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REVIEW

Hypothermia in the trauma patient

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Summary Hypothermia is a common finding in severely injured patients. Historically described as a consequence of wartime casualties where cold exposure was common, this topic has resurfaced in the trauma literature because of the increasing recognition of the morbidity and mortality associated with hypothermia. Hypothermia, along with acidosis and coagulopathy, has been identified as a component of the ''lethal triad'' in injured patients, and has been shown to contribute to increased mortality in these patients. Decreases in core temperature during the course of initial evaluation and resuscitation are common, and can contribute to poor outcomes in the injured patient. As induced hypothermia has been shown to be beneficial in some clinical situations, recent animal studies have attempted to investigate whether hypothermia in the trauma patient has any beneficial effects. This review examines the incidence and pathophysiology of hypothermia, and discusses mechanisms of heat loss and rewarming techniques that can be utilized in the trauma patient.

Introduction

Hypothermia in humans is defined as a core temperature below 35 °C,[4,37] and has traditionally been classified into zones of severity based on the physiologic changes that occur with decreasing body temperature.[12] Mild hypothermia, with core temperatures between 32—35 °C, is usually well tolerated, with compensatory cardiovascular changes designed to maintain temperature homeostasis. Below 32 °C, however, cardiac conduction disturbances become apparent, and at 28 °C serious dysrhythmias can occur. Below 28 °C heat production mechanisms begin to fail, and at 20 °C virtually all patients are asystolic.

These original definitions were introduced to describe hypothermia resulting from environmental exposures. In a multicenter study of over 400 cases of hypothermia due to exposure, core temperature less than 32 °C was associated with 21% mortality.[6] In trauma patients, however, the presence of hypothermia was associated with a much higher mortality than patients who had suffered exposure. Jurkovich found that in 71 adult trauma victims, core temperature less than 32 °C was associated with 100% mortality, independent of the presence of shock, injury severity score, or volume of fluid resuscitation.[30] Because the prognosis associated with hypothermia in the trauma victim is so poor, a separate classification of hypothermia has been developed for use in the injured patient (Table 1). As such, hypothermia in the trauma patient is classified as mild (36—34 °C), moderate (34—32 °C), or severe (below 32 °C).[15]

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Table 1 Traditional classification of hypothermia and revised definitions for the trauma patient

Degree of hypothermia	Traditional classification (°C)	Trauma classification (°C)
Mild	32–35	34–36
Moderate	28–32	32–34
Severe	20–28	<32
Profound	14–20	
Deep	<14	

Physiologic effects of hypothermia

Regulation of body temperature occurs in the hypothalamus, which receives input from thermoreceptors along the distribution of the internal carotid artery, the posterior hypothalamus, and peripheral skin receptors.[35] Homeothermic responses to cold include increased muscle tone and shivering, as well as metabolic increases from release of catecholamines and thyroxine.

Hypothermia can have marked physiologic effects on the cardiac, pulmonary, neurologic, and haemostatic systems (Table 2). In patients with mild hypothermia and normal thermoregulation, oxygen consumption can increase dramatically. In studies of postoperative patients, a temperature decrease of 0.3 °C decrease in the core temperature of postoperative patients was associated with a 7% increase in oxygen consumption.[70] Further decreases in core temperature of 1.2 °C resulted in a 92% increase in oxygen consumption.[52] Other studies have suggested that shivering can increase oxygen consumption by as much as 400%.[1]

Although mild hypothermia can cause increases in cardiac output and oxygen demand, moderate or severe hypothermia may cause cardiac depression.[46] As intropic functions of the heart are depressed, decreased cardiac output and hypotension can occur. Arrhythmias may occur as a result of cold-induced conduction abnormalities.[24] Atrial fibrillation is not uncommon with core temperatures of less than 30, and at 25 °C ventricular fibrillation appears spontaneously.[46] Furthermore, hypothermia reduces oxygen release from hemoglobin to tissue, which can further exacerbate impaired oxygen delivery, a potentially devastating complication in the multiply injured patient with severe oxygen debt.[7]

From a pulmonary standpoint, hypothermic patients often have a decreased respiratory rate. This is caused by depression of the brainstem respiratory centre. Respiratory arrest, however, is rarely seen unless core temperatures drop below 24 °C.[66] Gastrointestinal motility decreases with hypothermia, and mild ileus may be seen at temperatures less than 32 °C.[51] With severe hypothermia, gastric mucosal erosions and ulcers, and haemorrhagic pancreatitis have been reported. Hyperglycaemia may be accentuated by hypothermia. With high levels of circulating catecholamines, hyperglycaemia is common in the normothermic trauma patient. Hypothermia in the injured patient exacerbates hyperglycaemia by decreasing insulin production and increasing end organ insulin resistance.[51] Decreases in core temperature can also cause significant peripheral vasoconstriction, and may also result in significant shifts of fluid into the intravascular space. ''Cold diuresis'' may be a compensatory response in an effort to decrease overall blood volume, or may result from temperature related decreases in renal enzyme activity.[22]

Hypothermia has a profound effect on the central nervous system, as cerebral blood flow is highly sensitive to changes in core temperature. A 1 °C decrease in core temperature can cause a drop in cerebral blood flow of 6–7% and cause clinical

Table 2 Physiologic effects of hypothermia

Degree of hypothermia	Mild (32–35 °C)	Moderate (28–32 °C)	Severe (<28 °C)
General metabolic	Shivering, increased oxygen consumption	Increased oxygen consumption	Acidosis
Cardiac	Vasoconstriction	Atrial arrhythmias, bradycardia	Ventricular arrhythmias, decreased cardiac output
Respiratory	Tachypnea, bronchospasm	Decreased respiratory drive	Apnea
Neurologic	Confusion, hyperreflexia	Decreased level of consciousness, hyporeflexia	Coma, absent reflexes
Renal		Cold diuresis	
Gastrointestinal	Decreased motility		
Coagulation	Platelet dysfunction, impaired clotting enzyme function		

signs of impaired judgment, confusion, and clouded consciousness.[51] Reflexes are also altered by hypothermia. Hyperreflexia is seen at 32 °C, but these reflexes decrease as core temperature drops further. Below 27 °C, pupillary light reflexes and deep tendon reflexes are lost, and may cause the patient to appear dead.[46]

One of the most widely documented effects of hypothermia is coagulopathy. Hypothermia affects haemostasis on several levels, altering platelet function, enzyme kinetics of the coagulation cascade and the fibrinolytic system. Early animal studies have shown that hypothermia causes reversible platelet sequestration in the liver and spleen, contributing to continued blood loss.[64] Hypothermia also directly affects platelet function by decreasing production of thromboxane B_2 and expression of platelet surface molecules.[43,63]

More recent research has focused on the effects of hypothermia on the coagulation cascade. When clotting assays were performed at temperatures below 37 °C, the results were significantly prolonged.[20,49,50,53] Prothrombin time was significantly increased at temperatures below 35 °C, and activated partial thromboplastin time was similarly prolonged at temperatures below 33 °C. In contrast, when clotting tests using blood from hypothermic rats were performed at 37 °C, no significant abnormalities were seen. This suggests that the major mechanism of hypothermia-induced coagulopathy results from altered enzymatic activity. In fact, at 33 °C the clotting process is functionally equivalent to having a 33% factor-IX deficiency, even in the presence of normal clotting factor levels.[29] These effects are directly reversible with the correction of hypothermia, emphasizing the importance of maintaining normal core temperatures in injured patients. Hypothermia has also been noted to have an effect on the fibrinolytic system. Fibrinolysis was noted to increase in hypothermic animals, and is felt to be a result of impaired inhibitors of clot lysis, such as plasminogen activator inhibitor or alpha-2-antiplasmin.[69] In the clinical setting, the trauma patient's coagulation profile may be reported as normal, as the sample is usually heated to 37 °C in the laboratory before it is tested.[46]

In addition to the coagulation cascade, other metabolic systems that depend on enzyme activity are also adversely affected by hypothermia. As drug metabolism and elimination are temperature dependent, many drugs can remain in the body for extended periods in the hypothermic patient. Hypothermia also prolongs the duration of action of several commonly utilized drugs, such as muscle relaxants and benzodiazepines, by as much as 40–50%.[26,34]

Causes of hypothermia in the trauma patient

From the time of injury, trauma itself alters the normal central thermoregulation and blocks the shivering response. The thermoneutral zone is defined as the ambient temperature at which basal thermogenesis offsets continuing heat losses, and in humans, occurs at 28 °C.[12] Maintaining euthermia when ambient temperatures are below the thermoneutral zone requires increased heat production and oxygen consumption. When tissue oxygen consumption is limited by shock, insufficient heat production occurs and results in hypothermia. Animal studies have demonstrated that injured animals began to shiver at a lower core temperature than uninjured animals.[60] Similarly, shivering is inhibited during episodes of hypotension or hypoxaemia.[59,61] In fact, human studies have documented that shivering occurred in only one of 82 severely injured hypothermic trauma victims.[61]

Other studies have suggested that tissue hypoperfusion and resuscitation may elicit hypothermia as a response. In hypovolaemic shock, maximal tissue oxygen extraction is exceeded and oxygen consumption declines, resulting in decreased heat production. In rats, haemorrhagic shock was associated with hypothermia, with a rise in core temperature seen during resuscitation.[2] Other studies have shown that there is a significant correlation between blood pressure at admission and lowest intraoperative temperature, with 90% of patients with an intraoperative temperature of less than 33% arriving to the emergency room in shock.[3] Finally, some investigators have suggested that hypothermia provides a protective effect during shock by decreasing metabolic activity and oxygen consumption.[31,40] While hypothermia in the trauma patient is clearly multifactorial, it is, unfortunately, quite common. The incidence of admission hypothermia has varied in numerous studies, ranging from 10–66%.[17,36,58] However, several studies have shown that although admission core temperature may be normal, hypothermia often occurs during the evaluation and resuscitative period. Bernabei found that although only 12% of patients arrived in the emergency department hypothermic, 92% lost temperature during the initial evaluation.[3] Gregory et al. found that 57% of trauma victims requiring immediate operative intervention became hypothermic in the period between injury and completion of the initial operative procedure.[19] Trauma patients are often disrobed early during the evaluation, and the temperature gradient of 10–15 °C between body and room temperature can result in radiant heat losses that are not compensated

for by altered thermoregulatory response. Infusion of unwarmed intravenous fluid, especially in patients who receive large volumes of crystalloid during resuscitation, can exacerbate hypothermia. Thus, although a traumatically injured patient may arrive with a normal core temperature, the development of hypothermia during the initial treatment phase is common.

Management of hypothermia

The first step in correcting hypothermia is the determination of core temperature. Unfortunately, studies have indicated that temperature is not often measured in the trauma patient, especially those who are severely injured.[44] While oral and rectal temperatures are most commonly employed in the emergent setting, oesophageal and bladder temperatures have been shown to correlate more closely with core temperatures.[35] A more recent development is the use of infrared tympanic membrane thermometry, which uses radiant energy emitted from the external canal wall and tympanic membrane to estimate body core temperature.[8] Some studies have indicated that measurements using this technique are comparable to core temperature measurements taken from pulmonary artery catheters.[54] In other studies of patients undergoing cardiopulmonary bypass for cardiac revascularization, tympanic membrane thermometry showed a higher degree of correlation with venous blood temperature than that of oesophageal measurements.[23]

One important concept in the discussion of rewarming is specific heat. Specific heat is defined as the number of kilocalories required to raise the temperature of one kilogram of a substance by 1 °C. The specific heat of the human body is roughly 0.83 kcal/(kg °C). The heat lost by a hypothermic patient can be determined by the general thermodynamic equation

$$Q = mc(\Delta T)$$

where Q = heat loss (kcal), m = mass (kg), c = specific heat (kcal/kg °C) and ΔT is the chance in core temperature (°C).

There are four major mechanisms of heat transfer: convection, conduction, radiation, and evaporation. In convective loss, heat is transferred to the environment by air that is in contact with the body. The rate of heat transfer depends on air velocity, such as the heat loss generated by a fan blowing on a warm object. Conductive losses occur from heat transfer to another object by direct contact, and the rate of conductive losses depends on the characteristics of the two objects. For example, the rate of heat transfer from an object submerged in water is 32 times that in air. Radiation is heat loss resulting from a temperature gradient. This form of heat dissipation may account for 55–65% of the total body heat loss at basal metabolic rate. Finally, evaporative loss occurs with the conversion of liquid water to the gaseous phase, such as with breathing. Convection, conduction, and radiation are the most significant forms of heat transfer, and are also the most important means of warming hypothermic patients.

Rewarming of the injured patient can be undertaken using either passive or active methods. Passive rewarming consists of optimizing environmental conditions while allowing the patient's own heat-generating capabilities to correct decreases in core temperature. These techniques include manoeuvres such as removing the patient from the cold environment, increasing ambient room temperature, and providing blanket coverage while endogenous heat production restores normal core temperature. Based on measurements of resting oxygen consumption, the average person generates about 1 kcal/(kg h), and this heat production is just sufficient to offset ongoing heat losses in a thermoneutral environment.[16] The thermoneutral ambient temperature for humans is about 28 °C.[12] Thus, the basal heat production in the average (70 kg) patient produces a rewarming rate of about 1.2 °C/h if all other heat loss is prevented. Heat generating mechanisms, such as shivering, can increase the rate of rewarming to 3.6 °C/h. However, oxygen consumption can increase by three- to four-fold in these situations, and can be detrimental in the critically injured patient.[28] Overall, passive rewarming can result in significant anaerobic metabolism and lactic acidosis, and therefore should be used only for mildly hypothermic, healthy patients with intact thermoregulatory responses.

Active rewarming includes external methods of rewarming as well as methods directed at rewarming the core. External rewarming techniques include the use of heating blankets, convective air blankets, reflective blankets, and radiant heat shields. Both fluid and air circulating warming blankets are available, but convective air blankets are perhaps the most commonly used. In normal subjects, they transfer more heat than radiant warmers (heat lamp) or fluid circulating heating blankets.[55] One study documented that the use of convective warming devices increased core warming from 1.4 °C/h with the use of blankets alone, to 2.4 °C/h.[57] In more hypothermic patients, however, peripheral vasoconstriction can limit the efficacy of forced air warming. In the intoxicated trauma patient, who may have a chemically induced

peripheral vasodilation, such techniques may be more effective.

Radiant warmers use radiation heat transfer to raise core temperature. When using these devices a blanket should be placed over the patient to lessen the risk of direct thermal injury. Several studies have demonstrated that radiant warmers are comparable to convective air blankets and regular blankets in raising core temperature.[14,67] Airway rewarming works by limiting the normal heat and water loss from respiration, but because of the very low thermal conductivity and specific heat (0.24 kcal/(L °C)) of air, ventilation with warm air is an inefficient method of rewarming in the trauma victim.[46]

Body cavity lavage, on the other hand, can provide a means of transferring a large amount of heat to patients suffering from moderate to severe hypothermia. Not only is the specific heat of water greater than that of air (1 kcal/(l °C) versus 0.24 kcal/(l °C)), but the rate of heat transfer in water is 32 times that of air. Thus, efficient heat transfer can be achieved in hypothermic patients.[46] Peritoneal lavage is relatively simple, but achieving adequate return of fluid may sometimes be difficult. Contraindications would include haemoperitoneum, free intraabdominal air, and prior laparotomy. Peritoneal lavage can be an effective means of raising core temperature in the operating room, if the abdominal cavity has been opened for exploration. Pleural rewarming techniques have also been described, and provide an easier route of fluid return than peritoneal lavage.[7] However, both of these techniques are invasive, and other means of rewarming should be considered first.

Perhaps one of the simplest means of transferring a large amount of heat to the core of patients with moderate to severe hypothermia is the administration of warmed intravenous fluids. This becomes especially significant in patients who require massive fluid resuscitation. Warmed intravenous fluids increase core temperature by conduction, which is the most effect method of heat transfer. Crystalloid fluids can be warmed in a water bath or in the microwave, and generally are infused at a temperature of 40 °C. Animal studies have indicated that infusion of crystalloid fluids at temperatures up to 65 °C have not been associated with adverse effects.[11] Infusion of warmed blood products is critical because they are stored at 4 °C. These are most commonly warmed with blood warmers or by mixture with warmed saline. However, raising the temperature of one unit of 4 °C blood requires the addition of an equal volume of 70 °C saline. Even if this could be done efficiently, the mixture would need to be infused very rapidly, as marked cooling occurs when warm fluids are left at room temperature. As an example, a litre of fluid at 37 °C cools to 34 °C within 5 min and to 32 °C within 15 min when left at room temperature.

Conventional fluid warmers were introduced when maximum flow rates of 150 cm^3/min were considered adequate. However, many injured patients today require fluid infusion at much higher rates. Currently three types of in-line fluid warmers are in use—those that use dry heat, those that used still water bath and those that use counter current water baths. Dry heaters and still water baths utilize long plastic intravenous tubing coils and require long contact times. Because conduction is poor, little warming occurs at flow rates over 150 cm^3/min. The slow flow rates required for adequate heat transfer to the fluid ultimately result in significant cooling of the fluid once it has left the warmer, and in reality normothermic infusions are virtually impossible.[16]

These problems have been alleviated with the development of countercurrent fluid warmers. These devices pass fluid through a water bath contained in a length of thin aluminium tubing. Because the thermal conductivity of aluminium is much greater than that of plastic intravenous tubing, infusions of up to 750 ml of saline or one unit of blood per minute at euthermic temperatures are possible.[16]

Extracorporeal circulatory rewarming provides infusion of warm intravenous fluid and recirculation of the patient's blood, and is the currently the most effective method of restoring normal core temperatures. Cardiopulmonary bypass, developed decades ago, has been described as a method of rewarming for patients with severe hypothermia. This technique has primarily been utilized in patients with profound exposure-related hypothermia,[32,65,68] and prolonged resuscitation techniques are often necessary as cardiac asystole is a common finding in these patients. Attempts at rewarming trauma patients using these techniques were initially complicated by the need for systemic anticoagulation. The development of heparin-bonded systems has allowed the use of cardiopulmonary bypass in severely hypothermic trauma patients without fear of excessive bleeding.[47,62] Experience with these techniques in the trauma patient has been limited, although case reports and small series indicate that it might be useful in select situations.

When cardiopulmonary bypass equipment and expertise are not available, other methods of extracorporeal rewarming may be utilized. Arteriovenous and venovenous bypass techniques have been described and provide rapid core rewarming. A roller pump and countercurrent heat exchanger can warm the patient's blood or fluids to 40 °C at

150–400 ml/min.[17,18] One method of extracorporeal circulatory rewarming is continuous arteriovenous rewarming (CAVR). This technique takes advantage of the patient's own blood pressure to drive the blood through the heating mechanism of a countercurrent fluid warmer via percutaneously placed femoral and arterial venous catheters.[13] CAVR does not require a blood pump, membrane oxygenator, or systemic heparinization, making it applicable in the trauma patient.

Hypothermia in the operating room

Hypothermia in the injured patient has marked clinical implications. Trauma patients with a core temperature of less than 32 °C were noted to have 100% mortality,[30] compared to a 23% mortality for patients with the same temperature from exposure related hypothermia.[6] Although many of these deaths are attributed to underlying conditions or associated diseases, several studies have demonstrated a significantly higher mortality among trauma victims with hypothermia. These studies have shown that this increase in mortality is independent of hypotension, fluid requirements, age, injury severity, or duration of surgical intervention.[56,58] Injured patients with hypothermia often require greater fluid administration, increased blood transfusions, and longer hospital stays than their normothermic counterparts.[21,30]

Many injured patients arrive in the operating room with a combination of hypothermia, coagulopathy and acidosis. These metabolic disturbances may go unrecognized during a complicated surgical procedure, placing the patient at higher risks of complications and death. Feliciano et al. examined a series of 300 patients with abdominal gunshot wounds in 1988. While they found that the overall survival rate was almost 90%, in patients who suffered major vascular injuries the survival was only 60%, and this declined dramatically when other visceral injuries were simultaneously encountered.[9] They concluded that the triad of coagulopathy, hypothermia and acidosis contributed to a significant number of deaths in this study group. Similarly, Ferrara et al. reported that patients with hypothermia, coagulopathy and acidosis who required massive transfusions had over 90% mortality.[10]

Hypothermia induced coagulopathy may be difficult to recognize. In the clinical setting, as discussed above, the trauma patient's coagulation profile may be reported as normal, as the sample is usually heated to 37 °C in the laboratory before it is tested.[46] Animal studies have emphasized that the hypothermia-induced changes in the coagulation system may be refractory to blood product administration[33] and can only be reversed with rewarming.[27] Thus, the administration of blood products may not alter haemostasis if the patient remains persistently hypothermic.

As the presence of the ''lethal triad'' in the patient with multiple injuries is frequently life threatening, adequate rewarming, in addition to the replacement of coagulation factors and fluid resuscitation, is essential. Intra-operative manoeuvres to minimize heat loss, such as raising the ambient room temperature above the thermoneutral zone might be helpful. Active external rewarming, rapid administration of warm intravenous fluids and intra-operative peritoneal lavage with warm fluid can also be used to increase core temperature. Inability to correct these deficits in the operating room should prompt ''damage control manoeuvres'', with expeditious return of the patient to the intensive care setting. Abdominal packing and the use of temporary abdominal closures may aide in limiting the duration of operation. Completion of operative procedures should be undertaken only after adequate resuscitation, including correction of coagulopathy, acidosis and hypothermia.

Ongoing research and controversies

Despite the physiologic changes induced by hypothermia and the potential for complications in the trauma patient, hypothermia has been noted historically to improve outcomes in certain clinical situations. Induced hypothermia is routinely employed during organ transplantation, cardiac surgery, and neurologic surgery, as it limits tissue ischaemia by decreasing overall metabolic activity and cellular oxygen consumption. As such, some authors have proposed that hypothermia may provide a protective mechanism during shock,[12] and the question of whether hypothermia in the trauma patient may actually be beneficial is the topic of continuing research.

Induced hypothermia for the treatment of neurological injuries was used widely in the 1940s. Although this fell out of favour during the 1980s, it has recently resurfaced in the last decade as a potentially beneficial method for the treatment of severe head injuries. Some studies have shown lower mortality and better neurological outcomes with mild or moderate hypothermia.[39,48] There were, however, detrimental side effects of hypothermia, including electrolyte abnormalities, hypotension, and arrhythmias.[48] Issues of hypothermia-induced coagulopathy and effects of hypothermia on associated traumatic injuries were not

discussed in detail. Despite these results, a recent meta-analysis suggested that hypothermia is not beneficial in the treatment of severe head injuries,[25] and it appears that further studies are indicated before hypothermia can be recommended routinely in the treatment of head injuries.[38]

During the last decade, studies have also begun to determine whether there are any beneficial effects of hypothermia during resuscitation from haemorrhagic shock. Some studies, focusing on animal models of uncontrolled haemorrhagic shock, have suggested an improved outcome with moderate hypothermia after haemorrhagic shock.[5,31] Meyer and Horton reported that moderate hypothermia in dogs decreased metabolic needs and maintained cardiac contractile function after haemorrhagic shock.[41,42] In contrast, other studies have not found hypothermia to be protective. Krause et al. found that while volume replacement in pigs with haemorrhagic shock returned cardiac output to baseline levels in normothermic animals, it had little effect on cardiac output in animals with persistent hypothermia.[33] Another study by Mizushima et al. also found that prolonged hypothermia after haemorrhagic shock decreased myocardial contractility and resulted in depressed cardiac function. Restoration of normothermia during resuscitation significantly improved cardiac performance and visceral blood flow.[45] Several of these animal studies also examined the effects of hypothermia-induced coagulopathy and reiterated prior findings; namely, clotting times are prolonged with decreased core temperatures[27] and were refractory to correction with blood products during persistently low core temperatures.[33]

Thus, although hypothermia may be protective by decreasing oxygen consumption, the effect of prolonged hypothermia during resuscitation after haemorrhagic shock is as yet unclear. Given these findings, the current generally accepted practice of simultaneous aggressive rewarming with fluid resuscitation may be the best practice until further studies indicate otherwise.

Summary

Hypothermia is a common finding in the trauma patient, and contributes to increased morbidity and mortality in this group of critically ill patients. Although initial temperatures may be normal, decreases in core temperatures during the course of initial evaluation and resuscitation are common. Hypothermia contributes to alterations in physiologic functions, and through alterations of the normal coagulation function, can contribute to further haemorrhage and shock in the injured patient. Both passive and active rewarming techniques, including the use of warming blankets and other conductive heat devices, countercurrent fluid warmers, and extracorporeal devices in severe cases, should be utilized in cases of mild and moderate hypothermia. In the severely hypothermic patient, cardiopulmonary bypass using heparin-bonded equipment should be considered, particularly in the asystolic patient. Intraoperative correction of low core temperature is a critical component in reversing the lethal triad of hypothermia, acidosis and coagulopathy in the patient undergoing emergency procedures, and failure to correct these deficits should prompt the use of damage control techniques to minimize morbidity and mortality in these critically ill patients.

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