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Hypothermia in Trauma Victims

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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 al.[2] found that almost every second patient was hypothermic. Entrapped patients were at higher risk (98 percent versus 35 percent; 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 percent. Admission hypothermia was independently associated with increased adjusted odds of death after major trauma in a study of Pennsylvania trauma centers (n = 38,520 patients).[3] Perioperative hypothermia occurred in almost 50 percent of trauma patients requiring early surgery [Figure 1 on page 18]. 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 surgery may suffer additional hypothermic insults from heat loss to the cold operating room. Administration of anesthetics 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
- Spinal cord injury
- Shock (tissue hypoperfusion)
- Extremes of age
- General and neuroaxial anesthesia
- Associated medical conditions such as diabetes and cardiac failure
- Drugs and substances such as alcohol and tricyclic antidepressants

Increased Heat Loss

- Exposure

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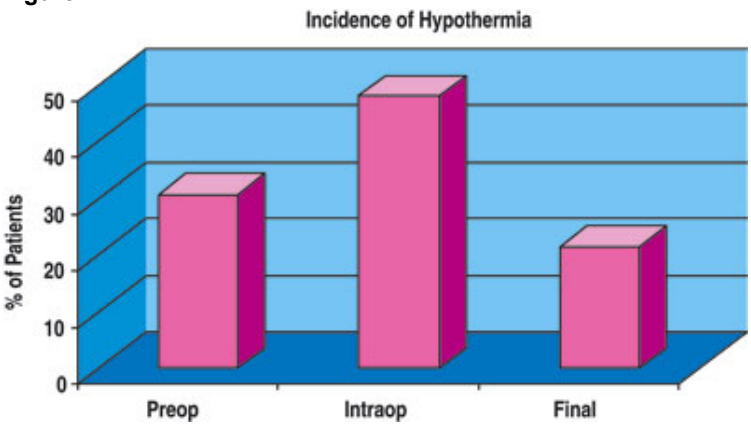
- Cold intravenous fluids and blood products
- Burns
- General and neuroaxial anesthesia

Figure 1



Incidence of hypothermia (<36ºC) in 660 trauma patients requiring surgery within 24 hours of admission at MetroHealth Medical Center, Cleveland, Ohio. Presented at MetroHealth Research Exposition and Ohio Society of Anesthesiologists Annual Meeting, September 2004.

Pathophysiological Consequences of Hypothermia

Hypothermia is associated with increased mortality and morbidity[1-4] with a decrease in survival at core temperatures below 34ºC. In trauma patients, the traditional severity classification of accidental hypothermia has been revised with 34-36ºC classified as mild, 32-34ºC as moderate and < 32ºC as severe hypothermia.[4] The increased morbidity and mortality is due to impaired coagulation, metabolic acidosis from poorly perfused tissues, hemodynamic instability, respiratory problems and infections. The adverse effects of hypothermia in the injured patient are shown in Table 2.[1-6] Hypothermia, together with acidosis and coagulopathy, has been identified as a component of the “lethal triad” in injured patients. Intense shivering may occur between 34ºC and 36ºC with resultant increased 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

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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
- 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
- Insulin resistance with hyperglycemia

Prevention and Treatment of Hypothermia in Trauma Patients

Nonintended hypothermia in trauma victims still is a common problem and occurs early during the resuscitative phase.[1-5] Even basic interventions such as warming the room (> 28ºC) can help prevent hypothermia.[8] Rewarming methods for the hypothermic trauma patient include both passive, active external and active internal rewarming.[3-6] Treatment of hypothermia in the trauma patient should begin with *prevention* of further heat loss.[9] Fluid resuscitation can result in substantial core temperature decreases, mandating use of efficient fluid-warming devices and

prewarmed fluids. Of the various noninvasive treatment modalities, convective (forced air) 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 arterial-venous rewarming (CAVR) increase core temperature by 1.5ºC to 2.5ºC/hour and can be life-saving in the hypothermic trauma patient with adequate perfusing rhythm.[10] In patients with arrested rhythms where cardiopulmonary bypass is not available or contraindicated, body cavity lavage with warmed fluids can increase core temperature by 1.5 to 2.0ºC/hour.

Role of Therapeutic Hypothermia in Trauma Patients

Hypothermia may prevent the initiation of the cascade of events after injury that leads to cell death.[11] Further, hypothermia may be protective by decreasing oxygen consumption. Still, the effect of prolonged hypothermia during resuscitation after hemorrhagic shock is as yet unclear.[12] 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.[13,14] The Advanced Life Support Task Force of the International Liaison Committee of Resuscitation now recommends that unconscious adults with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32-34oC for 12-24 hours when the initial rhythm was ventricular fibrillation.[15] Studies also have focused on the therapeutic use of mild hypothermia in traumatic head injury and spinal cord ischemia.[16-23] The role of therapeutic hypothermia (TH) in traumatic brain injury is still debated. Possible reasons for conflicting results include methodological issues such as excluding patients with hypoxia or hypotension after resuscitation, timing of the hypothermic intervention and duration of therapeutic hypothermia.[16-23] It also is possible that longer periods of hypothermia (> 48 hours) are needed, especially in patients with intracranial hypertension defined as an increase in intracranial pressure (ICP) > 25 mm Hg.[21-23] In his review of the potential for TH in different kinds of cerebral injury, Polderman[22,23] concluded that the successful application of TH in traumatic brain injury depends on its 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.[22,23]

Summary

Hypothermia often complicates the management of patients with blunt or penetrating trauma and has been associated with increased morbidity and mortality. Early control of bleeding and prevention of further heat loss are key factors in avoiding the lethal triad of hypothermia, acidosis and coagulopathy. On the other hand, induced hypothermia may be beneficial in selected patients with traumatic brain injury. Although more data are needed, we think the present evidence supports an aggressive approach to limit the burden of fever in head-injured patients, as well as inducing moderate hypothermia if intracranial hypertension remains a problem despite standard treatment.

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