MANAGEMENT OF HYPOTHERMIA

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Summary

Hypothermia is defined as a body core temperature (T_{CO}) below 35°C. It is classified as mild (T_{CO} 35-32°C), moderate (T_{CO} 32-30°C) or severe (T_{CO} < 30°C). Hypothermia commonly results from an injury in a cold environment, immersion in cold water or a prolonged exposure to low temperatures. Hypothermia causes characteristic changes (the Osborn J wave) in the electrocardiograph (EKG) and severe hypothermia can cause life-threatening dysrhythmias, or asystole. The typical sequence is a progression from sinus bradycardia through atrial fibrillation (AF) to ventricular fibrillation (VF) and ultimately asystole. Active warming must, therefore, commence in the field, with the caveat that patient handling is safe and controlled. The most practical method of active warming in the field is to place heat packs on the skin near to major blood vessels (neck, thoracic inlet, axillae, abdomen and groin). Warmed, humidified air/oxygen mixes have little thermal advantage. Arterio-venous anastomosis warming can be useful in a base camp setting or aboard ship; the arms, forearms, lower legs, and feet are immersed in water at 42 or 45°C, giving rewarming rates between 6.1 and 9.9°C per hour respectively. Whole-body immersion in hot water is contraindicated. This form of rapid surface warming will cause massive vasodilatation and hypotension, and is likely to provoke dysrhythmias and cardiovascular collapse. The in-hospital management of hypothermia follows the primary survey – resuscitation – secondary survey approach. Cardiovascular stability will only be achieved through stopping the fall in core temperature and establishing rewarming. Correction of metabolic and electrolyte disturbances and intravenous fluid replacement runs concurrently. Esophageal or urinary bladder electronic temperature probes are more accurate than rectal probes. Hospitals without CPB or ECMO should encourage transfer of patients in cardiac arrest directly from the scene to units that do have these facilities.

Key words: management, hypothermia, arterio-venous anastomosis

Rezumat. Managementul hipotermiei

Hipotermia este definită ca temperatura corporală (T_{CO}) sub 35°C. Se clasifică în hipotermie uşoară (T_{CO} 35-32°C), moderată (T_{CO} 32-30°C) şi severă (T_{CO} < 30°C). Hipotermia este o leziune secundară expunerii într-un mediu rece, imersie în apă rece sau expunere îndelungată la temperaturi joase. Hipotermia cauzează schimbări caracteristice (unda J. Osborn) pe electrocardiogramă (ECG) şi hipotermia severă cauzează disritmii ameninţătoare de viaţă sau asistolie. Scenariul tipic este progresia de la bradicardie sinusală şi fibrilaţie atrială spre fibrilaţie ventriculară şi în ultimă instanţă asistolie. Încălzirea activă trebuie iniţiată la locul accidentului şi în timpul transportului în condiţii de siguranţă şi securitate. Cea mai utilizată metodă de încălzire activă este plasarea pungilor în locurile vaselor sanguine mari (gât, axile, torace, abdomen, regiunea inghinală şi poplitee). Administrarea de aer/oxigen umidificat şi încălzit prezintă avantaje termice neînsemnate. În anumite circumstanţe (lagăre de câmp, la bordul unui vas) se poate utiliza şi încălzirea anastomotică arterio-venoasă prin scufundarea în apă caldă cu temperatura 42-45°C a mâinilor şi picioarelor care asigură o încălzire cu 6,1-9,9°C în oră. Scufundarea în apă caldă a întregului corp este contraindicată deoarece va provoca o vasodilatare masivă şi hipotensiune care vor genera disritmii cardiace şi colaps cardiovascular. În condiţii de spital managementul hipotermiei include examenul primar – resuscitarea şi examenul secundar. Stabilitatea cardiovasculară va fi asigurată prin oprirea pierderilor de temperatură corporală şi reîncălzire. Concomitent vor fi efectuate corecţiile dereglărilor metabolice electrolitice şi volemice prin administrare de fluide. Evaluarea în dinamică a temperaturii în esofag este informativă ca temperatura rectală. Pacienţii în stop cardiac trebuie transferaţi în spitale care posedă posibilităţi de by-pass cardiopulmonar (CPB sau oxigenare membranară extracorporeală).

Cuvinte-cheie: management, hipotermia, anastomoze arterio-venoase

Резюме. Менеджмент гипотермии

Гипотермия - переохлаждение определяется как снижение температуры тела ниже 35°C. Гипотермия классифицируется как легкая (35-32°C), умеренная (32-30° C) и тяжелая (<30°C). Гипотермия является вторичным результатом длительного нахождения в холодной окружающей среде, погружения в холодную воду или длительного воздействия низких температур. Переохлаждение вызывает характерные изменения (Дж. Осборн волны) на электрокардиограмме (ЭКГ), а так же тяжелые, угрожающие жизни аритмии и асистолии. Типичным сценарием является прогрессирование фибрилляции предсердий, синусовая брадикардия и, в конечном счете фибрилляции желудочков и асистолия. Активное обогревание следует начать на месте аварии и во время транспортировки пострадавших. Источники обогревания помещаются в зонах проекции крупных кровеносных сосудов (шея, подмышки, грудь, живот, пах и подколенные области). Показана ингаляция увлажненной воздушно-кислородной немного подогретой смеси. При определенных обстоятельствах (полевые лагеря, на борту судна) могут быть

использованы для раскрытия артерио-венозных анастомозов и обогревание путем помещения конечностей пострадавшего в емкость с теплой водой с температурой 42-42°C, что обеспечивает обогрев от 6,1 до 9,9°C в час. Погружение в горячую воду всего тела противопоказан, так как это вызывает резкую вазодилатацию и гипотонию, которая генерирует сердечную аритмию и сердечно-сосудистую недостаточность. Стабилизация функции сердечно-сосудистой системы обеспечивается остановкой потери температуры тела и обогревом. В то же время должна быть произведена коррекция объемов жидкости и устранение нарушений электролитного обмена. Оценка в динамике температуры пищевода является более информативной, чем ректальной температуры. Пациенты при остановке сердца должны быть госпитализированы в стационары, обладающих возможностями подключения систем искусственного кровообращения (экстракорпоральной мембранной оксигенации).

Ключевые слова: менеджмент, гипотермия, артерио-венозные анастомозы

Introduction

Doctors working in intensive care, emergency medicine, pre-hospital care, cardiac surgery and ECMO (extracorporeal membrane oxygenation) programs may be called upon to assist in the management of victims of severe environmental hypothermia.

The International Commission for Alpine Rescue (http://www.ikar-cisa.org), International Society for Mountain Medicine (http:www.ismmed.org), and the Union Internationale des Associations d'Alpinisme medical committee (International Mountaineering and Climbing Federation; http://www.uiaa.ch) have been instrumental in gathering data and publishing guidance for the pre-hospital triage and management of victims of deep hypothermia in the mountains. The principles guiding the resuscitation of victims of accidental hypothermia in the maritime or mountain environment may be applied to everyday emergency practice, even in an urban setting (7, 36).

Definitions

Hypothermia is defined as a body core temperature (T_{CO}) below 35°C. It is classified as mild (T_{CO} 35-32°C), moderate (T_{CO} 32-30°C) or severe (T_{CO} < 30°C).

Pathophysiology

Hypothermia commonly results from an injury in a cold environment, immersion in cold water or a prolonged exposure to low temperatures. Muscular activity and the catabolic processes of the body produce heat; heat is lost by radiation, convection, conduction, and evaporation (vaporization), particularly of water vapor from the skin and lungs. Thermoregulation is a balance between heat production and heat loss, allowing enzyme systems to operate optimally within a narrow temperature range. In mild hypothermia, thermoregulatory mechanisms operate fully in an attempt to combat the situation. If the condition is unchecked however, the thermoregulatory system diminishes until it fails, leading to death from cardiorespiratory failure.

Hypothermia causes characteristic changes (the Osborn J wave) in the electrocardiograph (EKG) and severe hypothermia can cause life-threatening dysrhythmias, or asystole. The typical sequence is a progression from sinus bradycardia through atrial fibrillation (AF) to ventricular fibrillation (VF) and ultimately asystole. Rough patient handling or sudden changes in posture may provoke VF at any time in the severely hypothermic patient. Initial tachypnea is replaced by a decrease in respiratory rate and tidal volume, and bronchorrhea predisposes to aspiration pneumonia. The oxyhemoglobin dissociation curve undergoes a leftwards shift impairing tissue oxygen delivery. The central nervous system is progressively depressed with a corresponding decrease in conscious level. Mild incoordination progresses through agitation and irritation to lethargy, and eventually coma. In hypothermia, the decreased cerebral oxygen requirements may protect the brain against anoxic or ischemic damage after cardiac arrest [18]. Cold diuresis occurs due to impaired renal concentration and an increased central intravascular volume due to peripheral vasoconstriction. Volume losses may be such that significant fluid resuscitation is required whilst managing the recovering victim. Adrenoceptors become dysfunctional in severe hypothermia, so vasoactive drugs (e.g., epinephrine) are ineffective and may accumulate to toxic thresholds, then exerting their toxic effects upon rewarming and reperfusion [31]. There is a plasma shift to the extravascular space, and the consequent hemoconcentration may lead to disseminated intravascular coagulation (DIC). Reversible platelet dysfunction occurs and the clotting time is prolonged due to derangement of the extrinsic pathway [39]. The immobile hypothermic patient is prone to rhabdomyolysis and acute tubular necrosis may occur through myoglobinuria and renal hypoperfusion. In the initial stages, increased insulin secretion and glycogenolysis mobilizes glucose reserves, but hypoglycemia supervenes as reserves are used up. Acidosis occurs due to respiratory depression and hypercarbia, and lactic acid production through shivering and poor tissue perfusion [46]. Hepatic function is depressed leading to accumulation of drugs that normally undergo

hepatic metabolism or detoxification. Finally, the extremities are vulnerable to frostbite as a result of the peripheral vasoconstriction, hypoperfusion, and hemoconcentration leading to "sludging" of the red cells within the small blood vessels.

Consequences of Hypothermia in Trauma Patients

From the late 1980s, the "lethal triad" of hypothermia, acidosis, and coagulopathy has been identified as a major cause of morbidity and mortality in the critically injured patient. Patients are more likely to die in the intensive care unit (ICU) from persistent acidosis and uncorrected coagulopathy, rather than in the operating room (OR) from failure to definitively repair a bowel injury, or to achieve intermedullary fixation of a comminuted long bone fracture. Even in urban settings when transport times are less than fifteen minutes more than 50% of patients with penetrating injury are hypothermic upon admission to the emergency room [20]. Compared to the normothermic, mortality of hypothermic patients increases by as much as 50% in case matched trauma studies [40]. Also at especially high risk for hypothermia are the very young and very old, patients with burns, the head injured patient (the thermostat mechanisms in the hypothalamus may be deranged) and the patient with a high spinal injury causing disruption to the sympathetic chain. These patients become poikilothermic. Prevention of further heat loss and rewarming (where appropriate) are, therefore, essential components of good trauma care [33].

Etiological Classification of Hypothermia

Acute Hypothermia

Severe cold stress overwhelms thermogenesis and rapid cooling ensues, but before the energy reserves are used and the intravascular fluid changes occur. This kind of hypothermia occurs for instance in the avalanche victim or during cold water immersion. Here, the cold shock response occurs in the first 3 to 4 minutes. This initiates peripheral vasoconstriction, the gasp reflex, hyperventilation and tachycardia, and may lead to submersion and drowning, or cause vagal arrest of the heart. In survivors of the cold shock, hypothermia may take up to 30 minutes to develop [10]. Survival time prediction is based on the interrelationship between the thermoregulatory response, clothing and insulation, sea temperature and sea conditions [43].

Sub-acute Hypothermia

This could affect a climber isolated in the mountains. There is slow but continuous heat loss and the energy reserves become gradually depleted. The rate of onset is related to the patient's physical and mental condition, his/her equipment and the severity of the environmental conditions. Complex fluid shifts occur between the various body compartments, leading to hypovolemia, and necessitating fluid resuscitation during rewarming.

Sub-chronic Hypothermia

The classic example is of the elderly patient, immobilized through a fractured neck of femur sustained in a fall at home. Hypothermia is slow in onset but complicated by depletion of energy reserves, rhabdomyolysis, acute renal failure, metabolic acidosis, and hypovolemia due to fluid compartment shifts. Resuscitation is challenged by cardiovascular instability, and by co-morbidities such as respiratory tract infection, which lead to high mortality. Rewarming should be slow and gentle in these patients.

Staging of Hypothermia in the Field According to Clinical Features

The "Swiss" (Swiss Society of Mountain Medicine) method is the most practicable as it is not based solely on the measurement of core temperature and can be performed by non-medical personnel [7]:

Stage I	Patient alert and shivering	(T_{co} 35-32°C)
Stage II	Patient drowsy and not shivering	(T_{co} 32-28°C)
Stage III	Patient unconscious, but with vital signs present	(T_{co} 28-24°C)
Stage IV	Absent vital signs; apparent death	(T_{co} 24- 13°C)
Stage V	Death due to irreversible hypothermia	(T_{co} < 13°C)

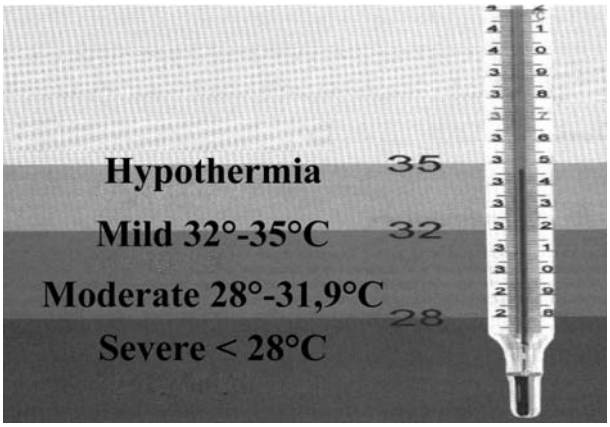

Figure 1. *Degree of Hipothermia*

Severe hypothermia (body temperature < 30°C (86°F)) is associated with marked depression of critical body functions, which may make the victim appear clinically dead during the initial assessment (**table 1**).

Table 1

Signs and symptoms of hypothermia

°F	°C	Signs and symptoms
99,6	37,6	"Normal" rectal temperature
98,6	37	"Normal" oral temperature
96,8	36	Increased metabolic rate in an attempt to balance temperature
95,0	35	Shivering maximum at this temperature
93,2	34	Patients usually responsive with normal blood pressure
91,4	33	
89,6	32	Consciousness clouded; pupils dilated; shivering ceases
87,8	31	Blood pressure difficult to obtain
86,0	30	Progressive loss of consciousness; increased muscular rigidity
85,2	29	Slow pulse and respiration; cardiac arrhythmia develops
82,4	28	Ventricular fibrillation may develop if heart is irritated
80,6	27	Voluntary motion lost along with pupillary light reflex; deep tendon and skin reflexes; appearance of death
78,8	26	Victim seldom conscious
77,0	25	Ventricular fibrillation may appear spontaneously
75,2	24	Pulmonary edema develops
73,4	23	
71,6	22	Maximum risk of fibrillation
69,8	21	
68,0	20	Heart standstill
66,2	19	
64,4	18	Lowest accidental hypothermic patient with recovery
62,6	17	Isoelectric electrocardiogram
48,2	9	Lowest artificially cooled hypothermic patient with recovery

Cold air alone is not nearly as dangerous a freezing factor as a combination of wind and cold. It is astounding but true that the chilly effect of a temperature of-6°C (20°F) combinated with a 45 mile/hour wind is identical to that of a -26°C (-15°F) temperature coupled with a 3 mile/hour wind. The "Wind Chill Chart" shows temperature effects of wind and cold (**table 2**).

Table 2

Wind Chill Chart

Wind Speed		Cooling Power of Wind Expressed as "Equivalent Chill Temperature"																				
Knots	MPH	Temperature (°F)																				
Calm	Calm	40	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50	-55	-60
		Equivalent Chill Temperature																				
3-6	5	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50	-55	-65	-70
7-10	10	30	20	15	10	5	0	-10	-15	-20	-25	-35	-40	-45	-50	-60	-65	-70	-75	-80	-90	-95
11-15	15	25	15	10	0	-5	-10	-20	-25	-30	-40	-45	-50	-60	-65	-70	-80	-85	-90	-100	-105	-110
16-19	20	20	10	5	0	-10	-15	-25	-30	-35	-45	-50	-60	-65	-75	-80	-85	-95	-100	-110	-115	-120
20-23	25	15	10	0	-5	-15	-20	-30	-35	-45	-50	-60	-65	-75	-80	-90	-95	-105	-110	-120	-125	-135
24-28	30	10	5	0	-10	-20	-25	-30	-40	-50	-55	-65	-70	-80	-85	-95	-100	-110	-115	-125	-130	-140
29-32	35	10	5	-5	-10	-20	-30	-35	-40	-50	-60	-65	-75	-80	-90	-100	-105	-115	-120	-130	-135	-145
33-36	40	10	0	-5	-15	-20	-30	-35	-45	-55	-60	-70	-75	-85	-95	-100	-110	-115	-125	-130	-140	-150
Winds above 40 have little additional effect		Little danger					Increasing danger (Flesh may freeze within 1 min.)						Great danger (Flesh my freeze within 30 seconds)									
DANGER OF FREEZING EXPOSED FLESH FOR PROPERLY CLOTHED PERSONS																						

Initial Care for Victims of Accidental Hypothermia

When the victim is extremely cold but has maintained a perfusing rhythm, the rescuer should focus on interventions that prevent further loss of heat and begin to rewarm the victim immediately. Additional interventions include the following:

- Preventing additional evaporative heat loss by removing wet garments and insulating the victim from further environmental exposures. Passive rewarming is generally adequate for patients with mild hypothermia (temperature > 34°C (93,2°F).
- For patients with moderate (30°C to 34°C (86°F to 93,2°F) hypothermia with a perfusing rhythm, external warming techniques are appropriate. Passive rewarming alone will be inadequate for these patients.
- For patients with severe hypothermia (< 30°C [86°F]) with a perfusing rhythm, core rewarming is often used, although some have reported successful rewarming with active external warming techniques. Active external warming techniques include forced air or other efficient surface-warming devices.
- Patients with severe hypothermia and cardiac arrest can be rewarmed most rapidly with cardiopulmonary by-pass. Alternative effective core rewarming techniques include warm-water lavage of the thoracic cavity and extracorporeal blood warming with partial bypass.
- Adjunctive core rewarming techniques include warmed i.v. or intraosseous (i.o.) fluids and warm humidified oxygen. Heat transfer with these measures is not rapid, and should be considered supplementary to active warming techniques.
- Do not delay urgent procedures such as airway management and insertion of vascular catheters. Although these patients may exhibit cardiac irritability, this concern should not delay necessary interventions.

Beyond these critical initial steps, the treatment of severe hypothermia (temperature < 30°C [86°F]) in the field remains controversial. Many providers do not have the time or equipment to assess core body temperature or to institute aggressive rewarming techniques, although these methods should be initiated when available.

Pre-Hospital Care (figure 2)

In the field the core temperature should be measured using an epitympanic low reading thermometer [7].

Severely hypothermic patients have been successfully resuscitated even after several hours of asystolic cardiac arrest. The current record for the lowest core temperature from which a victim has been resuscitated in accidental hypothermia is 13.7°C [12). Clearly the principal clinical challenge is in differentiating a stage IV from a stage V victim. The key being that in stage V the thorax and abdomen are not compressible, the cardiac rhythm is always asystole (stage IV, maybe asystole or VF), the core temperature is lower than 13°C, and the serum potassium is greater than 12 mmol/l. Of course, one cannot resuscitate all hypothermic casualties; hypothermia is a consequence of death whatever its cause and it would be inappropriate to attempt to re-warm these casualties. It is important to remember that an obvious lethal injury is a contraindication to resuscitation.

As a clinical example, in avalanche victims the burial time and presence of an air pocket are important prognostic factors as well. Patients buried for more than 35 minutes with no air pocket do not survive. However, when burial time exceeds 35 minutes, and the patient has an air pocket, managing severe hypothermia may become the key challenge in the extracted victim. Extrication should be careful and gentle as rough handling may provoke VF or asystole. In the absence of vital signs, if the victim is in presumed cardiac arrest, and has a core temperature of > 32°C then resuscitation follows Advanced Life Support (ALS) guidelines. If successful then the patient is transported to a critical care facility; if there is failure to respond after 20 minutes resuscitation then life may be pronounced extinct. In the case of prolonged burial when the victim's core temperature has dropped below 32°C, stage IV hypothermia must be assumed provided that there is an air pocket around the face of the victim and that the airway is clear from obstruction due to ice or vomitus. ALS is commenced, and the patient transported to definitive care where active internal rewarming can be performed. If, however, the airway is obstructed, then resuscitation is contraindicated, and life may be pronounced extinct

Resuscitation Guidelines According to Clinical Staging of Hypothermia

Stage I. Provide shelter and insulate from wind, rain or snow. Give hot sweet drinks and food. Encourage shivering or exercise to generate heat. Evacuation should be considered if there is the suspicion of an occult injury or co-morbidity that may have precipitated the hypothermic condition, such as a toxicological ingestion.

Stage II. This patient is not shivering, and is vulnerable to a dysrhythmia if handled roughly or inappropriately. He/she should be nursed horizontally in the side position to protect the airway (unless of course there is the suspicion of a spinal injury), and

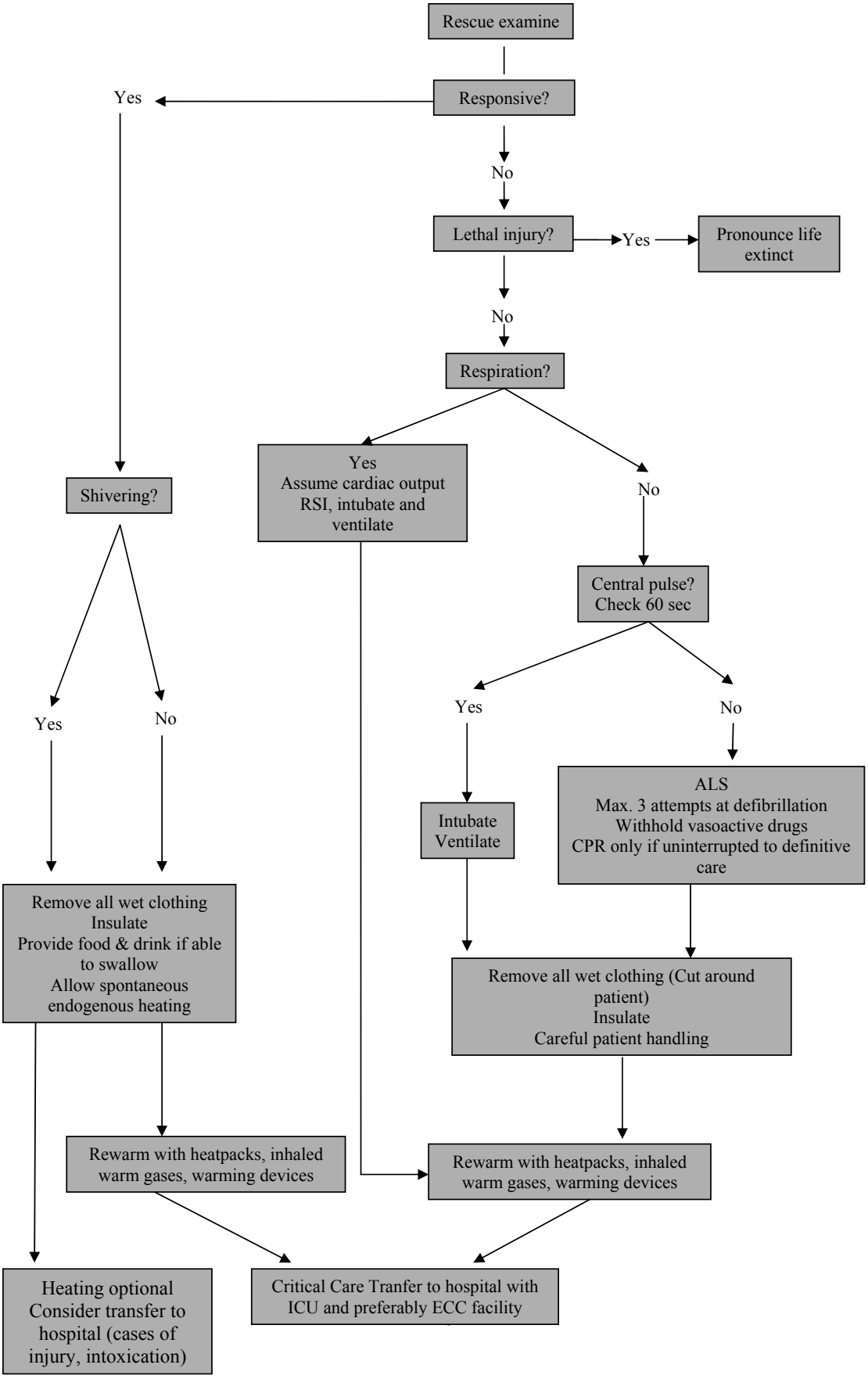

Figure 2. *Algorithm for pre-hospital care of patient with severe accidental hypothermia.*

Note: **ALS – advanced life support; CPR – cardiopulmonary resuscitation; RSI – rapid sequence induction of anesthesia; ICU – intensive care unit; ECC – extracorporeal circulation.**

he/she should be warmed. If the gag-reflex is present and the patient can swallow without risk of aspiration, then give hot sweet drinks and food. Disposition is to a hospital with an intensive care facility (ICU). Avalanche victims who were completely buried and the maritime victim who has been submersed, but survived, are at risk of late respiratory complications such as pulmonary edema, acute respiratory distress syndrome (ARDS), or aspiration pneumonitis [2].

Stage III. This patient will have a reduced level of consciousness, and will be on the cusp of VF or asystole if handled inappropriately. It is prudent to intubate and ventilate the patient, both to protect the airway and to optimize ventilation. Intravenous access can be challenging due to peripheral vasoconstriction, but is essential for rapid sequence induction of anesthesia. There are risks to intubation in that VF may be provoked by laryngoscopy and the time required to perform the procedure may prolong extrication and evacuation. Measures to rewarm and insulate against further loss of heat must be employed. Disposition is to an institution capable of active rewarming – preferably with cardiopulmonary bypass (CPB) or ECMO facilities.

Stage IV. This patient is severely hypothermic and apparently dead. Deep tendon reflexes are absent, and the pupils are fixed and dilated. Cardiopulmonary resuscitation (CPR) must be instituted immediately with the caveat that once commenced it must be continued uninterrupted through to definitive care [9]. The rationale for this is that at very low temperatures it may be difficult to confirm ventilation or cardiac activity and initiation of CPR may trigger VF. To then cease CPR would be a fatal insult to the patient [41]. These patients should be transported to a hospital with CPB or ECMO facilities; going to a hospital without these facilities wastes valuable time. In more isolated settings, such as parts of Alaska, other management guidelines have been published to reflect the practicalities of evacuation to a medical centre with re-warming facilities [5].

Advanced Life Support in Hypothermia

Intubate and ventilate, employing protective ventilatory strategies (high positive end-expiratory pressure [PEEP], low tidal volume). Palpate the carotid pulse and observe the EKG trace if possible, for up to 60 seconds before concluding that there is no cardiac output. If the victim is pulseless, or even if there is any doubt, then start chest compressions immediately. The ratio of ventilations to chest compressions is the same as for a normothermic patient. Intravenous fluids should be warmed. Ringer's lactate (Hartmann's solution) should be avoided because the hepatic metabolism of lactate may be diminished and lead to increased lactic acidosis [1]. Epinephrine has been shown experimentally to improve coronary perfusion pressure in hypothermic cardiac arrest in pigs, but not survival [26]. Epinephrine should be withheld in both the field and in the hospital until the core temperature exceeds 30°C, because: a) the adre-noceptor is less responsive at low temperatures; and b) decreased drug metabolism may lead to potentially toxic plasma concentrations of any drug given repeatedly [24]. Amiodarone is similarly affected [42]. Once 30°C is reached during rewarming, the intervals between doses should be doubled until core temperature approaches normal when standard protocols should be used. Arrhythmias other than VF tend to resolve spontaneously as the core temperature increases. Bradycardia is usually physiological and does not require pacing unless persistent after rewarming [37]. Defibrillation may be attempted pre-hospital, but must be limited to three shocks, even if VF or ventricular tachycardia (VT) persists, until the core temperature is greater than 30°C (45) **(figure 3)**.

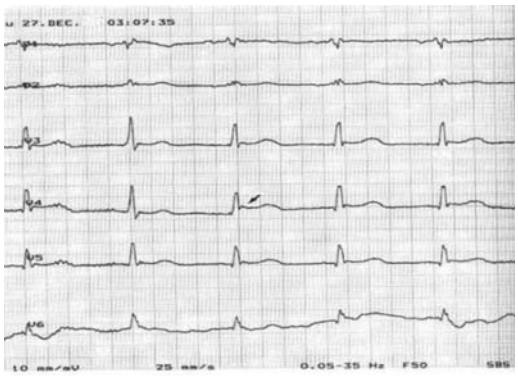

Figure 3. *Systemic hypothermia associated with distinctive bulging of J point Prominent J waves with hypothermia are referred to as Osborne waves*

Afterdrop

This is a continued fall in the core temperature after removal from the cold stress, and it may occur during rewarming [3, 51]. It is important because further cooling of the heart lowers the threshold for VF (14). It is caused by conductive heat loss along tissue thermal gradients (i.e., between the core of a muscle where the temperature is greater than at the cooler periphery of the skin) and convective heat loss through changes in peripheral blood flow as cold peripheral tissue becomes increasingly reperfused as rewarming proceeds [11].

Circum-rescue Collapse

There are many examples of victims being rescued (usually from cold water immersion) in an apparently stable and conscious condition, only to undergo a secondary or circum-rescue collapse, with symptoms ranging from syncope through to

ventricular fibrillation and cardiac arrest [15]. Deaths have been described just before, during, or soon after rescue, as well as up to 24 hours later [4, 21]. Vertical extrication leads to potentially fatal fluid shifts; these can be prevented by lifting the casualty horizontally [13].

Insulation and Rewarming

General measures include mitigating against the cold stress, gentle patient handling and removal of all layers of wet and damp clothing, usually by cutting. In the field, the victim should be insulated from the ground and from the wind, rain, or snow. Rewarming can be endogenous (encouraging the patient to shiver or exercise and so produce their own heat), passive external (blankets in a warm room, allowing endogenous heat production to gradually warm the patient – suitable only for conscious victims with mild hypothermia), active external (heat packs, convective warming blankets), or active internal (warmed intravenous fluids, warmed humidified air/oxygen mixes, gastric, pleural, peritoneal or bladder lavage with warmed fluids and ultimately extracorporeal blood warming [44].

Pre-hospital Rewarming

It is imperative to prevent any further cooling of the victim in the field. The heart must not be allowed to cool any further – the threshold for VF will be lowered. Active warming must, therefore, commence in the field, with the caveat that patient handling is safe and controlled [17]. The most practical method of active warming in the field is to place heat packs on the skin near to major blood vessels (neck, thoracic inlet, axillae, abdomen and groin). Warmed, humidified air/oxygen mixes have little thermal advantage [27]. Arterio-venous anastomosis warming can be useful in a base camp setting or aboard ship; the arms, forearms, lower legs, and feet are immersed in water at 42 or 45°C, giving rewarming rates between 6.1 and 9.9°C per hour respectively [6]. The arteriovenous anastomoses in the fingers and toes act as the heat exchanger in this elegant technique. Whole-body immersion in hot water is contraindicated [48]. This form of rapid surface warming will cause massive vasodilatation and hypotension, and is likely to provoke dysrhythmias and cardiovascular collapse [49].

In-Hospital Critical Care (figure 4)

The in-hospital management of hypothermia follows the primary survey – resuscitation – secondary survey approach. Cardiovascular stability will only be achieved through stopping the fall in core temperature and establishing rewarming [35]. Correction of metabolic and electrolyte disturbances and intravenous fluid replacement runs concurrently. Esophageal or urinary bladder electronic temperature probes are more accurate than rectal probes [25].

The reduction in the core temperature does not dictate the method or rapidity of rewarming – the presence or absence of a perfusing rhythm is the critical deciding factor [28]. Even in the patient with severe hypothermia, but with a perfusing rhythm, forced-air rewarming has been shown to be an effective method leading to eventual discharge with a very good functional recovery [32].

However, in the presence of a cardiorespiratory arrest and severe hypothermia, the priority is to restore a perfusing rhythm [50]. The rate of rewarming must be rapid – in excess of 2°C per hour – and achieved by invasive means [38]. In an institution without CPB or ECMO facilities, the options for extracorporeal warming of blood are limited to veno-veno hemofiltration, or else pleural, peritoneal and bladder lavage with warmed fluids that internally warm the heart and major blood vessels [19]. When the triage of patients is performed at the incident scene, it makes little sense to evacuate such patients to a hospital facility that does not have CPB or ECMO as these techniques remain the gold standard for rewarming the hypothermic victim [50]. Once a spontaneous circulation has been restored, it is recommended that standard strategies for post resuscitation care be employed. There is no evidence to support the routine use of steroids, nor of antibiotics, unless there is coexistent sepsis [34].

Principals of Extracorporeal Rewarming

Rewarming rates can be as high as 10°C per hour. Survival to discharge with excellent neurological function is possible even after 1 or 2 hours of asystolic cardiac arrest. Success rates may be as high as 64% in patients who are not asphyxiated prior to becoming hypothermic; in contrast, submersed patients who have drowned and then become hypothermic, or avalanche victims who have asphyxiated before becoming hypothermic have a very poor prognosis [8].

The rate of rewarming is a function of the temperature of the blood and the blood flow rate in the device. In elective cardiac surgery, it is well known that large temperature gradients between the blood in the circuit and the patient's core temperature cause a worse neurological outcome [16]. A gradient of 5-10°C is commonly used and allows thorough heating of the patient both centrally and peripherally and reduces the possibility of an afterdrop. Alpha stat acid base management should be employed during rewarming (where no correction for temperature is made during blood gas analysis). In all cases the blood temperature should never exceed 40°C as

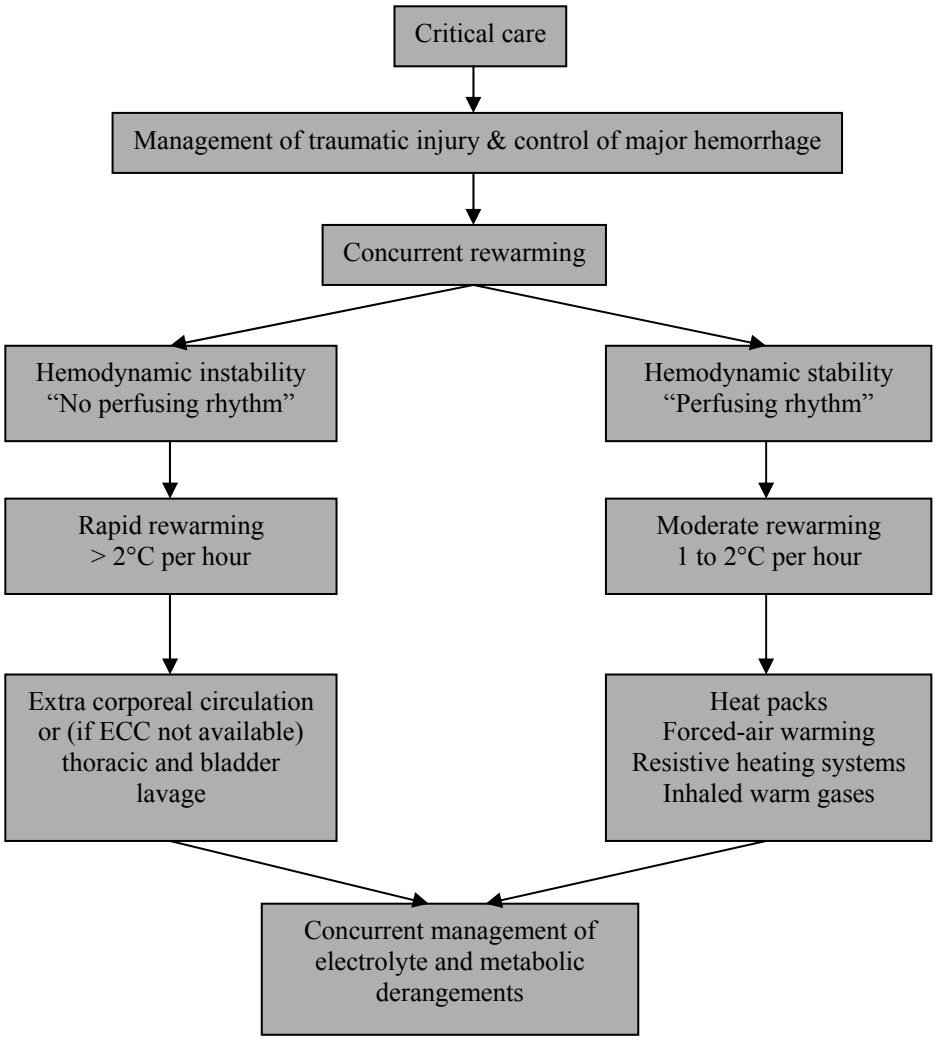

Figure 4. *Algorithm for in-hospital care of patients with severe accidental hypothermia*

higher temperatures cause denaturing of cellular and humoral elements of the blood (23).

Extracorporeal Rewarming Devices

These can be divided into devices applicable for patients with a cardiac output and those that also support the circulation.

Patients with cardiac output

I) Veno-venous rewarming circuit: A simple circuit consisting of 3/8" tubing (for adults), a centrifugal pump head and a heat exchanger, e.g., ECMO Therm (Medtronic). Access is percutaneous veno-venous and the circuit can run without heparin; it also makes an excellent rapid transfusion device (M Hines, Wake Forest, Personal communication). Avoiding full heparinization is obviously beneficial in trauma patients [23].

II) Continuous veno-venous hemofiltration: Unfortunately the blood flow rate of these devices is very limited, e.g., 180 ml/min. for the Gambro Prisma, which limits the thermal transfer capability.

Both these devices could be used for patients who suffer a cardiac arrest as long as cardiac massage is continued. However, the rewarming will be extremely slow as thermal transfer will be limited by the low cardiac output achievable with external cardiac massage, usually only 20% of normal. If the patient is presenting in cardiac arrest then one of the following devices should be used [19].

Patients in cardiac arrest

I) Cardio-pulmonary bypass: An adult circuit can be used to support patients larger than 40 kg. Smaller patients will need to go to a pediatric cardiac surgical unit. Blood flow rates of up to 2.4 l/m^2/min. allow full support of gas exchange and cardiac output. The circuit is relatively complex. Blood is usually drained into a venous reservoir from where it flows to the pump, of either centrifugal or roller type. The blood is then propelled through an oxygenator, which is usually made from polypropylene hollow fibers with an integral heat exchanger. There is often an arterial line filter to remove particulate debris before the blood is returned to the arterial system of

the patient, if applicable, a system of suction tubes collecting in a cardiotomy reservoir also allows shed blood from the surgical field to be recirculated. The circuit has many areas where blood is stationary and it therefore requires complete anti-coagulation with heparin 300 units/kg to give an activated clotting time (ACT) of 500-1000 seconds. This is obviously a disadvantage in patients who have suffered trauma, particularly intracerebral bleeding.

The circulation is usually accessed via direct cannulation of the heart and great vessels via a median sternotomy. This approach has the advantage of great speed, and the ability to decompress the left ventricle which may become distended once CPB is initiated, especially in the patient who has resistant VF. This is achieved by placing a vent either in the left atrium, left ventricle, or pulmonary artery according to preference. Often VF will revert spontaneously once the heart is decompressed. The other advantage of transthoracic cannulation is seen in small children where the femoral vessels are unusable for access. In this situation the right carotid and jugular vein are usually used if extra-thoracic access is employed. Ligation of the carotid and jugular during cardiac arrest has a much higher incidence of right-sided brain lesions in babies being cannulated for ECMO compared to those who were not in cardiac arrest [29]. In older children and adults the femoral vessels can be used for cannulation either percutaneously or by cutdown; typical adult femoral cannulae would be a 28F venous cannula and a 21F or 23F arterial return [47].

Even patients with a core temperature < 14°C can be rewarmed in 1-2 hours on CPB. Thorough warming is confirmed by measuring the bladder or peripheral temperatures before discontinuing CPB; these should usually be in excess of 35°C. After weaning from bypass, heparin is reversed with protamine and coagulopathy is corrected by transfusion of platelets, plasma and cryoprecipitate as appropriate. Anti-fibrinolytics such as tranexamic acid or aprotinin can also be helpful for post bypass hemorrhage as can recombinant activated factor seven (Novoseven, Novo-Nordisk).

II) extracorporeal membrane oxygenation: ECMO uses modified CPB technology to provide prolonged cardio-respiratory support in the ICU. It has several advantages over CPB for resuscitating patients with severe hypothermia.

• The circuit is designed to eliminate areas of stasis so there is no venous reservoir and no suction apparatus. This allows much lower doses of heparin to be used than are needed for CPB. Only 100 units/kg of heparin are given prior to cannulation and then 30-60 units/kg/h are given to maintain an ACT of 160-200 seconds. If there is recent trauma or ongoing bleeding micro-dose heparin (10 units/kg/min.) or even heparin free ECMO can be used for short periods of time. Aprotinin infusion is a useful adjunct to reduce bleeding on ECMO.

• ECMO can be used to provide prolonged respiratory support; for instance, in the immersion victim or trauma patient there may be significant lung injury which requires extra-corporeal gas exchange after rewarming.

• ECMO causes a much smaller inflammatory response than CPB [30].

It would be usual to opt for veno-arterial ECMO in a hypothermic patient in cardiac arrest but it is possible to use veno-venous bypass and cardiac massage. If the left heart is distended then a vent can be inserted in the same way as during CPB; the tubing is simply connected into the venous side of the circuit. Care must be taken not to allow any air to enter the circuit via the vent as the ECMO circuit is not designed to have air in it, and can easily pump this air back to the patient if a roller pump is being used.

The oxygenator is usually constructed from heparin coated poly-methyl pentene (PMP) and can be used safely for short periods without intravenous heparin, particularly in the presence of a coagulopathy. Older circuit designs use solid silicone membrane lungs, which are very effective but have a higher priming volume and cause slightly more blood activation than the PMP devices [22].

The same issues pertain to ECMO cannulation as those discussed above for CPB. To summarize, avoidance of carotid and jugular ligation is sensible in patients who are arrested, and the femoral vessels are the ideal choice in any patient where they are large enough (usually from the age of 2). Trans-thoracic cannulation is preferred initially in younger patients in cardiac arrest, moving to cervical cannulation to allow hemostasis after 12-24 hours.

The rewarming approach is the same as for CPB keeping the temperature gradient between the blood and core temperature 5-10°C and never allowing the blood temperature above 40°C. This will allow rewarming rates of 5-10°C per hour. It is worth attempting defibrillation once rewarming has been initiated, particularly if the heart has been decompressed. However, if it is unsuccessful, further attempts should be postponed until the core temperature is above 30°C. If the VF is resistant at this temperature, administration of magnesium and amiodarone can be helpful to facilitate cardioversion.

Conclusion

For doctors in tertiary care centers, the most important task is to identify patients who can benefit

from transfer to their facility. Hospitals without CPB or ECMO should encourage transfer of patients in cardiac arrest directly from the scene to units that do have these facilities. This will drastically reduce journey times and time-to-definitive treatment by eliminating the need for a secondary transfer. In determining the correct disposition for the patient, a balance has to be struck between the journey time entailed in a critical care transfer, the risk of further cooling of the patient (perhaps provoking a fatal dysrhythmia, or lowering to a core temperature from which resuscitation is impossible), the vulnerability of the patient during the transfer, and the advantages offered by advanced tertiary care facilities. The old adage 'not dead until warm and dead remains pertinent. If the heart has stopped send the patient to a hospital with CPB or ECMO!

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