | Timing is crucial |

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| **Note** | **In paraquat poisoning, use the minimum concentration of oxygen to achieve a PO2of about 60 mmHg (7.7 kPa). Ventilation with positive-pressure with low inspired oxygen tension should be instituted to achieve adequate arterial oxygen tension. Excessive oxygen enhances lung damage from paraquat (by aggravating lipid peroxidation reactions in the lung).** |

Use intravenous crystalloid solutions to treat fluid and electrolyte disturbances.

### Decontamination

Dermal paraquat exposure-induced systemic toxicity is rare but may be fatal. Patients are washed repeatedly with soap and water (contaminated clothes are bagged and treated thereafter).

### Initial therapy

Gastric lavage should be performed if within one hour of the ingestion. Fullerâs earth (300 g of a 30% solution) or 100 g of activated charcoal (the comparative efficacy is not determined) should be administered. They are more effective if given within four hours after ingestion. Activated charcoal may be repeated one to two hours later.

There are no specific antidotes to poisoning by chlorophenoxyacetic acid derivatives and bipyridyl derivatives.

### Monitoring

Mild intoxications can be managed conservatively. Monitoring (ECG, respiratory and mental status) and serum chemistries are recommended for at least 12 hours (because of possible delay in onset of symptoms).

### *Clinical diagnosis*

### History taking and physical examination

The following table summarises the key symptoms and signs of acute poisoning.

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| http://pact.esicm.org/courses/ENVHAZ/scorm/environmental_hazards/images/s432.jpg | **Symptoms and signs of acute poisoning – key herbicides** |

The degrees of paraquat toxicity based on the ingested dose are indicated in the following reference.

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### Laboratory investigations

In poisoning with chlorophenoxyacetic acid derivatives, the history of possible exposure, the presence of muscle weakness and elevated CPK are important for the diagnosis.

Measurement of the 2,4-D metabolite in the urine (if available on time) may help to establish the diagnosis. Likewise an occult heme test positive in the presence myoglobin.

The history of possible ingestion and the presence of oral burns and severe gastrointestinal symptoms are the basis for the diagnosis of paraquat poisoning.

Helpful non-specific studies include: glucose, creatinine, urea, electrolytes, liver enzymes, arterial blood gases, oximetry, chest X-ray and urinalysis.

Measurement of serum levels of paraquat and diquat may help to establish the diagnosis. However paraquat levels are very useful to establish the prognosis; levels as high as 2 mg/l four hours after ingestion or 0.6 mg/l six hours after ingestion or 0.1 mg/l 24 hours after ingestion may predict a poor outcome.

### *Treatment*

Treatment (after emergency and supportive measures) of metabolic acidosis, coma (dextrose, thiamine and naloxone), rhabdomyolysis (intravenous fluids, mannitol and sodium bicarbonate) and hypotension (intravenous fluids and inotropic therapy); a systematic approach to supportive organ failure including haemodiafiltration is performed as needed.

Treatment of paraquat poisoning after emergency measures includes methods to enhance excretion; charcoal haemoperfusion has been suggested to be beneficial mainly if it is started before toxic concentrations accumulate in the lung (patients who have been poisoned for less than 12 hours and with levels below 3 mg/l), however consensus has not been reached. On the basis of the Lin JL study (small numbers) and case report evidence (see on the next screen), there is increasing use of high dose steroids and cyclophosphamide in the management of paraquat poisoning.

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| **Anecdote** | *A patient poisoned with paraquat from dermal exposure, reached serum paraquat level of 0.13 mg/l sixty hours after exposure. He presented with severe hypoxaemia and was successfully treated with cyclophosphamide (15 mg/kg/day, total two days) and methylprednisolone (15 mg/kg/day, total six days) followed by continuous dexamethasone administration (5 mg every eight hours).* |

Further details in:

### *Understanding herbicide poisoning*

Both the chlorphenoxy and bipyridyl herbicides are cellular toxins. The chlorphenoxy compounds produce dose dependent damage to the cell membranes, uncouple oxidative phosphorylation and disrupt acetylcoenzyme A metabolism. Skeletal muscles are the main target, leading to weakness, hypoventilation and rhabdomyolysis.

Bipyridyl compounds produce superoxides when they undergo cyclic oxidation-reduction reactions in tissues. This leads to lipid-peroxidation of cellular membranes. Paraquat selectively accumulates in the lungs and causes damage to the alveolo-capillary membrane, ARDS and pulmonary fibrosis. All these effects are exacerbated by high inspired oxygen levels. Paraquat and diquat have toxic effects on various other organs as well, such as the liver, kidneys heart and CNS.

### *Outcome*

Patients who ingested more than 20-40 mg/kg of paraquat have a high likelihood of death; in short, the higher the dose the faster is the dying process. The lethal dose of chlorophenoxyacetic acid derivatives is above 6 g with the minimum toxic dose for 2,4-D being usually 3-4 g. Despite heroic efforts, death may be unavoidable and, in these patients, palliative care is appropriate

**Conclusion**

Environmental hazards such as those described in the four tasks of this module may happen throughout the world. After you have concluded this module, you should be able to:

* Initiate early burns management and identify patients to refer to a specialised unit with safety.
* Recognise associated early complications such as cyanide and carbon monoxide poisoning and late complications such as infection and musculoskeletal and neuropsychological sequelae.
* Describe principles of management and approach to temperature-related injuries.
* Prioritise and initiate timely treatment of near-drowning/drowning victims and manage the many possible associated conditions.
* Recognise the broad spectrum of pesticide toxicity and know the principles of, and approach to, management.
* **Patient challenges**
* **A 45-year-old female was brought to the Emergency Department after ingesting an unknown quantity of an organophosphate pesticide as a suicide attempt.** Clinical manifestations included dyspnoea due to bronchospasm and bronchorrhoea, bradycardia, salivation, miosis, urinary incontinence, fasciculations, muscle weakness and lethargy.

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif ABCs***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4ImmediateActionsABCs)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Cholinergic manifestations of poisoning***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4CInsectListSymp)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Atropine after resuscitatione***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4ImmediateActionsInitialTherapy) |

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| **Note** | **Latex or vinyl gloves are not adequate.** |

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| Question What is the most appropriate management of the patient at this time? |

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| **Note** | **Respiratory complications are the major cause of death in severely poisoned patients.** |

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| Question Would antidotes be useful at this point? Explain. |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Dosage regimen for atropine***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4ImmediateActionsInitialTherapy) |

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| **Note** | **Tachycardia occurs in about one third of cases but is not a contraindication for atropine treatment.** |

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| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Indications for oximes***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectOximes)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gifEvidence for oxime utilisation***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectOximes) |

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| Question How should the patient be decontaminated? |

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| **Note** | **All removed clothing, including shoes, should be treated as toxic waste.** |

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| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Indications for gastric lavage***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4ToxinAbsorpPrevention) |

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| Question Which investigations are helpful? |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif RBC level best reflects neuronal cholinesterase activity***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectLabInvest) |

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| **Note** | **Other conditions depress RBC and plasma cholinesterase levels.** |

* The third day after admission to the ICU, the patient is stabilised (heart rate 90 bpm, 4-5 mm pupils) on atropine continuous i.v. infusion of 2 mg/hr and she is sedated with 5 mg/hr of midazolam. Obidoxime infusion is stopped. Approximately six hours after this, the patient's heart rate decreases to 60 bpm and a few minutes later it further decreases to 30 bpm.

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Check ECG for prolonged QT interval***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectQTInterval)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module on Arrhythmia***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ARR&action=print&lp_id=1&preview=true)**>** |

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| **Note** | **Complications of stopping oxime infusion therapy.** |

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| Question What is the most likely cause of this event and what is the most appropriate management of the patient at this time? |

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| Question What non-cardiovascular complications may occur early after organophosphate intoxication? |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Early and late complications of organophosphate poisoning***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectManagCompl) |

* The obidoxime infusion is immediately recommenced and the patient again stabilised. The atropine infusion is increased and she is successfully weaned from obidoxime 24 hours later. Several days later, when weaning from invasive ventilation, it is noticed that although awake and alert, she is peripherally very weak.

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| Question What are the most likely reasons for this weakness ? |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Intermediate syndrome***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectManagingComplications)[***go.gif Delayed neuropathy***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectManagingCompsNeuropathies)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Guillain-Barré syndrome***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectManagingCompsNeuropathies)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Weakness of critically ill patients***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT4InsectManagingCompsNeuropathies) |

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| Question What delayed neurological complications may occur after organophosphate intoxication? |

* Investigations including serial clinical examinations, electromyography, lumbar puncture and CSF studies supported a diagnosis of intermediate syndrome associated with organophosphate poisoning. The patient made a full recovery and was discharged from the ICU two weeks later.
* **A 37-year-old male was admitted to the Accident and Emergency Department following a workshop fire.** His BP was 110/50 mmHg, heart rate 110/min, respiratory rate 22/min, and SaO2 of 90% on mask oxygen (35%). He was fully orientated (GCS = 15) but was shivering and in pain and gave a history of a flash burn when working with electrical equipment. He has a 10-year history of insulin-dependent diabetes which has been well controlled with no known complications. Body weight is estimated at 80 kg.

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| QuestionWhat is your initial response? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Think ABCs first***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Resuscitation specific to burns***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Carbon monoxide poisoning***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1CarbonMonPoison)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Cyanide poisoning***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1CyanidePoison)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Initial fluid therapy***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1TailorFluitTher) |

*
* This response results in some improvement in BP and heart rate and the oxygen saturation increases to 98%. Blood is drawn for full blood count, urea and electrolytes, coagulation screen, cross-match and arterial blood gases.

Primary and secondary surveys reveal extensive burns to arms, chest and neck. There is soot staining of the oropharynx and singeing of his eyebrows and hair. There are no other injuries to head, thorax, abdomen or limbs.

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| QuestionDoes this patient require respiratory intervention? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Airway inhalational injury***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Early pre-emptive tracheal intubation***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1) |
| QuestionWhat are the warning signs for airway burns injury? |

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| QuestionHow will you assess this patient's burns? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Assessment of burn***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Area of burn – 'Rule of Nines'***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Depth of burn***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Non-burn injuries (1)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Non-burn injuries (2)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1BurnsAssessFluidMgt)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Paediatric differences (Lund-Browder)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn) |

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| **Note** | **Palm estimation** |

*
* This patient is uneventfully sedated, intubated and ventilated.

Bronchoscopy reveals reddened, inflamed oropharyngeal tissues and vocal cords, and oedematous trachea and bronchi. There is extensive soot evident throughout.

Initial burn injury estimation shows:
9% full-thickness burns right forearm.
36% partial thickness burns to chest, neck and arms.

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| QuestionWhat are the implications of your assessment of the extent of the burn injury? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Planning medical management***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Prognostication***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Consultation and referral***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1Treatment)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Timing of surgery (1)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Timing of surgery (2)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1Treatment)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Consider transfer to Burns Unit***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1Treatment) |
| QuestionHow will you calculate this patient's fluid requirements for the next 24 hours? |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Ongoing fluid management***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ContinueFluidManag) |

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| QuestionHow will you monitor ongoing fluid requirements and adequacy of resuscitation? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Monitoring fluid requirements***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ContinueFluidManag) |

*
* The burns to the right forearm are circumferential, compromising the vascular supply to that hand, therefore requiring urgent escharotomy.

Central venous access and intra-arterial monitoring are established and the patient transferred to the operating theatre. Intra-operatively the patient remained haemodynamically stable although blood loss reached 2000 ml requiring transfusion of two units of RBC and four litres of crystalloid in addition to his ongoing burn resuscitation fluid. On return to the ICU he is noted to be hypotensive (BP 90/40 mmHg) with a tachycardia of 130 bpm, including frequent ventricular ectopy.

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Urgent escharotomy for limb salvage***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1AssessBurn)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Peri-operative fluid requirements***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ContinueFluidManag)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Intravascular volume monitoring***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ContinueFluidManag) |
| QuestionWhat is your initial management? |

*
* The patient is given a total of one litre of crystalloid and one litre of colloid over approximately one hour. CVP increases from 9 to 12. Despite the fluid bolus he remains hypotensive and oliguric, passing notably dark urine, and continues to have frequent ventricular ectopic activity. Results on blood taken immediately postoperatively reveal a haemoglobin of 9 g/dl, platelets of 90, K = 4.9, urea = 8, creatinine = 140 with a metabolic acidosis pH = 7.25, PCO2 = 3.9, HCO3 = 17.

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| QuestionTaking into account the history of this patient what are your primary concerns here? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Electrocution injury***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ElectricInj)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Cardiac injury in burn patients***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1GeneralTreat)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Cardiac injury in burn patients (2)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ElectricInj)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Rhabdomyolysis (1)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1GeneralTreat)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Rhabdomyolysis (2)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ElectricInj)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module on Oliguria and anuria***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=OLIANU&action=print&lp_id=1&preview=true) |
| QuestionWhat is the potential for haemorrhage and fluid loss as a cause for hypotension in a patient with a haemoglobin of 9 and a CVP of 12? |

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| QuestionWhat does the lack of response to volume therapy and CVP augmentation suggest? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Carbon monoxide and cyanide poisoning***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1CarbonMonPoison) |
| QuestionWhat might have caused the ventricular ectopic beats? What investigations will be helpful? |

*
* ECG and troponin levels are normal; transoesophageal echocardiography (TOE) demonstrates a hyperdynamic, well contracting heart with no wall abnormalities. CK levels are hugely elevated at 32 000 IU/L and K+ level is >6 mmol/l. Despite aggressive management of his rhabdomyolysis, he progresses to anuric acute renal failure requiring continuous veno-venous haemodiafiltration (CVVHDF). On day nine post burn insult he develops a temperature of 39.5 °C, a new leukocytosis of 24 000 and requires increasing FiO2 to maintain O2 saturations >95 %.

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Rhabdomyolysis (1)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1GeneralTreat)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Rhabdomyolysis (2)***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1ElectricInj)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Acute renal failure in burns***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1GeneralTreat)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module on Oliguria and anuria***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=OLIANU&action=print&lp_id=1&preview=true)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module on Acute renal failure***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ACREFA&action=print&lp_id=1&preview=true) |
| QuestionWhat is the likely aetiology of this deterioration? |

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| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Sepsis***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1InfSepsis)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Respiratory infection***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1GeneralTreat)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Burn wound superinfection***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1InfSepsis) |

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| QuestionHow will you manage this complication? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Sepsis management***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1InfSepsis)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif Catheter infection***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=ENVHAZ&action=print&lp_id=1&preview=true#ENVHAZT1InfSepsis) |

*
* Sputum, (large amounts and noted to be purulent), urine, blood and swabs from burn sites have been sent to lab for multiple chemical sensitivity (MCS). Apart from an increasing leukocytosis, all other bloods including liver function tests are unremarkable. The first of three faecal samples for *C. difficile* toxin has been collected. All catheters have been changed. CXR shows a new left-sided consolidation; he now requires FiO2 >80% to maintain saturation and he has become hypotensive despite increasing doses of vasopressor.

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| QuestionHow will you treat this respiratory infection? |
| **Learning issues** |  |
| [***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module Sepsis and MODS***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=SEPMOD&action=print&lp_id=1&preview=true)[***http://pact.esicm.org/courses/TemplateV2/images/goPatch.gif PACT module Severe infection***](http://pact.esicm.org/main/newscorm/lp_controller.php?cidReq=SEVINF&action=print&lp_id=1&preview=true) |

*
* He was commenced on piperacillin/tazobactam, fluconazole and oral metronidazole and rapidly improved in the following 48 hours. Sputum cultures showed a heavy growth of sensitive pseudomonas as did his burn site swabs. In the absence of significant fungal growth, with all samples negative for C. *difficile* toxin, the fluconazole and metronidazole were discontinued. Following a slow recovery he was transferred to the ward dialysis-free with near normal indices.

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| **On reflection**,**Patient 2** presented with a severe burn injury, which included inhalational burns. Although young, the past medical history of type 1 diabetes mellitus, albeit uncomplicated to date, may have led to a degree of underlying chronic renal disease and predisposed to acute renal failure. The diabetes may also have increased the risk of infection. As he was burned in an enclosed space the potential for carbon monoxide and cyanide poisoning had to be considered and the possibility of electrical injury addressed. Aggressive management of his nosocomial pneumonia with initial broad-spectrum antibiotics improved outcome.**Patient 1** illustrated that patients with acute organophosphate poisoning may initially appear to recover quickly but the effects of acute poisoning may be prolonged and delayed neurological complications are reasonably common. Abrupt withdrawal of therapy may precipitate a sudden deterioration. The potential for delayed neurological developments must be borne in mind but in general the treatment of environmental hazards in the ICU may be particularly worthwhile as favourable outcomes may be anticipated in very critically ill patients. |

### Q1. A 27-year-old male presents to the ED following a fire in his bedroom. Drowsy, but haemodynamically stable, he is oxygenating well on room air. He has sustained approximately 20% full thickness burns to his hands, forearms and lower limb.

Top of Form

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|   | Your answers |

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| A. 40%-60% oxygen should be given to keep his oxygen saturation above 95% |  |
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| *The correct answer is :****False****- 100% oxygen via a non-rebreathing mask should be administered pending carboxyhaemoglobin results* |  |

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| B. As his oxygen saturations are excellent, he is unlikely to have had an inhalational injury or carbon monoxide poisoning |  |
|  |  |
| *The correct answer is :****False****- SaO<indice>2</indice> monitoring will not diagnose an inhalational injury or carbon monoxide poisoning* |  |

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| C. As he is stable, he will not need referral to a specialist burns centre |  |
|  |  |
| *The correct answer is :****False****- Burns to hands can be extremely debilitating and require management at a specialist centre to achieve optimal results* |  |

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| D. His drowsiness is secondary to the shock associated with burns |  |
|  |  |
| *The correct answer is :****False****- There is a high risk of carbon monoxide poisoning or cyanide poisoning in this patient. Consideration must also be given to the potential for recreational drug use, prescribed medications and the possibility of other intracerebral pathology, post ictal etc.* |  |

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| E. Persistent metabolic acidosis could be due to cyanide exposure |  |
|  |  |
| *The correct answer is :****True****- However, common causes of persistent metabolic acidosis in this setting include underresuscitation and carbon monoxide poisoning* |  |

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| **Your total score is 0/5** |
| Q2. Hypothermic patients:Top of Form

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|   | Your answers |

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| A. Are easily identified |  |
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| *The correct answer is :****False****- Symptoms may be vague; accurate thermometers must be used* |  |

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| B. Almost always have underlying contributing medical conditions |  |
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| *The correct answer is :****False****- Many older patients may have medical co-morbidities, but a large group are young, previously fit and healthy adults who have had environmental exposure through outdoor pursuits, immersion injuries etc.* |  |

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| C. Should all have empirical antibiotics |  |
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| *The correct answer is :****False****- Although sepsis can be a precipitant and a consequence of hypothermia, many cases of mild hypothermia are uncomplicated and do not require antibiotic therapy* |  |

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| D. Should be actively rewarmed if temperature is 30 °C (86 °F) |  |
|  |  |
| *The correct answer is :****True*** |  |

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| E. May present with non-specific symptoms such as confusion |  |
|  |  |
| *The correct answer is :****True*** |  |

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| **Your total score is 0/5** |
| Q3. A 42-year-old male is admitted to the Emergency Department following an accident in which his truck severed overhead power lines. He was thrown from the truck, and although initially unconscious, is now alert, orientated and haemodynamically stable.Top of Form

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|   | Your answers |

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| A. Normal ECG and troponin levels indicate no cardiac damage has occurred? |  |
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| *The correct answer is :****False****- Initial ECG and troponin levels may be normal. ECHO may be helpful, but the history indicates a need for continuous cardiac monitoring and serial ECG and troponins* |  |

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| B. He will require at least 24 hours cardiac monitoring |  |
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| *The correct answer is :****True****- This patient has been exposed to high voltage electricity and has a history of loss of consciousness, both of which indicate a need for cardiac monitoring* |  |

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| C. Lack of obvious burn damage on body surface suggests minimal electrical damage |  |
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| *The correct answer is :****False****- Body surface burns are a poor indicator of overall damage in electrical injuries* |  |

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| D. An elevated CK measurement is most likely secondary to prolonged convulsions at the time of electrical shock |  |
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| *The correct answer is :****False*** |  |

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| E. A rising lactate level is most likely secondary to prolonged convulsions at the time of electrical shock |  |
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| *The correct answer is :****False****- Prolonged seizure activity can elevate lactate, however levels will usually decline rapidly once seizures have ceased. A more likely and more serious reason for rising lactate levels is organ or visceral ischaemia* |  |

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| **Your total score is 0/5** |
| 4. Which of the following is true regarding near-drowning?Top of Form

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|   | Your answers |

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| A. Aspirated water is the main cause of asphyxia: the Heimlich manoeuvre is always performed |  |
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| *The correct answer is :****False****- Aspiration of water may not occur or may be insignificant: the Heimlich manoeuvre is used only if an airway obstruction with a solid foreign body is present or highly suspected* |  |

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| B. Initial normal chest X-rays in a patient suspected of having had a submersion injury excludes the development of late pulmonary complication |  |
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| *The correct answer is :****False****- Late onset severe pulmonary complications have been reported* |  |

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| C. Non-invasive ventilation is contraindicated in the near-drowning victim |  |
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| *The correct answer is :****False****- NIV may be used in an alert patient with compromised oxygenation but still with PCO<indice>2</INDICE> below 45 mmHg (6 kPa)* |  |

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| Electrolyte disturbances are rarely seen in survivors |  |
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| *The correct answer is :****True****- If they exist they tend to be rapidly corrected and are not observed in survivors* |  |

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| A single neurologic status examination performed on the Emergency Department admission has limited predictive value for neurologic outcome |  |
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| *The correct answer is :****True****- It is needed to repeat neurologic evaluations in the first 72 hours to have more reliable indicators of prognosis* |  |

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| **Your total score is 0/3** |
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Q5. Which of the following statements about paraquat intoxication is/are correct?Top of Form

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|   | Your answers |

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| A. Dermal exposure may induce severe systemic toxicity |  |
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| *The correct answer is :****True****- Paraquat is absorbed through injured or abraded skin and this may result in severe systemic toxicity, although rarely* |  |

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| B. Pulmonary injury is enhanced by high concentration of oxygen |  |
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| *The correct answer is :****True****- Oxygen aggravates lipid peroxidation reactions in the lung: the minimum concentration of oxygen to achieve a PO<indice>2</indice> of 60 mmHg is used* |  |

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| C. Within four hours of ingestion, Fuller's earth administration is clearly more effective than activated charcoal |  |
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| *The correct answer is :****False****- Both have been found to effectively adsorb paraquat: the comparative efficacy has not been determined* |  |

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| D. Paraquat levels are useful to establish the diagnosis and the prognosis |  |
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| *The correct answer is :****True*** |  |

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| E. Diquat and paraquat have identical pharmacokinetics, are both corrosive and cause renal failure |  |
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| *The correct answer is :****True****- Diquat and paraquat have similar properties concerning pharmacokinetics and the ability to produce corrosion and renal failure* |  |

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| **Your total score is 0/5** |
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