Severe accidental hypothermia: a brief review of the literature

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Essential learning points

- Severe accidental hypothermia is a core temperature <28°C
- It is a reversible cause of cardiac arrest because it is treatable in appropriate patients if the clinician recognises the features and knows what to do
- Refractory arrhythmias and cardiac arrest are the biggest problems in the early stages of resuscitation
- Management involves advanced techniques and will require an admission to intensive care
- With correct patient selection and management, the prognosis is excellent

INTRODUCTION

Hypothermia is defined as a core body temperature below 35°C.⁽¹⁾ The term 'accidental' is used to differentiate unintentional hypothermia from the deliberate hypothermia that is used in medical practice. Hypothermia can be classified clinically as primary (accidental) caused by exposure to a cold environment, or secondary due to a variety of medical conditions such as spinal cord injury, pancreatitis, myxoedema and sepsis.

Severe accidental hypothermia is a core temperature <28°C and is an uncommon occurrence. Nevertheless, it is important to be aware of the special requirements of these patients, particularly if accompanied by cardiac arrest. Patients with severe accidental hypothermia may appear to be dead, yet have the potential for full recovery with appropriate management. This has given rise to the guiding principle that 'no one is dead until warm and dead'.⁽¹⁾ A prolonged period of cardiopulmonary resuscitation (CPR) may be required, yet patients have survived even after 6.5 hours of CPR.⁽²⁾ Passive methods of rewarming are ineffective in these cases. Invasive methods are the mainstay of treatment, but may only be available at specialist centres. Following resuscitation, the patient is likely to need a prolonged period in intensive care and rehabilitation. In many cases, the prognosis can be excellent if appropriate measures are taken promptly. This article will review the current published best practice for these cases. It can be read in conjunction with the case report published in this issue in which local staff successfully managed a case a few years ago. (See pages 184 to 186)

A BRIEF HISTORY

The effects of exposure to extreme cold or wet have been recognised for thousands of years. The signs were well described by one of Napoleon's surgeons in his journal written during the retreat from Moscow, and deaths from cold used to be collected in official statistics in the USA and the UK. Hypothermia as an entity could not be diagnosed until it became possible to measure body temperature in the late 19th century.⁽³⁾ However, it was not until the 1960s, when a report described 23 incidents which produced 25 deaths, that it became clear that exposure to a more temperate climate can also lead to hypothermia particularly if other factors are present such as wetting, wind and exhaustion.^(4,5) This is not surprising. The ambient temperature in the UK is usually significantly below body temperature, even on a summer's day. Therefore, a person who is not participating in physical activity will tend to cool down to the environmental temperature unless well insulated. Although hypothermia is seen as an outdoor hazard, it has been reported in an unconscious patient rescued from an air-conditioned building.⁽⁶⁾

CATEGORIES OF HYPOTHERMIA

Accidental hypothermia can be categorised by speed of onset and severity.

Speed of onset⁽²⁾

Grade of hypothermia by speed of onset

- *Acute hypothermia* (also called immersion hypothermia) is caused by sudden exposure to overwhelming cold such as immersion in cold water or entrapment in an avalanche. The body cools rapidly before energy reserves are exhausted. There has been no time for a cold diuresis (see below).
- *Sub-acute hypothermia* (exhaustion hypothermia) occurs over several hours. It is caused by exposure to moderately cold environments, particularly in the presence of wet and/or wind, in combination with a lack of food and physical exhaustion. Unlike in acute hypothermia, glucose stores are depleted so that heat can no longer be generated by shivering.
- *Chronic hypothermia* occurs in conditions of mild cold and comes on over a period of days or weeks. It is mainly seen in the elderly.

Severity

This is graded according to core body temperature.^(7,8)

Severity of hypothermia by core body temperature

- Mild (35-32°C)
- Moderate (32-28°C)
- Severe (<28°C)
- A fourth category of profound is sometimes used to denote a temperature of <20°C

A practical way for on-site staging of hypothermia is that of the International Commission for Mountain Emergency Medicine (ICAR-MEDCOM) that relates the clinical signs to body temperature (see box). This is particularly useful as it is unlikely that it will be possible to measure core temperature in the field. The patient's temperature range can be estimated from the clinical features.⁽⁹⁾ Currently, 13.7°C is the lowest recorded body temperature due to accidental hypothermia from which an adult has survived and made a complete recovery.⁽¹⁰⁾ The lowest temperature for infant survival is 14.8°C,⁽¹¹⁾ and 14.2°C for a child.⁽¹²⁾

ICAR-MEDCOM on-site staging of hypothermia

- Stage I – Clearly conscious and shivering (35-32°C)
- Stage II – Impaired consciousness without shivering (32-28°C)
- Stage III – Unconscious (28-24°C)
- Stage IV – Apparent death (24-13.7°C)
- Stage V – Death due to irreversible hypothermia (<13.7°C)

PHYSIOLOGICAL EFFECTS OF HYPOTHERMIA^(7,8,13,14)

Thermogenesis by shivering can increase heat production by 2-5 times. Peak shivering occurs at a body temperature of 35°C. This gradually decreases as the temperature falls but when it actually stops varies in different reports.⁽¹⁴⁾ This may reflect body energy supplies, as shivering cannot continue when glycogen stores are depleted, which usually occurs when the body temperature is in the low 30s.

CARDIAC EFFECTS OF SEVERE ACCIDENTAL HYPOTHERMIA

Cardiovascular system

The initial response to cold is vasoconstriction, which causes blood to be shunted from the periphery to the core. This protective effect is prevented by ethanol. Vasoconstriction fails when the temperature is <24°C and heat loss increases further. There is an initial tachycardia in response to the physical stress and the increased cardiac output required for shivering. This response fails with progressive hypothermia so that at 25°C, the cardiac index is 45% normal.

The greatest problem in the management of severe hypothermia is the effect on cardiac conduction.⁽¹⁵⁾ At temperatures <32°C, the most characteristic ECG abnormality is the J (Osborn) Wave at the end of the QRS complex (see figure). The heart rate gradually slows, reaching about 50% of the normal resting rate at 28°C. This is caused by decreased spontaneous depolarisation of pacemaker cells, so it is therefore resistant to atropine. At lower temperatures, the bradycardia increases, reaching about ten beats per minute at 20°C.⁽¹⁴⁾ There is a high likelihood of sudden asystole when the temperature is <24°C.^(14,15) Atrial fibrillation is common, but usually converts back to sinus rhythm spontaneously when the heart is rewarmed. A cardiac output may still be

present at body temperatures well below 28°C. For example, a casualty retrieved from a mountain by Keswick Mountain Rescue team in 2010 had a temperature of 24°C and was in slow atrial fibrillation.⁽¹⁶⁾

Below 32°C, all types of arrhythmias may be seen. Conduction delay through the AV node leads to any degree of atrioventricular block. ST elevation or depression and T-wave inversion may also occur, indicating increasing myocardial acidosis and ischaemia. The duration of QRS complexes increases. QT prolongation occurs,⁽¹⁵⁾ and can persist for days after rewarming.⁽¹⁴⁾

Ventricular fibrillation (VF) is increasingly likely to occur spontaneously as the temperature falls below about 29°C.⁽¹³⁾ Severe hypothermia decreases the myocardial fibrillatory threshold so that VF may be triggered just by seemingly innocuous stimuli such as moving the casualty,⁽¹⁷⁾ or some invasive procedures, eg insertion of a central venous catheter. Patients in ICAR-MEDCOM Stage II or worse should be kept lying horizontal as raising the legs can cause an influx of cold blood from the peripheries into the core and this can be enough to lower the cardiac temperature sufficiently to trigger VF.⁽¹⁾

Should hypothermic patients be intubated?

It has been suggested that VF can be precipitated by tracheal intubation. However, reports from three large series of patients suggests that this risk may have been overstated and is in fact related to a failure to pre-oxygenate the patient.⁽²⁾ In one series, intubation was performed on 117 patients, of which 97 had a temperature of <32°C. All were preoxygenated and no arrhythmias occurred.⁽¹⁸⁾ The relative safety of intubation in the presence of adequate oxygenation is illustrated by a study on anaesthetised dogs.⁽¹⁹⁾ The animals were intubated and extubated every two degrees starting at 27°C. There was only one episode of VF during 42 intubations performed at temperatures <28°C, whereas there were eight separate episodes of spontaneous VF unrelated to intubation in five dogs.

Treating arrhythmias

All arrhythmias are extremely resistant to defibrillation and all current drugs. Once VF has occurred, a review of case reports suggests that successful defibrillation is sometimes possible in patients with temperatures at or below 30°C.^(20,21) If it occurs,

Severe accidental hypothermia

refractory VF usually continues until the heart rewarms to 28–30°C. Many case reports illustrate the ineffectiveness of traditional methods for treating VF in severely hypothermic patients. In one such case, there were 13 attempts at defibrillation, five doses of adrenaline, two doses of atropine, and two doses of amiodarone administered in the first hour.⁽²²⁾ All were unsuccessful.

Resuscitation Council guidelines state that defibrillation can be attempted if an ECG monitor indicates an appropriate arrhythmia, but if VF/VT persists after three shocks, further defibrillation should not be attempted until the core temperature is above 30°C.⁽¹⁾ Adrenaline, amiodarone and atropine should be withheld until the patient has been rewarmed to 30°C. Once this temperature has been reached, the drug administration intervals should be doubled in comparison with normothermia because drug metabolism is slower, leading to potentially toxic levels if repeated doses are given.⁽¹⁾

In an animal model, subjects with a temperature as low as 30°C exhibited a better response to defibrillation than normothermic animals.^(23,24) However, this picture is not seen in clinical situations. This may be because in addition to species differences, study animals are usually anaesthetised beforehand and this may affect outcome. In addition, the duration of hypothermia in these experiments is considerably less than that seen in the clinical setting.

Normothermic patients who have suffered a cardiac arrest are at increased risk for re-fibrillation.^(25,26) The situation is the same in hypothermia, particularly whilst the cardiac temperature (rather than the body temperature) is below about 30°C for the reasons described above.

Central nervous system

In mild hypothermia, patients are often lethargic or confused and may exhibit altered states of judgement. The phenomenon of 'paradoxical undressing', sometimes seen on the mountains, is when the sufferer removes their warm clothing even though they are literally freezing to death. This is believed to be due to fatiguing of peripheral vasoconstriction so that the peripheries become flooded with warmer blood from the core.⁽²⁷⁾ Reflexes become increasingly sluggish as body temperature falls and become absent as the temperature falls below about 28°C. The pupils become dilated and unresponsive to light at this temperature.⁽¹⁴⁾ The EEG becomes flat below about 20°C. Cerebrovascular autoregulation remains intact

until the temperature drops below 25°C.^(6,14) Below 35°C, there is a decrease in cerebral metabolism by 6–10% per 1°C fall in core temperature.^(1,8) This decrease in cerebral oxygen requirements protects the brain against ischaemic damage after cardiac arrest. At a body temperature of 20°C, the ischaemic tolerance of the brain is increased tenfold compared to normothermic conditions.⁽²⁸⁾

Respiratory system

As the body temperature falls, the tidal volume, respiratory rate, pulmonary compliance and thoracic elasticity decrease. The respiratory rate may only be five breaths per minute when the body temperature is below 30°C. The cough reflex is obtunded and there is an increase in the volume and viscosity of secretions. This can lead to atelectasis. Hypoxaemia is therefore likely to be present. It should be remembered when ventilating these patients that CO₂ production falls by 50% for each 8°C fall in temperature, ie it is about 50% of normal at 30°C. Nevertheless, there can be a respiratory acidosis due to centrally mediated respiratory depression. The oxygen-haemoglobin dissociation curve shifts to the left leading to decreased oxygen release from haemoglobin into the tissues at low partial pressures of oxygen. Body oxygen consumption falls by approximately 6% per 1°C fall in core temperature and reaches 50% at 28°C, 75% at 22°C and 92% at 10°C.^(1,8)

Renal, fluid balance, acid-base and electrolytes

There is an initial cold-induced diuresis. This is partly due to the relative central hypervolaemia resulting from peripheral vasoconstriction, but also from a reduction in antidiuretic hormone release and an absence of thirst. The presence of ethanol will double the diuresis. Acute renal failure is seen in almost half of the patients admitted to an intensive care unit (ICU).⁽¹⁴⁾ Changes in vascular permeability lead to a loss of plasma into the extravascular space, which exacerbates the hypovolaemia caused by the diuresis.⁽¹⁴⁾ This causes haemoconcentration, with an increase in haematocrit of about 2% for every 1°C fall in temperature. Renal blood flow and glomerular filtration rate fall to 50% at 27°C. Blood urea and creatinine levels rise reflecting dehydration. Hyperglycaemia is common and is due to catecholamine-induced glycogenolysis, decreased insulin release and inhibition of insulin transport. This exacerbates the hypovolaemia by causing an osmotic diuresis.

Hypothermia can cause a mixed acidosis. In addition to a respiratory acidosis (mentioned above), there is a metabolic component due to lactate from shivering and poor tissue perfusion. Hepatic function is also depressed so lactate is not metabolised. Blood gases are difficult to interpret in hypothermic patients. The solubility of CO₂ and O₂ in plasma increases, and CO₂ production falls. Blood gas samples are analysed at 37°C. When evaluating and acting on the blood gas results, it is important to know whether they are corrected or not. If the arterial PaCO₂ and pH are reported at the patient's temperature, the pH will be higher and the PaCO₂ lower than in normothermia, but there is little data on what the normal should be in hypothermia. It is therefore easier to appreciate acid-base disturbance if the results are uncorrected. O₂ is different because the left shift of the oxygen-haemoglobin dissociation curve means that for any given oxygen content, the PaO₂ will be lower than in normothermia. When the blood sample is rewarmed to 37°C, the dissociation curve shifts back, but since the oxygen content has not changed, the PaO₂ will rise and appear artificially high. Therefore, to maintain true PaO₂ in the normal range, the measured PaO₂ should be corrected for current body temperature.^(29,30)

Serum electrolytes fluctuate with temperature, duration of exposure, and the rewarming technique selected. Both membrane permeability and sodium-potassium pump efficiency change with temperature. Plasma electrolyte levels are also affected by ongoing fluid shifts, the state of hydration prior to cooling, rehydration, and endocrine or gastrointestinal dysfunction. Hyperkalaemia has a particular significance as a prognostic indicator (see below).

Blood

There are a number of reports of disseminated intravascular coagulopathy with no obvious cause other than the hypothermia.⁽¹⁴⁾ The action of clotting factors is impaired at low temperature. For example, the activated partial thromboplastin time is increased by 50% at 29°C. This may be missed in the laboratory as the blood is warmed to 37°C before testing. Importantly, the reversible clotting defect created by hypothermia cannot be corrected by transfusion, as the infused clotting factors will cool down to body temperature and fail to function. Rewarming will reactivate the clotting mechanism. Platelet dysfunction occurs, and thrombocytopaenia develops possibly due to sequestration of platelets in the spleen.

Other effects

Rhabdomyolysis has been described as a complication following severe hypothermia and appears to be related to immobility.⁽⁷⁾ The extremities are vulnerable to frostbite due to the peripheral vasoconstriction, hypoperfusion, and haemoconcentration leading to 'sludging' of red blood cells in the small blood vessels.⁽⁷⁾

IN WHICH PATIENTS SHOULD RESUSCITATION BE ATTEMPTED?

In spite of the guiding principle that 'no one is dead until they are warm and dead', in view of the costs, time and effort involved, there is a responsibility to ensure that, where possible, attempts are only made to resuscitate patients who stand a chance of being resuscitated. It is actually fair to say that sometimes 'people are dead when they're cold and dead'.⁽³¹⁾

A patient who is severely hypothermic often appears dead with a very slow, small volume, irregular pulse and unrecordable blood pressure. Nevertheless, although extremely low, the cardiac output but may be just sufficient to meet minimal metabolic demands. Triage criteria can be used to help differentiate those who appear dead from those who really are dead. Four factors should be considered as contraindications to resuscitation when assessing non-perfusing, severely hypothermic victims.

Reasons not to start resuscitation⁽¹⁾

- Presence of asphyxia, eg avalanche victims,⁽³²⁾ drowning⁽³⁸⁾
- Presence of injuries incompatible with life
- The chest is incompressible, as this precludes closed-chest CPR
- Probable core temperature below 10-12°C

The presence of extreme hyperkalaemia is generally believed to reflect a period of asphyxia prior to the onset of hypothermia.⁽³²⁾ A level of >10mmol/L has been seen as an indicator of non-survival in an animal model,⁽³³⁾ and human case reviews.^(34,35) However, a child with a potassium of 11.8mmol/L was resuscitated from a temperature of 14.2°C,⁽¹²⁾ so the upper limit may be different in children. The current recommendation for avalanche victims is a cut-off of 12 mmol/L.^(1,9,33) Relevant factors that can raise plasma potassium and potentially lead to an error in triage are suxamethonium,⁽³⁶⁾ and compression injury.

Victims of submersion may develop hypothermia. If this occurs in icy water (<5°C), hypothermia may develop sufficiently quickly to provide some neurological protection against hypoxia,⁽¹⁾ particularly in children.⁽³⁷⁾ If the water temperature is >6°C, survival is unlikely if submersion is >30 minutes. By contrast, if the water temperature is 6°C or below, the survival time is extended to 90 minutes, beyond which survival is extremely unlikely.⁽³⁸⁾

PREHOSPITAL MANAGEMENT^(1,8)

The rescuer can expect that casualties with ICAR-MEDCOM Stage II or worse will have an irritable myocardium, hypovolaemia and a large temperature gradient between the periphery and the core. Any injuries should be stabilised, and the casualty should be immobilised and handled carefully. Oxygen should be administered, as requirements will increase as the body rewarms, particularly if shivering occurs. The body should be dried and insulated. Wet clothes should be cut off to prevent excessive movement of the casualty that can occur during undressing.

Conscious patients who are mildly hypothermic should be given hot sweet drinks to help with rewarming and provide a substrate to allow shivering to occur. They can be allowed to mobilise as movement increases heat production and is more effective at rewarming than shivering. By contrast, movement can be risky in more severely hypothermic casualties because muscle activity will pump cold blood from the peripheries back to the core. The airway must be patent, and protected if necessary with intubation. This will also ensure good oxygenation. It is important to remember that the pressure in

Continues on page 187

Case report and commentary

Andrzej Szymczakowski, MD; Paul Grout, FCEM; Commentary by Lucy Belson, RGN

A middle-aged woman was rescued from a river on a freezing night. At the scene, her Glasgow Coma Score (GCS) was 6/15 (2,2,2), with a blood pressure of 130/90mmHg, a heart rate 56/min, a respiratory rate of 12 breaths/min and an SpO₂ of 99% on 10 l/min of oxygen via facemask. The transport to hospital took approximately 40 minutes. In the ambulance, she suffered an asystolic cardiac arrest. CPR was commenced, her trachea was intubated and 1mg of iv adrenaline was administered.

Staff in hospital deployed a mechanical cardiac compression device (Auto-Pulse®, Zoll) because it was assumed that she would need a prolonged period of cardiorespiratory support because of her hypothermia (20.7°C on admission). This provided sufficient cardiac output to produce a palpable radial pulse. Core warming was commenced by use of an intravenous infusion of 0.9% saline through a fluid warmer, gastric/bladder lavages and external warming with the use of a warming blanket. Intermittent positive pressure ventilation with humidified oxygen was started approximately 40 minutes post-admission and radial artery and internal jugular vein cannulation was performed. After insertion of the arterial catheter, the direct arterial pressure remained at 60/20mmHg so it was decided to repeat the dose of 1mg adrenaline, which resulted in increase of arterial pressure to 110/40mmHg. After two hours of continuous bladder/gastric lavage and mechanical ventilation with 100% humidified oxygen, the core temperature increased from 21°C to 25°C.

We interrupted chest compression only for essential measures such as central vein cannulation, chest X-ray and battery changes for the Auto-Pulse machine. Initial arterial gases showed a lactic acidosis, good oxygen exchange, normocapnia and a significant hypokalaemia. In the initial two hours, 4mg of adrenaline was given in 1mg increments (achieving pressures of 100/40mmHg). Following this period, we transferred to the intensive care unit (ICU). We placed a haemofiltration line in the right femoral vein for access for veno-veno haemofiltration (CVVH) as a further means of core warming.

We used a continuous infusion of adrenaline (0.44mg/kg/min), potassium (50mmol/3h), magnesium (16mmol/2h) and insulin (6-10u/h) during this initial period after admission to ICU. Haemofiltration was started with an exchange rate of 5,000ml/hour and with fluid removal of 100ml/h. The filter temperature (the temperature of substitution fluid) was set at the maximum setting of 38.5°C and an infusion of normal saline, through a fluid warmer, at a rate of 100ml/h, maintained a neutral fluid balance: 5,000 units of heparin were added to the priming fluid and further doses were to be guided by activated clotting time measurement, after a low dose heparin infusion (4u/kg/h) had begun. The core temperature had risen to 28°C within two hours, but the patient remained in asystole and unresponsive to several attempts of external cardiac pacing.

Four and a half hours after resuscitation had been commenced, all available batteries for the Auto-Pulse and physical strength of the available staff to substitute for battery power had been exhausted. Failure seemed imminent. At this point, it was felt that there was little to be lost by deviating from standard protocols and we used a single 200J biphasic DC shock in asystole. To our surprise and relief, spontaneous sinus rhythm returned for a few minutes but

then changed to ventricular fibrillation (VF). A second defibrillation was unsuccessful so it was necessary to continue with chest compressions and use amiodarone, again a desperate measure outwith the normal recommendations. A third DC defibrillation resulted in a sinus bradycardia and restoration of sustained cardiac output. A significant amount of adrenaline (3.26mg/kg/min) and noradrenaline was needed together with 3mg of atropine to maintain satisfactory blood pressure during the first ten to 15 minutes of spontaneous circulation. The broad ECG complexes initially observed on the monitor narrowed over the first 30 minutes of spontaneous circulation. When cardiac output had improved, we sedated the patient: 100mls of blood-stained urine was noted in the urinary bag, triggering a decision to give 125mls of 20% mannitol, which effectively provoked a diuretic response. CVVH, fluid infusion and notropes were continued.

Haemofiltration was discontinued after eight hours. At this stage, it was possible to reduce inotropic support. We introduced forced alkaline diuresis with normal saline (100ml/hr) and 1.26% sodium bicarbonate (100ml/hr) infusions and four incremental doses of 20mg frusemide to prevent the renal complications of rhabdomyolysis. We are pleased to report that the patient was fit to be discharged from hospital a few days later.

DISCUSSION

Rarely do NHS staff get exposed to such cases. In this case, the late-night presentation presented logistical difficulties as there were few staff available for the sustained physical effort of cardiopulmonary resuscitation.

The use of a mechanical compression liberates the limited number of night staff from performing prolonged and physically exhausting chest compressions, and generated a better cardiac output, confirmed on invasive arterial monitoring, than staff were able to achieve performing manual external cardiac massage.

To our knowledge, simultaneous use of a mechanical compression device and CVVH has not been described in the literature before. Neither the Resuscitation Council (UK)⁽¹⁾ nor the American Heart Association⁽²⁾ in recommending the use of extracorporeal blood warming with partial bypass in hypothermic cardiac arrest, mention CVVH. We are aware of only two cases reporting successful resuscitation from hypothermic cardiac arrest with the use of CVVH.^(3,4) This seems to suggest CVVH is either not considered as a tool in this setting or its efficacy is not adequate. Indeed, assuming a theoretical speed of rewarming of 2-3°C/h,⁽¹⁾ the time to achieve a temperature at which re-occurrence of spontaneous circulation (ROSC) can take place or death can be confirmed could be estimated as longer than six hours in our patient. It should be mentioned that the 2-3°C/h gradient comes from patients who maintained their own circulation. Our case, as well as the one presented by Alfonzo,⁽³⁾ revealed a slower than described rewarming gradient (1°C/h), which in both cases resulted in delivery of electric shocks in asystole. In our case, continuation of chest compressions became impossible, because of exhaustion of the team. ROSC occurred earlier in the case presented by Hughes⁽⁴⁾ – at a temperature of 23°C with 2°C/h rewarming gradient – when an additional heat

source was used on the return line of the CVVH machine. Faster warming of the heart with a delay in distribution of heat within the central compartment could be a possible explanation, but without direct measurements this is pure assumption. In both cases, circulation re-occurred after defibrillation in non-shockable rhythms.

Treatment modalities varied in each case. Hughes used transfusion of blood products to treat anaemia and clotting abnormalities. We used infusions of inotropes, insulin, potassium and magnesium. Exchanging volumes differed – we used a so-called ‘septic cycle’, with up to 60ml/kg/hour of fluid exchange, whereas Alfonzo’s case reports flow of up to 200ml/min. In both cases, haemofiltration was discontinued following ROSC; however, we made the decision to continue with haemofiltration for several hours using high-flow fluid exchange. There is some data suggesting that use of high-flow haemofiltration can contribute to prevention of acute renal failure caused by myoglobin.^(5,6) Our patient did not require either renal replacement therapy or intermittent positive airway pressure ventilation after extubation.

Guidelines are useful for medical practice but the authors accept that occasionally a clinical situation occurs in which guidelines can be ignored. This is particularly important where it is felt that there is nothing to be lost by deviation. In this case, we deviated from the guidelines in five respects:

- we used adrenaline as we felt fit and ignored advice about its lack of efficacy in hypothermia
- we used DC defibrillation in asystole
- we used 100% oxygen only until we established an arterial line, thereafter we were guided by laboratory measurements of arterial PaO₂
- we used amiodarone in hypothermia
- we used insulin and other infusions – potassium, magnesium

Use of warmed and humidified oxygen/inspiratory gas mixture is a basic and effective method of warming a hypothermic patient, but to our knowledge neither ambulances nor emergency departments in the UK are equipped with such devices. Our patient received oxygen, via a facemask, from a bottled supply, prior to arrival in the emergency department. Oxygen administered in this way is dry and cold and could have contributed to the cooling of the patient. Need for gas warmers and humidifiers mandates early transfer to an intensive care area.

The use of vasopressors and amiodarone in patients with hypothermic cardiac arrest is not standard practice according to the Resuscitation Council (UK) or the American Heart Association.^(1,2) A recently published literature review, however, suggests re-evaluation of these guidelines in cases of ventricular fibrillation.⁽⁷⁾ In our case, the use of adrenaline, initially in increments followed by continuous infusion guided by values of invasive blood pressure, provided physiologically normal mean arterial pressures (MAP). Considering coronary perfusion pressures from experimental data, and its obvious dependency on MAP, the use of adrenaline in hypothermic cardiac arrest may be beneficial. Most probably, this is one of those rare cases (if not the only one in the literature) in which direct arterial pressures were measured during resuscitation and the use of catecholamines guided by its measurement.

Glycaemic control during prolonged resuscitation seems to be an underestimated measure in the presence of other serious life-threatening disturbances. Hyperglycaemia in patients

undergoing on-pump coronary artery by-pass graft is associated with increased risk of neurocognitive dysfunction.⁽⁸⁾ After admission to the ICU, an insulin infusion was commenced and the highest glucose level was 24.8mmol/l just after spontaneous circulation was restored. This dropped to 15mmol/l over the next two hours. Including the resuscitation and rewarming time and recognising the inotropic role of insulin,^(9,10) in our opinion, insulin should be used to stabilise glycaemia in similar cases, even before spontaneous circulation is restored.

CONCLUSIONS

The use of a mechanical cardiac compression device is effective in maintaining cardiac output for a prolonged period in cardiac arrest in severely hypothermic patients.

CVVH is a potentially useful means of extracorporeal core rewarming and can be used in conjunction with a mechanical cardiac compression device.

Further studies on the use of vasopressors and insulin in hypothermia and hypothermic cardiac arrest are needed to define their definitive role in hypothermia.

Use of defibrillation in apparent asystole in hypothermic cardiac arrest with a core temperature below 30°C requires further experimental evaluation.

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Commentary . . .

Lucy Belson is the senior resuscitation officer responsible for training staff across all the hospital sites.

This is a challenging case involving prolonged chest compressions for a hypothermic patient. The use of the mechanical chest compression (MCC) device (AutoPulse®Zoll), along with other interventions, played a key role in this patient's survival from a four and half hour cardiac arrest.

AutoPulse®Zoll device

The evidence that manual chest compressions are poor already exists.⁽¹⁾ Kern suggests that only 30-40% of normal blood flow to the brain is achieved through manual chest compressions, whereas mechanical chest compressions give improved haemodynamics including coronary perfusion.^(2,3)

I certainly hope that this case stimulates further discussion. For example, should MCC be considered an essential piece of equipment in all Emergency Departments (EDs)? I strongly support this, and it is the case that MCC is currently used in a number of London hospitals. Two minutes of manual chest compressions is physically tiring; EDs are usually extremely busy and can at times be short staffed; there is evidence to show that manual chest compressions are not very effective – it's a strong case. We could go further and ask if all emergency ambulances should have an MCC device. There are obvious drawbacks to performing chest compression in the back of a moving ambulance: it's a danger to the paramedics and is unlikely to be sufficiently effective.

The resuscitation department has previously run the 'Zoll CPR Challenge'. This is when staff are asked to perform two minutes of chest compressions on a manikin which is connected up to a laptop and defibrillator. This then gives frighteningly accurate feedback on the quality of chest compressions, including both rate and depth. It is often a humbling experience: the machine does not recognise the status or experience of the participant. In 2008 a porter from Westmorland General Hospital was watching the challenge

being performed in the main entrance. Towards the end of the day he came over and said he'd give it a go as no-one else was around. He was outstanding and scored more than any other participant (and, by the way, he was 68 years old at the time).

Clinical staff must keep their basic life support skills up to date on an annual basis and the resuscitation department has a number of sessions available. To book onto our courses please go to the KELD pages of the trust intranet and click on resuscitation, where there are details on how to book a session.

MCC device deployed in the field

Although this has been a rare event, shouldn't we prepare ourselves for it becoming much more common? Countries such as Canada, Switzerland and the Scandinavian countries deal with this on a regular basis and it would be interesting to explore this further – particularly if our very cold British winters continue!

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Continued from page 183

the tracheal tube cuff will rise substantially as the patient is rewarmed. A common error in intubated patients is overenthusiastic ventilation. Hypocapnia can induce ventricular irritability.⁽⁸⁾

Palpation of pulses is extremely difficult in vasoconstricted patients with a bradycardia and hypotension. The rescuer should auscultate and palpate for at least one minute to find pulses. An ECG monitor can be useful. Importantly, iatrogenic VF can easily occur if chest compressions are used when not indicated. The management of cardiac arrest has been outlined above.

PRACTICALITIES OF MEASURING CORE TEMPERATURE

The ideal is to measure oesophageal temperature, as this not only gives the core but most importantly, the cardiac temperature. The probe tip must be behind the heart so as not to give a falsely high reading from warmed tracheal air. In prehospital areas, an oral device is available that measures down to 26°C. Some rescue teams use thermometers that were designed for non-clinical purposes, with a long-reach probe which is inserted into the oesophagus. Rectal temperature can be misleading. It can read falsely low if the legs are frozen or during rewarming if the probe tip is inserted into a mass of cold faeces when it will lag well behind oesophageal temperature,^(8,10) or falsely high if peritoneal lavage is used for rewarming.

INITIAL MANAGEMENT IN HOSPITAL^(7,8)

Casualties who are brought directly to the Emergency Department should be managed along similar lines to those described above. However, there are many more options in hospital. Invasive monitoring can be used, though it is wise to avoid placing catheters in the heart as these can precipitate an arrhythmia. Traditional monitoring is usually unhelpful, eg pulse oximetry, non-invasive blood pressure. Doppler ultrasonography can be used to search for evidence of cardiac activity. A full general blood screen should be done bearing in mind the caveats outlined above about interpreting the blood results of hypothermic patients when the assays are run at normal body temperature. In addition, it is appropriate to check for specific items, eg thyroid function, cortisol, toxicology, etc in cases of secondary hypothermia.

Fluid resuscitation will be required for the reasons outlined above. Most patients will be significantly dehydrated. Fluid shifts within the body will reverse during rewarming. However, additional crystalloid will be required for the fluid lost by diuresis. Lactate solutions such as Hartmann's solution should not be used initially as the cold liver cannot metabolise the lactate.⁽⁸⁾ Intravenous fluid replacement should be continued throughout the rewarming phase as the peripheral circulation opens up.

STRATEGIES FOR REWARMING^(1,7)

It must be remembered that cardiovascular stability will only be achieved by stopping the fall in core temperature and establishing rewarming. There are three different principles for rewarming: passive external, active external and active internal.

Indications for active rewarming

- Cardiovascular instability
- Moderate hypothermia (core 32°C) or less
- Inadequate rate of rewarming or failure to rewarm
- Endocrine insufficiency causing decreased heat production (thyroid, adrenal, pituitary, etc)
- Obligatory vasodilation caused by drugs or poisons
- Presence of other predisposing factors, eg age, spinal cord injury, multiple sclerosis, etc

Passive rewarming
Passive rewarming means that the patient raises their own body temperature, and intervention is limited to applying full body insulation and a warm environment. It is only appropriate in conscious patients who have mild hypothermia but are still able to shiver or move around to generate heat. In addition, the patient should be given hot sweet drinks to provide an energy substrate to allow shivering to continue.

Active rewarming
Active rewarming is the direct application of exogenous heat to the patient. The indications are:⁽⁸⁾

Active external rewarming
This delivers heat directly to the skin. Examples include forced-air blanket such as the Bair Hugger®, immersion, plumbed garments such as the Artic Sun®,⁽³⁹⁾ hot water bottles, heating pads and radiant heat sources. Whole body immersion in hot water should not be done, but immersing the extremities in hot water at 42° or 45°C can give rewarming rates between 6.1° and 9.9°C per hour in mild hypothermia by opening cutaneous arteriovenous anastomoses.^(7,8) Radiant heat and heating pads are also helpful. Active external warming with blankets has been shown to increase temperatures by 1-2°C/hour;⁽²³⁾ Forced air rewarming is very effective in severe hypothermia without cardiac arrest, rewarming at about 1.7°C/hour;^(7,40) Focussing the rewarming around the torso produces a fast core rewarming rate.⁽⁴¹⁾

Active internal (core) rewarming
Many options are available for active internal warming. Examples are:

- Warmed humidified air raises core temperature by 1-1.5°C/hour;⁽²³⁾ Breathing accounts for about 21% of total heat loss,⁽⁴²⁾ so airway rewarming minimises respiratory heat loss if the temperature and moisture content is high enough. Passive heat-moisture exchangers are less effective than active devices.^(8,43)
- Heated intravenous infusions.⁽⁸⁾
- Lavage of a body cavity or large organ with warmed fluid – peritoneal, gastric, colonic, bladder, mediastinal, and pleural (thoracic)^(44,45) – have all been described.⁽⁸⁾ These will raise the core temperature by about 1-2.5°C/hour.^(18,23)

Extracorporeal rewarming

Three routinely available methods have been used for rewarming the blood outside the body:

- Continuous venovenous haemofiltration, which can raise the core temperature by about 1-3°C/hour.^(7,8,46,47,48,49)
- Haemodialysis, which can raise the core temperature by about 2-3°C/hour.^(8,47,50,51)
- Rewarming via a multi lumen femoral vein catheter that is usually used to produce hypothermia in patients with cardiac arrest. Warm water at 37.5°C can be circulated through the balloons that run along the catheter. However, it only rewarms at about 0.75°C per hour.^(52,53)

The drawback with these techniques is that they do not support the circulation. If they are to be used in a patient without a perfusing rhythm, then arrangements must be made to continue CPR, perhaps for many hours, until the heart has rewarmed sufficiently to be able to achieve and sustain a circulation. In the presence of severe hypothermia with cardiorespiratory arrest, the priority is to restore a perfusing rhythm as quickly as possible by rewarming rapidly – well in excess of 2°C per hour – in order to minimise the length of time necessary for CPR. This can only be achieved using cardiopulmonary bypass (CPB) or extracorporeal membrane oxygenation (ECMO). These methods produce the most rapid rewarming (8-12°C/hour)⁽¹⁾ – whilst obviating the need for continuing CPR during the rewarming period. Rewarming speeds greater than 10-12°C are technically possible, but evidence suggests that this can be dangerous because the temperature gradient between the circulating blood and the cold tissue is too great.⁽⁸⁾

The medical literature contains many case reports and retrospective reviews dating back to the 1980s that confirm the effectiveness of CPB and ECMO for rewarming casualties with severe hypothermia who have had a cardiac arrest, and this is the method recommended in the latest Resuscitation Council guidelines.⁽¹⁾ CPB is generally done via the femoral vessels in this situation, and is extremely effective.^(5,54,55,56,57,58) A portable CPB device that is normally employed in the management of cardiogenic shock has been used successfully to rewarm hypothermic patients. It has the advantages over traditional CPB of low cost, is less invasive, and has a short setup time.⁽⁵⁹⁾ ECMO also works extremely well, though it is less generally available in the UK. It has a number of potential advantages over CPB including less systemic heparinisation, it can be used for prolonged respiratory support, and it may cause less of an inflammatory response than CPB. Survival rates may be higher with ECMO.^(7,60)

A recent comprehensive review has provided a practical approach to the prehospital management of severe accidental hypothermia and recommends transfer of appropriate cases directly to a facility able to deliver CPB or ECMO.⁽⁷⁾ If a severely hypothermic patient with cardiac arrest has initially been brought to a hospital without a cardiac unit, then there will obviously be a practical problem with transferring them to another facility for rewarming. If transfer is possible, eg by helicopter, then it should be considered. Chest compressions will have to be maintained using a mechanical device such as the Zoll AutoPulse or the Physio-Control LUCAS™. The Zoll device has received a certificate of airworthiness and has been used during flight by a number of Air Ambulances in the UK.⁽⁶¹⁾

Core temperature afterdrop

Afterdrop is a further fall in temperature that occurs after removal from the cold. The main mechanism appears to be as equilibration occurs between the periphery and the core, cold blood moves from the limbs and causes a fall in core temperature. The incidence and magnitude vary widely. It is more likely to occur when a large temperature gradient exists between the periphery and the core, ie in dehydrated, chronically cold patients. It can also occur if core rewarming is too rapid leaving the peripheries still very cold. For these reasons, it is unwise to try to stimulate the peripheral circulation or raise the legs in severely hypothermic individuals. In cold-water immersion, post-rescue collapse may also result from abrupt hypotension after loss of the hydrostatic squeezing effect of the water.^(7,8)

THE POST-WARMING PHASE FOR VICTIMS OF SEVERE HYPOTHERMIA

Survivors of the initial phase face several problems. These are related to the hypothermia and the rewarming technique. Examples are cardiac dysfunction, acute renal failure, ARDS, or similar. Some have even needed ECMO for 3-4 days because of oxygenation problems. Neurological problems are common but usually resolve in time. Although some patients recover within days, most have needed a protracted period (weeks) in intensive care. However, once they have got as far as ICU, the longterm prognosis is generally very good. Examples of the problems that can occur are:

- A skier became partially trapped under ice over a stream and was clinically dead when pulled out. At the scene, basic CPR was carried out and was continued in a helicopter for 90 minutes until arrival at hospital. Femoral access CPB was established 130 minutes after arrest. During rewarming, VF started which spontaneously converted to a perfusing rhythm at an oesophageal temperature 31.5°C (pharyngeal temperature 25°C, rectal temperature 14.2°C). The patient subsequently required ECMO for 5 days, was in ICU for 28 days, and had a post-illness polyneuropathy. On day 60 she was moved to a rehabilitation unit and returned to work after five months.⁽¹⁰⁾
- A 26-year-old man was found in a forest 15 hours after a suicide attempt. His core temperature was 19°C (oesophageal) with circulatory arrest (VF). Defibrillation failed and he was transferred to hospital whilst receiving continuous CPR. Femoral access CPB was instituted. Rewarming took 158 minutes and he was weaned from CPB without inotropes. He spent eight days on the general ICU. The only complication was rhabdomyolysis. The patient was discharged from hospital four weeks after the event.⁽⁶²⁾

CONCLUSION

The outcome can be extremely good, particularly in young people, with survival rates reaching 60-70%.^(10,57,63,64,65) If hypothermia is associated with trauma and acidosis (the 'Lethal Triad'), the mortality rate is very high.⁽⁶⁶⁾ Severe accidental hypothermia is a challenging clinical problem, yet many patients can be saved by awareness of the issues

involved. Given the complexity and rarity of the condition, all hospitals with A&E departments should have clear guidelines for managing these patients effectively. The development of such guidelines should involve the relevant emergency services.

Finally, an International Registry has been set up to collect information on patients with severe accidental hypothermia (see <http://www.hypothermia-registry.org>). The aim is to increase knowledge about the subject, and enable comparison of rewarming techniques and outcomes. Any doctor can add patients to the database and extract information.

Photographs courtesy of Nick Owen (LAMRT)

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