ride in high doses can overcome some of the adverse effects, it rarely restores normal cardiovascular status. Haemodynamic instability may respond to high doses of insulin given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and inotropic drugs.[133–148] Other potentially useful treatments include glucagon, vasopressin and phosphodiesterse inhibitors.[139,149]

Digoxin

Although cases of digoxin poisoning are fewer than those involving calcium channel and beta-blockers, the mortality rate from digoxin is far greater. Other drugs including calcium channel blockers and amiodarone can also cause plasma concentrations of digoxin to rise. Atrioventricular conduction abnormalities and ventricular hyperexcitability due to digoxin toxicity can lead to severe arrhythmias and cardiac arrest.

Patients at risk of cardiac arrest

Standard resuscitation measures and specific antidote therapy with digoxin-specific antibody fragments should be used if there are arrhythmias associated with haemodynamic instability.[150–163] Antibody-specific therapy may also be effective in poisoning from plants as well as Chinese herbal medications containing digitalis glycosides.[150,164,165] Digoxin-specific antibody fragments interfere with digoxin immunoassay measurements and can lead to overestimation of plasma digoxin concentrations.

Cyanide

Cyanide is generally considered to be a rare cause of acute poisoning; however, cyanide exposure occurs relatively frequently in patients with smoke inhalation from residential or industrial fires. Its main toxicity results from inactivation of cytochrome oxidase (at cytochrome a3), thus uncoupling mitochondrial oxidative phosphorylation and inhibiting cellular respiration, even in the presence of adequate oxygen supply. Tissues with the highest oxygen needs (brain and heart) are the most severely affected by acute cyanide poisoning.

Patients at risk of cardiac arrest

Patients with severe cardiovascular toxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation, including oxygen. Initial therapy should include a cyanide scavenger (either intravenous hydroxocobalamin or a nitrite – i.e., intravenous sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by intravenous sodium thiosulphate.[166–175] Hydroxocobalamin and nitrites are equally effective but hydroxocobalamin may be safer because it does not cause methaemoglobin formation or hypotension.

Modifications to BLS/ALS

In case of cardiac arrest caused by cyanide, standard ALS treatment will fail to restore spontaneous circulation as long as cellular respiration is blocked. Antidote treatment is needed for reactivation of cytochrome oxidase.

Carbon monoxide

Carbon monoxide poisoning is common. There were about 25,000 carbon monoxide related hospital admissions reported in the US in 2005.[176] Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if return of spontaneous circulation is achieved; however, hyperbaric oxygen therapy may be considered in these patients as it may reduce the risk of developing persistent or delayed neurological injury.[177–185] The risks inherent in transporting critically ill post-arrest patients to a hyperbaric facility may be significant, and must be weighed against the possibility of benefit on a case-by-case basis. Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least 7 years after the event; it is reasonable to recommend cardiology follow-up for these patients.[186,187]

8c. Drowning

Overview

Drowning is a common cause of accidental death in Europe. After drowning the duration of hypoxia is the most critical factor in determining the victim's outcome; therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. Immediate resuscitation at the scene is essential for survival and neurological recovery after a drowning incident. This will require provision of CPR by a bystander and immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes. Research into drowning is limited in comparison with primary cardiac arrest and there is a need for further research in this area.[188] These guidelines are intended for healthcare professionals and certain groups of lay responders that have a special interest in the care of the drowning victim, e.g., lifeguards.

Epidemiology

The World Health Organization (WHO) estimates that, worldwide, drowning accounts for approximately 450,000 deaths each year. A further 1.3 million disability-adjusted life-years are lost each year as a result of premature death or disability from drowning[189]; 97% of deaths from drowning occur in low- and middle-income countries.[189] In 2006 there were 312 accidental deaths from drowning in the United Kingdom[190] and 3582 in the United States,[191] yielding an annual incidence of drowning of 0.56 and 1.2 per 100,000 population, respectively.[192] Death from drowning is more common in young males, and is the leading cause of accidental death in Europe in this group.[189] Factors associated with drowning (e.g., suicide, traffic accidents, alcohol and drug abuse) varies between countries.[193]

Definitions, classifications and reporting

Over 30 different terms have been used to describe the process and outcome from submersion- and immersion-related incidents.[194] The International Liaison Committee on Resuscitation (ILCOR) defines drowning as "a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident".[195] Immersion means to be covered in water or other fluid. For drowning to occur, usually at least the face and airway must be immersed. Submersion implies that the entire body, including the airway, is under the water or other fluid.

ILCOR recommends that the following terms, previously used, should no longer be used: dry and wet drowning, active and passive drowning, silent drowning, secondary drowning and drowned versus near-drowned.[195] The Utstein drowning style should be used

when reporting outcomes from drowning incidents to improve consistency in information between studies.[195]

Pathophysiology

The pathophysiology of drowning has been described in detail.[195,196] In brief, after submersion, the victim initially breath holds before developing laryngospasm. During this time the victim frequently swallows large quantities of water. As breath holding/laryngospasm continues, hypoxia and hypercapnia develops. Eventually these reflexes abate and the victim aspirates water into their lungs leading to worsening hypoxaemia. Without rescue and restoration of ventilation the victim will become bradycardic before sustaining a cardiac arrest. The key feature to note in the pathophysiology of drowning is that cardiac arrest occurs as a consequence of hypoxia and correction of hypoxaemia is critical to obtaining a return of spontaneous circulation.

Treatment

Treatment of a drowning victim involves four distinct but interrelated phases. These comprise (i) aquatic rescue, (ii) basic life support, (iii) advanced life support, and (iv) post-resuscitation care. Rescue and resuscitation of the drowning victim almost always involves a multi-professional team approach. The initial rescue from the water is usually undertaken either by bystanders or those with a duty to respond such as trained lifeguards or lifeboat operators. Basic life support is often provided by the initial responders before arrival of the emergency medical services. Resuscitation frequently continues into hospital where, if return of spontaneous circulation is achieved, transfer to critical care often follows. Drowning incidents vary in their complexity from an incident involving a single victim to one that involves several or multiple victims. The emergency response will vary according to the number of victims involved and available resources. If the number of victims outweighs the available resources then a system of triage to determine who to prioritise for treatment is likely to be necessary. The remainder of this section will focus on the management of the individual drowning victim where there are sufficient resources available.

Basic life support

Aquatic rescue and recovery from the water. Always be aware of personal safety and minimize the danger to yourself and the victim at all times. Whenever possible, attempt to save the drowning victim without entry into the water. Talking to the victim, reaching with a rescue aid (e.g., stick or clothing), or throwing a rope or buoyant rescue aid may be effective if the victim is close to dry land. Alternatively, use a boat or other water vehicle to assist with the rescue. Avoid entry into the water whenever possible. If entry into the water is essential, take a buoyant rescue aid or flotation device.[197] It is safer to enter the water with two rescuers than alone. Never dive head first in the water when attempting a rescue. You may lose visual contact with the victim and run the risk of a spinal injury.

Remove all drowning victims from the water by the fastest and safest means available and resuscitate as quickly as possible. The incidence of cervical spine injury in drowning victims is very low (approximately 0.5%).[198] Spinal immobilisation can be difficult to perform in the water and can delay removal from the water and adequate resuscitation of the victim. Poorly applied cervical collars can also cause airway obstruction in unconscious patients.[199] Cervical spine immobilisation is not indicated unless signs of severe injury are apparent or the history is consistent with the possibility of severe injury.[200] These circumstances include a history of diving, water-slide use, signs of trauma or signs of alcohol intoxication. If the victim is pulseless and apnoeic remove them from the water as quickly as possible (even if a back support device is not available), while attempting to limit neck flexion and extension.

Rescue breathing. The first and most important treatment for the drowning victim is alleviation of hypoxaemia. Prompt initiation of rescue breathing or positive pressure ventilation increases survival.[201–204] If possible supplement rescue breaths/ventilations with oxygen.[205] Give five initial ventilations/rescue breaths as soon as possible.

Rescue breathing can be initiated whilst the victim is still in shallow water provided the safety of the rescuer is not compromised. It is likely to be difficult to pinch the victim's nose, so mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation.

If the victim is in deep water, open their airway and if there is no spontaneous breathing start in-water rescue breathing if trained to do so. In-water resuscitation is possible,[206] but should ideally be performed with the support of a buoyant rescue aid.[207] Give 10–15 rescue breaths over approximately 1 min.[207] If normal breathing does not start spontaneously, and the victim is <5 min of from land, continue rescue breaths while towing. If more than an estimated 5 min from land, give further rescue breaths over 1 min, then bring the victim to land as quickly as possible without further attempts at ventilation.[207]

Chest compression. The victim should be placed on a firm surface before starting chest compressions as compressions are ineffective in the water.[208,209] Confirm the victim is unresponsive and not breathing normally and then give 30 chest compressions. Continue CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be less effective and should be avoided.

Automated external defibrillation. Once CPR is in progress, if an AED is available, dry the victim's chest, attach the AED pads and turn the AED on. Deliver shocks according to the AED prompts.

Regurgitation during resuscitation. Although rescue breathing is difficult to perform perfectly on a drowning victim because of the need for very high inflation pressures or the presence of fluid in the airway, every attempt should be made to continue ventilation until advanced life support providers arrive. Regurgitation of stomach contents and swallowed/inhaled water is common during resuscitation from drowning.[210] If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible. Care should be taken if spinal injury is suspected but this should not prevent or delay life-saving interventions such as airway opening, ventilations and chest compressions. Abdominal thrusts can cause regurgitation of gastric contents and other life-threatening injuries and should not be used.[211]

Advanced life support

Airway and breathing. Give high-flow oxygen, ideally through an oxygen mask with reservoir bag, during the initial assessment of the spontaneously breathing drowning victim.[205] Consider non-invasive ventilation or continuous positive airway pressure if the victim fails to respond to treatment with high-flow oxygen.[212] Use pulse oximetry and arterial blood gas analysis to titrate the concentration of inspired oxygen. Consider early tracheal intubation and controlled ventilation for victims who fail to respond to these initial measures or who have a reduced level of consciousness. Take

care to ensure optimal preoxygenation before intubation. Use a rapid-sequence induction with cricoid pressure to reduce the risk of aspiration.[213] Pulmonary oedema fluid may pour from the airway and may need suctioning to enable a view of the larynx.

After the tracheal tube is confirmed in position, titrate the inspired oxygen concentration to achieve an SaO_2 of 94–98%.[205] Set positive end-expiratory pressure (PEEP) to at least 5–10 cm H_2O, however higher PEEP levels (15–20 cm H_2O) may be required if the patients is severely hypoxaemic.[214]

In the event of cardiopulmonary arrest protect the airway of the victim early in the resuscitation attempt, ideally with a cuffed tracheal tube – reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.

Circulation and defibrillation. Differentiating respiratory from cardiac arrest is particularly important in the drowning victim. Delaying the initiation of chest compressions if the victim is in cardiac arrest will reduce survival.

The typical post-arrest gasping is very difficult to distinguish from the initial respiratory efforts of a spontaneous recovering drowning victim. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable.[215] When available additional diagnostic information should be obtained from other monitoring modalities such as ECG trace, end-tidal CO_2, and echocardiography to confirm the diagnosis of cardiac arrest.

If the victim is in cardiac arrest, follow standard advanced life support protocols. If the victims core body temperature is less than 30 °C, limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above 30 °C (see Section 8d).

During prolonged immersion, victims may become hypovolaemic from the hydrostatic pressure of the water on the body. Give IV fluid to correct hypovolaemia. After return of spontaneous circulation, use haemodynamic monitoring to guide fluid resuscitation.

Discontinuing resuscitation efforts

Making a decision to discontinue resuscitation efforts on a victim of drowning is notoriously difficult. No single factor can accurately predict good or poor survival with 100% certainty. Decisions made in the field frequently prove later to have been incorrect.[216] Continue resuscitation unless there is clear evidence that such attempts are futile (e.g., massive traumatic injuries, *rigor mortis*, putrefaction etc), or timely evacuation to a medical facility is not possible. Neurologically intact survival has been reported in several victims submerged for greater than 60 min however these rare case reports almost invariably occur in children submerged in ice-cold water.[217,218]

Post-resuscitation care

Salt versus fresh water. Much attention has focused in the past on differences between salt-water and fresh-water drowning. Extensive data from animal studies and human case-series have shown that, irrespective of the tonicity of the inhaled fluid, the predominant pathophysiological process is hypoxaemia, driven by surfactant wash-out and dysfunction, alveolar collapse, atelectasis, and intrapulmonary shunting. Small differences in electrolyte disturbance are rarely of any clinical relevance and do not usually require treatment.

Lung injury. Victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) after submersion.[219] Although there are no randomised controlled trials undertaken specifically in this population of patients it seems reasonable to include strategies such as protective ventilation that have been shown to improve survival in patients with ARDS.[220] The severity of lung injury varies from a mild self-limiting illness to refractory hypoxaemia. In severe cases extracorporeal membrane oxygenation has been used with some success.[221,222] The clinical and cost effectiveness of these interventions has not been formally tested in randomised controlled trials.

Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit,[223] although they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.[200,224]

Hypothermia after drowning. Victims of submersion may develop primary or secondary hypothermia. If the submersion occurs in icy water (<5 °C or 41°F), hypothermia may develop rapidly and provide some protection against hypoxia. Such effects, however, have typically been reported after submersion of children in ice-cold water.[189] Hypothermia may also develop as a secondary complication of the submersion and subsequent heat loss through evaporation during attempted resuscitation (see Section 8d).

Case reports describing patients with severe accidental hypothermia have shown that survival is possible after either passive or active warming.[200] In contrast, there is evidence of benefit from induced hypothermia for comatose victims resuscitated from pre-hospital cardiac arrests.[225,226] To date, there is no convincing evidence to guide therapy in this patient group. A pragmatic approach might be to consider rewarming until a core temperature of 32–34 °C is achieved, taking care to avoid hyperthermia (>37 °C) during the subsequent period of intensive care (International Life Saving Federation, 2003).

Other supportive care. Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has been shown to alter outcome. In fact, signs of intracranial hypertension serve as a symptom of significant neurological hypoxic injury, and there is no evidence that attempts to alter the ICP will affect outcome.[200]

Follow-up. Cardiac arrhythmias may cause rapid loss of consciousness leading to drowning if the victim is in water at the time. Take a careful history in survivors of a drowning incident to identify features suggestive of arrhythmic syncope. Symptoms may include syncope (whilst supine position, during exercise, with brief prodromal symptoms, repetitive episodes or associated with palpitations), seizures or a family history of sudden death. The absence of structural heart disease at post-mortem examination does not rule the possibility of sudden cardiac death. Post-mortem genetic analysis has proved helpful in these situations and should be considered if there is uncertainty over the cause of a drowning death.[227–229]

8d. Accidental hypothermia

Definition

Accidental hypothermia exists when the body core temperature unintentionally drops below 35 °C. Hypothermia can be classified arbitrarily as mild (35–32 °C), moderate (32–28 °C) or severe (less than 28 °C).[230] The Swiss staging system[231] based on clinical signs can be used by rescuers at the scene to describe victims: stage I – clearly conscious and shivering; stage II – impaired consciousness without shivering; stage III – unconscious; stage IV – no breathing; and stage V – death due to irreversible hypothermia.

Diagnosis

Accidental hypothermia may be under-diagnosed in countries with a temperate climate. In persons with normal thermoregulation, hypothermia may develop during exposure to cold environments, particularly wet or windy conditions, and in people who have been immobilised, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is also increased by drug or alcohol ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness. Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient. A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature measured in the lower third of the oesophagus correlates well with the temperature of the heart. Epitympanic ('tympanic') measurement – using a thermistor technique – is a reliable alternative but may be lower than the oesophageal temperature if the environmental temperature is very cold, the probe is not well insulated, the external auditory canal is blocked or during cardiac arrest when there is no flow in the carotid artery.[232] Widely available 'tympanic' thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.[233] In the hospital setting, the method of temperature measurement should be the same throughout resuscitation and rewarming. Use oesophageal, bladder, rectal or tympanic temperature measurements.[234,235]

Decision to resuscitate

Cooling of the human body decreases cellular oxygen consumption by ~6% per 1 °C decrease in core temperature.[236] At 28 °C oxygen consumption is reduced by ~50% and at 22 °C by ~75%. In some cases, hypothermia can exert a protective effect on the brain and vital organs[237] and intact neurological recovery may be possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia. Beware of diagnosing death in a hypothermic patient because cold alone may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone is unreliable for declaring death. At 18 °C the brain can tolerate periods of circulatory arrest for ten times longer than at 37 °C. Dilated pupils can be caused by a variety of insults and must not be regarded as a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7 °C after immersion in cold water with prolonged CPR.[238] In another case, a severely hypothermic patient was resuscitated successfully after six and a half hours of CPR.[239]

In the pre-hospital setting, resuscitation should be withheld only if the cause of a cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible. In all other patients the traditional guiding principle that "no one is dead until warm and dead" should be considered. In remote wilderness areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting involve senior doctors and use clinical judgment to determine when to stop resuscitating a hypothermic arrest victim.

Resuscitation

All the principles of prevention, basic and advanced life support apply to the hypothermic patient. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult. Do not delay urgent procedures, such as inserting vascular catheters and tracheal intubation. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.[240]

Clear the airway and, if there is no spontaneous respiratory effort, ventilate the patient's lungs with high concentrations of oxygen. Consider careful tracheal intubation when indicated according to advanced life support guidelines. Palpate a central artery, look at the ECG (if available), and look for signs of life for up to 1 min before concluding that there is no cardiac output. Echocardiography or ultrasound with Doppler may be used to establish whether there is a cardiac output or peripheral blood flow. If there is any doubt about whether a pulse is present, start CPR immediately. Once CPR is under way, confirm hypothermia with a low-reading thermometer.

The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drugs given repeatedly.[241] The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. For instance, in severe hypothermic cardiac arrest, adrenaline may be effective in increasing coronary perfusion pressure, but not survival.[242,243] The efficacy of amiodarone is also reduced.[244] For these reasons, withhold adrenaline and other CPR drugs until the patient has been warmed to a temperature higher than approximately 30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared with normothermia intervals. As normothermia is approached (over 35 °C), standard drug protocols should be used. Remember to rule out other primary causes of cardiorespiratory arrest using the 4 Hs and 4 Ts approach (e.g., drug overdose, hypothyroidism, trauma).

Arrhythmias

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.[245] Once in hospital, severely hypothermic victims in cardiac arrest should be rewarmed with active internal methods. Arrhythmias other than VF tend to revert spontaneously as the core temperature increases, and usually do not require immediate treatment. Bradycardia may be physiological in severe hypothermia, and cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming.

The temperature at which defibrillation should first be attempted and how often it should be tried in the severely hypothermic patient has not been established. AEDs may be used on these patients. If VF is detected, give a shock at the maximum energy setting; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30 °C.[246] If an AED is used, follow the AED prompts while rewarming the patient. CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.[246]

Rewarming

General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital. In the field, a patient with moderate or severe hypothermia (Swiss stages ≥ II) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.[241] Wet clothes should be cut off rather than stripped off; this will avoid excessive movement of the victim. Conscious victims can mobilise as exercise rewarms a person more rapidly than shivering. Exercise can increase any after-drop, i.e., further cooling after removal from a cold environment. Somnolent or comatose victims have a low threshold for developing VF or pulseless VT and should be

immobilised and kept horizontal to avoid an after-drop or cardiovascular collapse. Adequate oxygenation is essential to stabilise the myocardium and all victims should receive supplemental oxygen. If the patient is unconscious, the airway should be protected. Pre hospital, prolonged investigation and treatments should be avoided, as further heat loss is difficult to prevent.

Rewarming may be passive, active external, or active internal. Passive rewarming is appropriate in conscious victims with mild hypothermia who are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, cap and warm environment. The application of chemical heat packs to the trunk is particularly helpful in moderate and severe hypothermia to prevent further heat loss in the pre hospital setting. If the patient is unconscious and the airway is not secured, insulation should be arranged around the patient in the recovery (lateral decubitus) position. Rewarming in the field with heated intravenous fluids and warm humidified gases is not efficient. Infusing a litre of 40 °C warm fluid to a 70 kg patient at 28 °C elevates the core temperature by only about 0.3 °C.[241] Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available. In general, alert hypothermic and shivering victims without an arrhythmia may be transported to the nearest hospital for passive rewarming and observation. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming.

Several active in-hospital rewarming techniques have been described, although in a patient with stable circulation no technique has shown better survival over others. Active external rewarming techniques include forced air rewarming and warmed (up to 42 °C) intravenous fluids. These techniques are effective (rewarming rate 1–1.5 °C h^{-1}) in patients with severe hypothermia and a perfusing rhythm.[247,248] Even in severe hypothermia no significant after-drop or malignant arrhythmias have been reported. Rewarming with forced air and warm fluid has been widely implemented by clinicians because it is easy and effective. Active internal rewarming techniques include warm humidified gases; gastric, peritoneal, pleural or bladder lavage with warmed fluids (at 40 °C), and extracorporeal rewarming.[237,249–253]

In a hypothermic patient with apnoea and cardiac arrest, extracorporeal rewarming is the preferred method of active internal rewarming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8–12 °C h^{-1}.[253] Survivors in one case-series had an average of 65 min of conventional CPR before cardiopulmonary bypass,[254] which underlines that continuous CPR is essential. Unfortunately, facilities for extracorporeal rewarming are not always available and a combination of rewarming techniques may have to be used. It is advisable to contact the destination hospital well in advance of arrival to make sure that the unit can accept the patient for extracorporeal rewarming. Extracorporeal membrane oxygenation (ECMO) reduces the risk of intractable cardiorespiratory failure commonly observed after rewarming and may be a preferable extracorporeal rewarming procedure.[255]

During rewarming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential. Avoid hyperthermia during and after rewarming. Although there are no formal studies, once ROSC has been achieved use standard strategies for post-resuscitation care, including mild hypothermia if appropriate (Section 4g).[24a]

Avalanche burial

In Europe and North America, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Death from avalanches is due to asphyxia, trauma and hypothermia. Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and should be informed by the likelihood of survival.[256] Avalanche victims are not likely to survive when they are:

- buried >35 min and in cardiac arrest with an obstructed airway on extrication;
- buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of <32°;
- buried initially and in cardiac arrest on extrication with an initial serum potassium of >12 mmol.

Full resuscitative measures, including extracorporeal rewarming, when available, are indicated for all other avalanche victims without evidence of an unsurvivable injury.

8e. Hyperthermia

Definition

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions, or secondary to endogenous heat production.

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, to heat stroke (HS) and finally multiorgan dysfunction and cardiac arrest in some instances.[257]

Malignant hyperthermia (MH) is a rare disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics and depolarizing muscle relaxants.[258,259]

The key features and treatment of heat stress and heat exhaustion are included in Table 8.2.

Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature above 40.6 °C, accompanied by mental state change and varying levels of organ dysfunction. There are two forms of HS: classic non-exertional heat stroke (CHS) occurs during high environmental temperatures and often effects the elderly during heat waves[260]; The 2003 heatwave in France was associated with an increased incidence of cardiac arrests in those over 60-years old.[261] Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity usually effects healthy young adults.[262] Mortality from heat stroke ranges between 10 and 50%.[263]

Predisposing factors

The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms and limited social support. There are several risk factors: lack of acclimatization, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema,

Table 8.2
Heat stress and heat exhaustion.

Condition	Features	Treatment
Heat stress	Normal or mild temperature elevation Heat oedema: swelling of feet and ankles Heat syncope: vasodilation causing hypotension Heat cramps: sodium depletion causing cramps	Rest Elevation of oedematous limbs Cooling Oral rehydration Salt replacement
Heat exhaustion	Systemic reaction to prolonged heat exposure (hours to days) Temperature > 37 °C and < 40 °C Headache, dizziness, nausea, vomiting, tachycardia, hypotension, sweating muscle pain, weakness and cramps Haemoconcentration Hyponatraemia or hypernatraemia May progress rapidly to heat stroke	As above Consider IV fluids and ice packs for severe cases

scleroderma, burn, cystic fibrosis), hyperthyroidism, phaeochromocytoma and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Clinical presentation

Heat stroke can resemble septic shock and may be caused by similar mechanisms.[264] A single centre case-series reported 14 ICU deaths in 22 heat stroke patients admitted to ICU with multiple organ failure.[265] Features include:

- core temperature 40.6 °C or more;
- hot, dry skin (sweating is present in about 50% of cases of exertional heat stroke);
- early signs and symptoms, e.g., extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea;
- cardiovascular dysfunction including arrhythmias[266] and hypotension;
- respiratory dysfunction including ARDS[267];
- central nervous system dysfunction including seizures and coma[268];
- liver and renal failure[269];
- coagulopathy[267];
- rhabdomyolysis.[270]

Other clinical conditions need to be considered, including:

- drug toxicity[271,272];
- drug withdrawal syndrome;
- serotonin syndrome[273];
- neuroleptic malignant syndrome[274];
- sepsis[275];
- central nervous system infection;
- endocrine disorders, e.g., thyroid storm, phaeochromocytoma.[276]

Management

The mainstay of treatment is supportive therapy based on optimizing the ABCDEs and rapidly cooling the patient.[277–279] Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39 °C. Patients with severe heat stroke need to be managed in a critical-care setting. Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities as described in Section 8a.

Cooling techniques

Several cooling methods have been described, but there are few formal trials to determine which method is best. Simple cooling techniques include drinking cool fluids, fanning the completely undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) may also be useful. Surface cooling methods may cause shivering. In cooperative stable patients, immersion in cold water can be effective[280]; however, this may cause peripheral vasoconstriction, shunt blood away from the periphery and reduce heat dissipation. Immersion is also not practical in the sickest patients.

Further techniques to cool patients with hyperthermia are similar to those used for therapeutic hypothermia after cardiac arrest (see Section 4g).[24a] Cold intravenous fluids will decrease body temperature. Gastric, peritoneal,[281] pleural or bladder lavage with cold water will lower the core temperature. Intravascular cooling techniques include the use of cold IV fluids,[282] intravascular cooling catheters[283,284] and extracorporeal circuits,[285] e.g., continuous veno-veno haemofiltration or cardiopulmonary bypass.

Drug therapy in heat stroke

There are no specific drug therapies in heat stroke to lower core temperature. There is no good evidence that antipyretics (e.g., non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling.[286] Dantrolene (see below) has not been shown to be beneficial.[287–289]

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarizing neuromuscular blocking drugs, occurring during or after anaesthesia.[290] Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.[291]

Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.[292]

Modifications to cardiopulmonary resuscitation and post-resuscitation care

There are no specific studies on cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard procedures for basic

and advanced life support and cool the patient. Cooling techniques similar to those used to induce therapeutic hypothermia should be used (see Section 4g).[24a] There are no data on the effects of hyperthermia on defibrillation threshold; therefore, attempt defibrillation according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.[293,294] The risk of unfavourable neurological outcome increases for each degree of body temperature >37 °C.[295] Provide post-resuscitation care according to normal guidelines.

8f. Asthma

Introduction

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma.[296] The worldwide prevalence of asthma symptoms ranges from 1 to 18% of the population with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia).[296] International differences in asthma symptom prevalence appears to be decreasing in recent years, especially in adolescents.[297] The World Health Organisation has estimated that 15 million disability-adjusted life-years (DALYs) are lost annually from asthma, representing 1% of the global disease burden. Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence.[296] National and international guidance for the management of asthma already exists.[296,298] This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity.[299] Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation;
- a hospitalisation or emergency care for asthma in the past year[300];
- low or no use of inhaled corticosteroids[301];
- an increasing use and dependence of beta-2 agonists[302];
- anxiety, depressive disorders and/or poor compliance with therapy.[303]

Causes of cardiac arrest

Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in those with asthma has been linked to:

- severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths);
- cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia.[304] Arrhythmias can also be caused by stimulant drugs (e.g., beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e., auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

Diagnosis

Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy. SaO_2 may not reflect progressive alveolar hypoventilation, particularly if oxygen is being given. The SaO_2 may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation and may initially increase intrapulmonary shunting.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,[305] pneumonia, foreign bodies, pulmonary embolism, bronchiectasis and subglottic mass.[306]

The severity of an asthma attack is defined in Table 8.3.

Key interventions to prevent arrest

The patient with severe asthma requires aggressive medical management to prevent deterioration. Base assessment and treatment on an ABCDE approach. Patients with SaO_2 < 92% or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement. Experienced clinicians should treat these high-risk patients in a critical-care area. The specific drugs and the treatment sequence will vary according to local practice.

Oxygen

Use a concentration of inspired oxygen that will achieve an SaO_2 94–98%.[205] High-flow oxygen by mask is sometimes necessary.

Table 8.3
The severity of asthma.

Asthma	Features
Near-fatal	Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures
Life-threatening	Any one of: PEF < 33% best or predicted bradycardia SpO_2 < 92%, dysrhythmia PaO_2 < 8 kPa, hypotension Normal $PaCO_2$ (4.6–6.0 kPa (35–45 mmHg)), exhaustion Silent chest, confusion Cyanosis, coma Feeble respiratory effort
Acute severe	Any one of: PEF 33–50% best or predicted Respiratory rate > 25 min^{-1} Heart rate > 110 min^{-1} Inability to complete sentences in one breath
Moderate exacerbation	Increasing symptoms PEF > 50–75% best or predicted No features of acute severe asthma
Brittle	Type 1: wide PEF variability (>40% diurnal variation for >50% of the time over a period >150 days) despite intense therapy Type 2: sudden severe attacks on a background of apparently well controlled asthma

PEF, peak expiratory flow.