

# Deep accidental hypothermia during the Queensland summer

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## Clinical record

A 52-year-old woman with a history of major depression, generalised anxiety disorder and multiple previous attempts at self-harm was found unresponsive on the floor of her locked bedroom after a presumed polypharmacy overdose. She had last spoken to a friend about 6 hours previously, although the exact period of unconsciousness was unknown. Initial assessment recorded by the ambulance service on their arrival 5 minutes later were: Glasgow Coma Score (GCS), 3; no palpable pulse; a partially obstructed airway; and evidence of agonal respiration. The room was noted to be very cool, with a split-system air conditioning unit in operation.

Following basic airway manoeuvres and bag mask ventilation, electrocardiographic monitoring revealed ventricular fibrillation, and advanced cardiac life support was begun. This comprised closed chest compressions, electrical defibrillation with a 150J biphasic waveform (HeartStart 4000, Laerdal, Oakleigh, VIC), administration of intravenous (IV) adrenaline (1 mg) every 3–5 minutes, atropine (1.2 mg), lignocaine (100 mg) and sodium bicarbonate (50 mmol). Endotracheal intubation was successful, and the patient was hand-ventilated at a compression-to-ventilation ratio of 5:1. After about 50 minutes of resuscitation at the scene, during which the patient received 18 electrical defibrillation shocks, 12 mg of IV adrenaline and 1.5L IV crystalloid, return of spontaneous circulation was achieved, and she was transferred to hospital.

On arrival, she had marked bradycardia (heart rate, 30–40 beats per minute) and a systolic blood pressure of 50 mmHg. Transcutaneous pacing was begun at 100 beats per minute. Further bolus doses of adrenaline were administered, along with further IV crystalloid (1L). A central venous line and intra-arterial line were inserted into the right femoral vein and artery, respectively, and she was mechanically ventilated with 100% oxygen. Adrenaline and noradrenaline infusions were commenced at 0.5 µg/kg per min. The patient's temperature at this point was recorded as 26.8°C via a digital tympanic thermometer. The GCS remained 3, her pupils were fixed and dilated, and there was no evidence of a gag or cough reflex. The first arterial blood gas analysis showed metabolic acidosis, hyperlactaemia, and hyperglycaemia: pH, 7.18 (reference range [RR], 7.35–7.45); P<sub>CO<sub>2</sub></sub>, 46 mmHg (RR, 35–45 mmHg); P<sub>O<sub>2</sub></sub>, 536 mmHg (RR, 75–100 mmHg); base excess, -11 mmol/L (RR, -3 to 3 mmol/L); serum concentrations of Na<sup>+</sup>, 131 mmol/L (RR, 135–145 mmol/L); K<sup>+</sup>, 2.9 mmol/L (RR, 3.2–4.5 mmol/L); glucose, 24.4 mmol/L (RR, 3.0–7.8 mmol/L); and lactate, 10.0 mmol/L (RR, 0.7–2.5 mmol/L).

## ABSTRACT

A 52-year-old woman presented with severe accidental hypothermia associated with out-of-hospital cardiac arrest after a polypharmacy overdose. Deep hypothermia developed while she lay unconscious, with a split-system air-conditioning unit rapidly cooling the confined area of her bedroom. Despite the need for lengthy resuscitative efforts at the scene and in hospital, she went on to a full neurological recovery. The neuroprotective role of accidental hypothermia is reviewed, as are the guidelines for resuscitation in this setting. We conclude that hypothermia must be considered even in unlikely circumstances, such as the Queensland summer, when ambient temperatures are high.

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Her blood pressure remained refractory (about 55–65 mmHg systolic) despite high-dose inotropic and vasopressor support. An insulin infusion was begun to achieve optimal glucose control, and IV potassium was administered. A simultaneous transthoracic echocardiogram performed in the emergency department showed adequate left ventricular function, with no evidence of left ventricular outflow tract obstruction, and no pericardial effusion. Hydrocortisone (100 mg IV) was administered along with a further 3L of intravenous crystalloid. An intra-aortic balloon pump (IABP) and temporary pacing wire were inserted via a femoral approach, and the patient was transferred to the intensive care unit.

An initial serum paracetamol level was 265 mg/L, which was treated with *N*-acetyl-cysteine. Urinary toxicology screen also showed evidence of benzodiazepines. A bolus dose of flumazenil failed to increase her level of consciousness. Troponin I was not detectable, and an electrocardiogram (ECG) showed slow atrial fibrillation with a prolonged QT interval. Active external re-warming was instituted via a forced air blanket. Over 3 hours, her core temperature rose to more than 30°C and, over the next 6 hours, to 36°C, although the goal had been to maintain a core temperature of 33°C over the following 24 hours. The adrenaline infusion was weaned, and the noradrenaline infusion was reduced to 0.1 µg/kg per min, achieving a mean arterial pressure greater than 70 mmHg (Figure 1).

About 20 hours after arrival in the emergency department, and despite remaining heavily comatose throughout this period, with fixed and dilated pupils, the patient began to rouse and subsequently woke rapidly, to a GCS of 10 (E4, M6, VT). She was extubated 12 hours later, and the balloon pump

and temporary pacing wire were removed (Figure 1). The noradrenaline infusion was weaned over the next 24 hours, and her acid–base balance gradually improved over a similar period. She had no further arrhythmias and remained haemodynamically stable.

Subsequent neurological assessment failed to reveal any focal abnormalities. The patient recalled consuming large quantities of fluvoxamine, paracetamol, zolpidem and temazepam before her collapse, but was unable to recall any subsequent events. She was reviewed by psychiatric services and discharged to the ward after a stay of 56 hours.

## Discussion

Severe hypothermia (defined by the American Heart Association as a core temperature less than 30°C<sup>1</sup>) is associated with a number of potentially fatal arrhythmias, as well as a variety of clinical features involving central nervous system, respiratory, renal, neuromuscular and haematological dysfunction. Patients with a severely depressed core temperature are likely to be deeply unconscious, with non-reactive pupils, apnoea, oliguria and asystole or a ventricular arrhythmia.<sup>2</sup> Conditions that predispose to the development of severe hypothermia

include old age, malnutrition, self-neglect, dementia, endocrine dysfunction, alcohol consumption, substance misuse, burns, neuroleptic drugs, and environmental exposure.<sup>3</sup>

Our patient appeared to have become deeply unconscious after ingesting a large quantity of sedative agents (zolpidem and temazepam), leading to impairment of her thermoregulatory mechanisms. In addition, she was confined to a small room with an air-conditioner in operation, which resulted in her becoming markedly hypothermic. The ambient outside air temperature was 29.1°C.<sup>4</sup>

There are some important specific considerations regarding resuscitation in the setting of hypothermia. Management is primarily dictated by the presence or absence of a cardiac output and the degree of hypothermia (mild, >34°C; moderate, 30–34°C; or severe, <30°C).<sup>1</sup> In the case of severe hypothermia and cardiac arrest, the focus is towards rapid rewarming through active internal means while cardiopulmon-

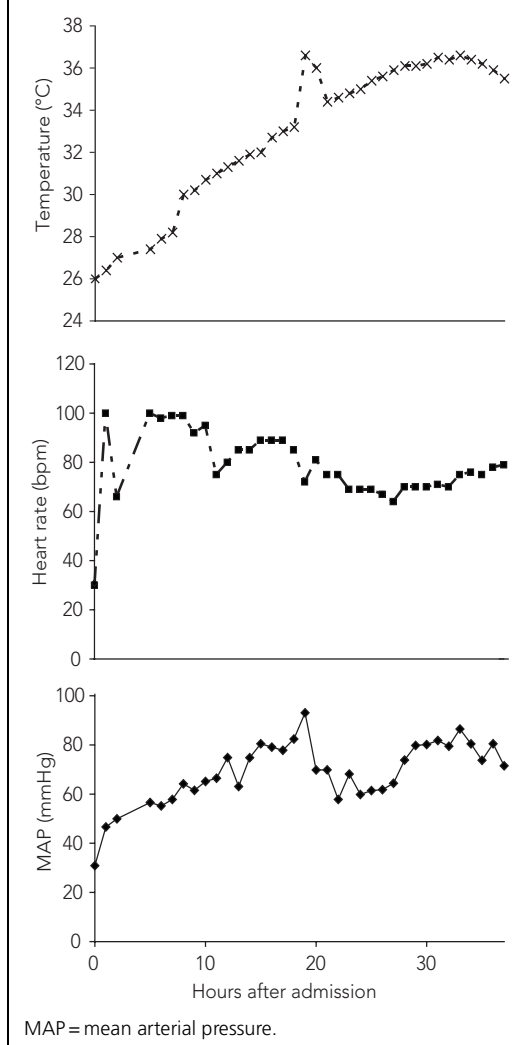
ary resuscitation is continued. Use of extracorporeal circulation is now considered the most rapid and efficacious means of achieving this.<sup>5,6</sup> Although cases of successful defibrillation have been reported at very low temperatures,<sup>7</sup> the myocardium is generally considered refractory, and only a single attempt at defibrillation is suggested until the core temperature is raised to greater than 30°C.<sup>1</sup> Administration of vasoactive drugs is also cautioned against at very low temperatures, as drug metabolism is likely to be impaired, and concerns have been raised about accumulation to toxic levels on rewarming.<sup>1</sup>

In this circumstance, return of spontaneous circulation was achieved in the field before the patient's arrival at our hospital, through repeated attempts at defibrillation. The attending ambulance officers were unlikely to have considered severe hypothermia as a contributory factor, as cardiac arrest associated with accidental hypothermia is extremely uncommon in Queensland. This is further underscored by the lack of a documented temperature at the scene. Subsequent management was aimed at supporting the patient's cardiovascular system while she was actively externally rewarmed. Cardiopulmonary bypass was not immediately available for active invasive rewarming.

The use of an IABP in this setting represents, to our knowledge, one of the first reported cases, and is an intervention more widely available in Australasian centres than extracorporeal membrane oxygenation techniques. While echocardiography demonstrated adequate ventricular function, the beneficial effects of an IABP relate more to augmenting cerebral perfusion and oxygenation, rather than cardiac function. Such a role for this device has already been reported in cases of cerebral vasospasm after subarachnoid aneurysm refractory to conventional therapy,<sup>8</sup> and in augmenting cerebral oxygenation during hypothermic cardiopulmonary bypass.<sup>9</sup>

Numerous cases have been reported involving profound hypothermia associated with a successful neurological outcome despite lengthy periods of circulatory arrest.<sup>10–12</sup> Furthermore, in recently published clinical trials, induced mild hypothermia has been shown to improve the neurological outcome of selected comatose survivors following out-of-hospital cardiac arrest.<sup>13,14</sup>

**Figure 1. Physiological variables in a woman with out-of-hospital cardiac arrest and deep hypothermia**



## CASE REPORTS

However, the neuroprotective effects of hypothermia remain poorly understood. Conventional theory has centred around a temperature-dependent reduction in the cerebral metabolic rate for oxygen (CMRO<sub>2</sub>), although a decrease in the release of excitatory amino acids (glutamate),<sup>15</sup> and improved coupling of cerebral blood flow and metabolic demand — which allows improved distribution of oxygen to the brain as a whole — probably have a central role.<sup>16</sup> Benzodiazepines also reduce the CMRO<sub>2</sub><sup>17</sup> and, in this respect, are considered to have a neuroprotective effect.<sup>18</sup> In our patient, the concomitant ingestion of such an agent may have had a significant role in the favourable neurological outcome.

Prognostication can be very difficult in these circumstances, and death cannot reliably be certified until the core temperature has reached 35°C. Despite advances in rewarming techniques, severe accidental hypothermia remains associated with a high mortality, between 30% and 80% depending on the mechanism involved and patient population.<sup>6</sup> Some authors have proposed prognostic markers of poor outcome, such as plasma potassium level >9 mmol/L, central venous pH ≤ 6.5 or activated clotting time >400s.<sup>19</sup> However, these factors have not been validated in other series<sup>20</sup> and continue to be of limited utility. Asphyxial hypothermic arrest (as seen with drowning and avalanche victims) appears to be associated with a much poorer outcome,<sup>6</sup> underpinning the concept that rapid cooling must precede hypoxia if any degree of cerebral protection is to be obtained.

In summary, accidental hypothermia should be considered in a variety of clinical circumstances, regardless of any usually benign weather conditions. The association with substance misuse or attempted suicide should prompt the clinician to ensure normothermia before considering stopping resuscitation. Efforts should focus on rapid rewarming, with deferment of defibrillation and further vasoactive drug therapy until an appropriate core body temperature has been achieved. Extracorporeal circuits, if available, appear to be the most efficacious method of rewarming.

Although the outcome is often poor, prognostication in this setting can be difficult, and the clinician must be mindful that favourable neurological outcomes may be possible, particularly in cases of non-asphyxial hypothermic cardiac arrest.

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