

HYPOTHERMIA

Hypothermia in Trauma Victims—Friend or Foe?

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Learning Objectives: 1) To describe the pathophysiology of hypothermia in trauma, 2) to review methods to prevent and treat hypothermia, and 3) to discuss the role of therapeutic hypothermia in trauma.

Abstract

Trauma is a common cause of hypothermia. Risk factors for hypothermia in the trauma patient include environmental exposure, alcohol intoxication, burns, spinal cord injuries, head injury, shock, and general anesthesia. Patients requiring emergency surgical intervention may suffer additional hypothermic insults due to heat loss in the cold operating room environment and iatrogenic causes. General anesthesia alters the threshold for thermoregulatory vasoconstriction and shivering. Hypothermia has been associated with increased morbidity and mortality, likely due to impaired coagulation, peripheral vasoconstriction, elevated oxygen consumption associated with rewarming, excess production of lactic acid and persistence of metabolic acidosis, hemodynamic instability, cardiac arrhythmias, and wound infections. Methods to prevent and treat hypothermia include passive warming, active external warming, and active internal warming. Choice of warming method depends on the degree of hypothermia, the patient's clinical condition, the experience and resources of the clinicians caring for the patient, and thermodynamic considerations. Deliberate use of hypothermia may prevent the initiation of the cascade of events after injury that leads to cell death. Hypothermia increases the tolerance time for cardiac arrest because of reduced metabolism and inhibition of deleterious effects of hypoxia. The successful application of therapeutic hypothermia after traumatic brain injury is still debated.

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Incidence and Causes of Hypothermia in Trauma Patients

Hypothermia is a well-recognized and life-threatening consequence of injury.¹ In a prehospital study of 302 injured patients, Helm et al² found that almost every second patient was hypothermic. There was no relation between season of the year and frequency of hypothermia. Entrapped patients, however, were at higher risk (98% vs. 35%; $P < 0.001$), as were patients older than 65 years ($P < 0.001$). Clinical symptoms of hypothermia such as shivering were only noted in 4%. In 94 tracheally intubated patients brought to one regional trauma centre, Luna et al³ found that almost two thirds of the patients had a core temperature of less than 36°C.

Trauma in itself, as well as bleeding with tissue hypoperfusion, alters thermoregulation and results in hypothermia.⁴ Some of the preventable factors that contribute to the high incidence of hypothermia in the trauma population are prolonged exposure in the field and administration of cold intravenous fluids (Table 1).^{1,5} Patients requiring emergency surgical intervention may suffer additional hypothermic insults from cold surgical preparation solutions and heat loss in the cold operating room environment. Administration of anaesthetic agents impairs the ability to maintain thermal homeostasis and causes internal redistribution of body heat from the warmer core to the cooler peripheral tissue, thereby further reducing core temperature in the exposed patient.⁶

Table 1. Causes of Hypothermia in Trauma Patients

Impaired Thermoregulation and Decreased Heat Production

- Injury per se
- Central nervous system injury
- Shock (tissue hypoperfusion)
- Extremes of age
- General and neuroaxial anaesthesia
- Associated medical conditions such as diabetes and cardiac failure
- Drugs and substances such as alcohol and tricyclic antidepressants

Increased Heat Loss

- Exposure
- Cold intravenous fluids and blood products
- Burns
- General and neuroaxial anaesthesia

Pathophysiological Consequences of Hypothermia

Hypothermia is associated with increased mortality and morbidity,^{1,4} with a dramatic decrease in survival at core temperatures below 34°C. In trauma patients, the traditional severity classification of accidental hypothermia has been revised, with 34–36°C classified as mild, 32–34°C as moderate,

and less than 32°C as severe hypothermia.⁴ The increased morbidity and mortality are likely due to impaired coagulation, metabolic acidosis from poorly perfused tissues, hemodynamic instability, respiratory problems, and infections. The potential adverse effects of hypothermia in the injured patient are shown in Table 2.^{1,6} Decreases in core temperature during the course of initial evaluation and resuscitation are common, and can contribute to poor outcomes in the injured patient. Hypothermia, together with acidosis and coagulopathy, has been identified as a component of the “lethal triad” in injured patients. Intense shivering may occur between 34°C and 36°C with resultant increase in oxygen demand and metabolic rate.^{1,6} During rewarming, there may be release of sequestered cold blood and acid metabolites from peripheral vascular beds, and dilation of the systemic vasculature, with resultant cardiac instability. Hemodynamic instability due to “rewarming shock” is characterized by hypotension, myocardial depression, and release of metabolic acids.⁷

Table 2. Adverse Effects of Hypothermia in Trauma

Impaired cardiorespiratory function

- Cardiac depression
- Myocardial ischemia
- Arrhythmias
- Peripheral vasoconstriction
- Impaired tissue oxygen delivery
- Elevated oxygen consumption during rewarming
- Blunted response to catecholamines
- Increased blood viscosity
- Metabolic acidosis

Bleeding diathesis

- Decreased kinetics of coagulation factors
- Reduced platelet function

Reduced clearance of drugs

- Decreased hepatic blood flow
- Decreased hepatic metabolism of drugs
- Decreased renal blood flow

Increased risk of infection

- Decreased white blood cell number and function
- Impaired cellular immune response
- Wound infection:
 - Thermoregulatory vasoconstriction
 - Decreased subcutaneous oxygen tension
 - Impaired oxidative killing by neutrophils
 - Decreased collagen deposition
- Pneumonia
- Sepsis

Hypothermia exerts a negative inotropic effect on the myocardium, and depression of left ventricular contractility occurs with moderate hypothermia.⁸ Hypothermia is also associated with atrial and ventricular arrhythmias. The initial electrocardiogram change seen with hypothermia is sinus tachycardia. As the core temperature decreases, progressive bradycardia ensues. The cardiac response to catecholamines may also be blunted in hypothermic hearts, and cold cardiac tissue poorly tolerates hypervolemia and hypovolemia.⁹⁻¹⁰

Intraoperative hypothermia increases the incidence of morbid cardiac events such as myocardial ischemia, unstable angina, and ventricular tachycardia.¹¹ Increased cardiovascular morbidity may be partly due to a greater degree of peripheral vasoconstriction, increased norepinephrine levels, and altered alpha-adrenoceptor tone in response to cold. Hypothermia may promote surgical wound infection because of thermoregulatory vasoconstriction, decreased subcutaneous oxygen tension, impaired oxidative killing by neutrophils, and decreased collagen deposition.^{6,12}

Blood glucose concentrations increase with hypothermia, which may require intensive insulin therapy to reduce mortality.¹³ Hypothermia may lead to a bleeding diathesis as a result of impaired platelet function, inhibition of clotting enzyme kinetics, and activation of the fibrinolytic cascade.^{1,4,14} Life-threatening coagulopathy in patients with severe injury who required massive transfusion is linked to temperatures below 34°C and a progressive metabolic acidosis.¹⁴

Prevention and Treatment of Hypothermia in Trauma Patients

Unintended hypothermia in trauma victims remains a common problem, and occurs early during the resuscitative phase.¹⁵ We are probably still paying too little attention to this. Even basic interventions easily prevent hypothermia.¹⁵ Rewarming methods for the hypothermic trauma patient include both passive rewarming, active external rewarming, and active internal rewarming.^{3,6} Treatment of hypothermia in the trauma patient should begin with prevention of further heat loss. Fluid resuscitation can result in substantial core temperature decreases, mandating the use of efficient fluid-warming devices and prewarmed fluids. Of the various noninvasive treatment modalities, convective warming is effective in restoring heat to the core,⁶ although radiant heat may be easier to apply to the multiply injured trauma patient. Active core rewarming techniques such as continuous arteriovenous rewarming (CAVR) increase core temperature by 1.5–2.5°C per hour, and can be life-saving in the hypothermic trauma patient with adequate perfusing rhythm.¹⁶ CAVR uses percutaneously placed femoral arterial and venous catheters and the patient’s own blood pressure to create an arteriovenous fistula that diverts a portion of the cardiac output through a commercially heparin-bonded heat exchanger and tubing (SIMS Level 1, Rockland, Massachusetts). Systemic heparinization is not required.

The Role of Therapeutic Hypothermia in Trauma Patients

Hypothermia may prevent the initiation of the cascade of events after injury that leads to cell death. Further, hypothermia may be protective by decreasing oxygen consumption. Still, the effect of prolonged hypothermia during resuscitation after hemorrhagic shock is as yet unclear.¹⁷ Therefore, current accepted practice, both in blunt and penetrating injury, is to stop the bleeding and resuscitate with fluids, while keeping the patient as close to normothermia as possible.

Studies have found mild hypothermia to be protective in anoxic brain injury following resuscitation from prehospital cardiac arrest.^{18,19} The Advanced Life Support Task Force of the International Liaison Committee of Resuscitation (ILCOR) now

recommends that unconscious adults with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32–34 °C for 12–24 hours when the initial rhythm was ventricular fibrillation.²⁰ Studies have also focused on the therapeutic use of mild hypothermia in traumatic head injury and spinal cord ischemia.^{21–28} The role of therapeutic hypothermia in traumatic brain injury is still debated. Possible reasons for the conflicting results include method issues such as excluding patients with hypoxia or hypotension after resuscitation, timing of the hypothermic intervention, and duration of therapeutic hypothermia.^{21–28} It is also possible that longer periods of hypothermia (>48 hours) are needed, especially in patients with intracranial hypertension, defined as an increase in intracranial pressure (ICP) more than 25 mm Hg.^{26–28} In his excellent review of the potential for therapeutic hypothermia in different kinds of cerebral injury, Polderman^{27,28} concluded that the successful application of therapeutic hypothermia in traumatic brain injury depends on the use in carefully selected patients (those with increased ICP), strict protocols, and close monitoring to avoid complications such as hypovolemia, hypotension, and hyperglycemia. Further, he emphasized that hemodynamically stable, brain-injured patients already mildly hypothermic at admission should not be immediately rewarmed. Finally, after prolonged periods of cooling, rewarming must be slow and controlled.^{27,28}

Summary

Hypothermia often complicates the management of patients with severe blunt or penetrating injury, and has been associated with increased morbidity and mortality. Early control of bleeding and prevention of further heat loss are key factors to avoid the lethal triad of hypothermia, acidosis, and coagulopathy. Hence, hypothermia is still considered a foe in these patients. On the other hand, induced hypothermia may be beneficial (“a friend”) in selected patients with traumatic brain injury. Some centers have already brought the latter concept into clinical practice. Although more data are needed, we think the present evidence supports an aggressive approach to limit the burden of fever in these patients as well as inducing moderate hypothermia if intracranial hypertension remains a problem despite standard treatment.

References

- Jurkovich GJ, Greiser WB, Luterman A, Curreri PW. Hypothermia in trauma victims: an ominous predictor of survival. *J Trauma* 1987;27:1019-24.
- Luna GK, Maier RV, Pavlin EG, Anardi D, Copass MK, Oreskovich MR. Incidence and effect of hypothermia in seriously injured patients. *J Trauma* 1987;27:1014-8.
- Helm M, Lampl L, Hauke J, Bock KH. [Accidental hypothermia in trauma patients. Is it relevant to preclinical emergency treatment?] *Anaesthesist* 1995;44:101-7.
- Tsuei BJ, Kearney PA. Hypothermia in the trauma patient. *Injury* 2004;35:7-15.
- Gregory JS, Flancbaum L, Townsend MC, Cloutier TC, Jonasson O. Incidence and timing of hypothermia in trauma patients undergoing operations. *J Trauma* 1991;31:795-800.
- Sessler DI. Consequences and treatment of perioperative hypothermia. *Anesthesiol Clin North Am* 1994;12:425-56.
- Wong KC. Physiology and pharmacology of hypothermia. *West J Med* 1983;138:227-32.
- Greene PS, Cameron DE, Mohlala ML, et al. Systolic and diastolic left ventricular dysfunction due to mild hypothermia. *Circulation* 1980;80:44-8.
- Nicodemus HF, Chaney RD, Herold R. Hemodynamic effects of inotropes during hypothermia and rapid rewarming. *Crit Care Med* 1981;9:325-8.
- Oung CM, English M, Chiu RC, Hinchey EJ. Effects of hypothermia on hemodynamic responses to dopamine and dobutamine. *J Trauma* 1992;33:671-8.
- Frank SM, Fleisher LA, Breslow MJ, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events. A randomized clinical trial. *JAMA* 1997;277:1127-34.
- Kurz A, Sessler DI, Lenhardt R. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of Wound Infection and Temperature Group. *N Engl J Med* 1996;334:1209-15.
- Van den Berghe G, Wouters PJ, Bouillon R, et al. Outcome benefit of intensive insulin therapy in the critically ill: Insulin dose versus glycemic control. *Crit Care Med* 2003;31:359-66.
- Cosgriff N, Moore EE, Sauaia A, Kenny-Moynihan M, Burch JM, Galloway B. Predicting life-threatening coagulopathy in the massively transfused trauma patient: hypothermia and acidosis revisited. *J Trauma* 1997;42:857-62.
- Husum H, Olsen T, Murad M, Heng YV, Wisborg T, Gilbert M. Preventing post-injury, hypothermia during prolonged prehospital evacuation. *Prehosp Disast Med* 2002;17:23-6.
- Gentilello LM, Jurkovich GJ, Stark MS, Hassantash SA, O'Keefe GE. Is hypothermia in the victim of major trauma protective or harmful? A randomized, prospective study. *Ann Surg* 1997;226:439-49.
- Tisherman SA. Suspended animation for resuscitation from exsanguinating hemorrhage. *Crit Care Med* 2004;32(2 Suppl):S46-50.
- Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurological outcome after cardiac arrest. *N Engl J Med* 2002;346:549-56.
- Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med* 2002;346:557-63.
- Nolan JP, Morley PT, Hoek TL, et al. Therapeutic hypothermia after cardiac arrest. An advisory statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation. *Resuscitation* 2003;57:231-5.
- Marion DW, Penrod LE, Kelsey SF, et al. Treatment of traumatic brain injury with moderate hypothermia. *N Engl J Med* 1997;336:540-6.
- Shiozaki T, Kato A, Taneda M, et al. Little benefit from mild hypothermia therapy for severely head injured patients with low intracranial pressure. *J Neurosurg* 1999;91:185-91.
- Zhi D, Zhang S, Lin X. Study on therapeutic mechanism and clinical effect of mild hypothermia in patients with severe head injury. *Surg Neurol* 2003;59:381-5.
- Polderman KH, Tjong TJR, Peerdeman SM, Vandertop WP, Girbes AR. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. *Intensive Care Med* 2002;28:1563-73.
- Harris OA, Colford JM Jr, Good MC, Matz PG. The role of hypothermia in the management of severe brain injury: a meta-analysis. *Arch Neurol* 2002;59:1077-83.
- Bernard SA, Buist M. Induced hypothermia in critical care medicine: a review. *Crit Care Med* 2003;31:2041-51.
- Polderman KH. Application of therapeutic hypothermia in the ICU: opportunities and pitfalls of a promising treatment modality. Part 1: Indications and evidence. *Intensive Care Med* 2004;30:556-75.
- Polderman KH. Application of therapeutic hypothermia in the intensive care unit. Opportunities and pitfalls of a promising treatment modality—Part 2: Practical aspects and side effects. *Intensive Care Med* 2004;30:757-69.