Hypothermia and the trauma patient

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Hypothermia has profound effects on every system in the body, causing an overall slowing of enzymatic reactions and reduced metabolic requirements. Hypothermic, acutely injured patients with multisystem trauma have adverse outcomes when compared with normothermic control patients. Trauma patients are inherently predisposed to hypothermia from a variety of intrinsic and iatrogenic causes. Coagulation and cardiac sequelae are the most pertinent physiological concerns. Hypothermia and coagulopathy often mandate a simplified approach to complex surgical problems. A modification of traditional classification systems of hypothermia, applicable to trauma patients is suggested. There are few controlled investigations, but clinical opinion strongly supports the active prevention of hypothermia in the acutely traumatized patient. Preventive measures are simple and inexpensive, but the active reversal of hypothermia is much more complicated, often invasive and controversial. The ideal method of rewarming is unclear but must be individualized to the patient and is institution specific. An algorithm reflecting newer approaches to traumatic injury and technical advances in equipment and techniques is suggested.

Conversely, hypothermia has selected clinical benefits when appropriately used in cases of trauma. Severe hypothermia has allowed remarkable survivals in the course of accidental circulatory arrest. The selective application of mild hypothermia in severe traumatic brain injury is an area with promise. Deliberate circulatory arrest with hypothermic cerebral protection has also been used for seemingly unrepairable injuries and is the focus of ongoing research.

L'hypothermie a de profonds effets sur tous les systèmes du corps et provoque un ralentissement global des réactions enzymatiques et une baisse des besoins métaboliques. Des patients hypothermiques, qui ont subi une blessure grave et des traumatismes multisystémiques, se rétablissent moins bien que des patients témoins normothermiques. Les patients traumatisés ont une prédisposition inhérente à l'hypothermie causée par toutes sortes de facteurs intrinsèques et iatrogènes. Les séquelles touchant la coagulation et le système cardiaque constituent les préoccupations physiologiques les plus pertinentes. L'hypothermie et la coagulopathie obligent souvent à aborder des problèmes chirurgicaux complexes d'une façon simplifiée. On suggère une modification des systèmes traditionnels de classification de l'hypothermie qui s'appliquent aux patients traumatisés. Les études contrôlées sont peu nombreuses, mais les avis cliniques appuient fermement la prévention active de l'hypothermie chez le patient atteint de traumatisme aigu. Les mesures de prévention sont simples et peu coûteuses, mais l'inversion active de l'hypothermie est beaucoup plus compliquée, souvent effractive, et controversée. La façon idéale de réchauffer un sujet n'est pas claire, mais il faut la personnaliser et elle est particulière à l'établissement. On suggère un algorithme reflétant de nouvelles façons d'aborder les lésions traumatiques et de tenir compte des progrès techniques.

Par ailleurs, l'hypothermie présente certains avantages cliniques lorsqu'elle est utilisée comme il se doit dans des cas de traumatisme. Une hypothermie sévère a permis des survies remarquables dans des cas d'arrêt accidentel de la circulation. L'application sélective d'une hypothermie légère dans des cas de traumatismes graves au cerveau semble prometteuse. On a aussi arrête délibérément la circulation tout en protégeant le cerveau par hypothermie dans le cas de lésions qui semblaient irréparables. Des recherches en cours portent sur cette technique.

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Hypothermia is simply defined as a body temperature significantly below 37 °C.[1] It has a profound effect on every system of the body, akin to that of a double-edged sword, causing both reduced oxygen and metabolic demands as well as disruptions in temperature-dependent enzymatic processes.[2]

Hypothermia and trauma outcomes

In trauma, several retrospective studies have found an association between higher death rate and an increasing degree of hypothermia, even accounting for differences in the severity of injury.[3,4] Jurkovich and associates[3] found that no trauma patient whose core temperature fell below 32 °C survived, and they regarded this as the critical temperature for survival. Hypothermia is recognized as one pillar of a "lethal triad" (hypothermia, acidosis and coagulopathy) of homeostatic failure that is believed to mark the limits of ongoing intervention, and necessitates an "abbreviated" laparotomy.[5–9] The "damage control" approach evolved after surgeons noted a subset of trauma patients who no matter how aggressively they were resuscitated and supported became physiologically exhausted and intolerant of further surgical intervention. Procedures must be accomplished quickly without exhausting the patients' physiologic reserve through continued blood and heat loss.

Classification of hypothermia

In general, hypothermia is classified as mild (32° to 35 °C), moderate (28° to 32 °C) and severe (less than 28 °C).[10–16] The Committee on Trauma of the American College of Surgeons has focused on the immediate harmful effects of hypothermia in the post-injury period. The Committee recommended considering any temperature below 36 °C as hypothermic and 32 °C and below as severely hypothermic.[17] Further definitions of hypothermia arise from research exploring the potential benefits of hypothermia on human viability. Researchers have defined further levels of hypothermia: deep, between 10° and 20 °C; profound, less than 10 °C; and ultraprofound, from 0 to 5 °C.[18–21]

Current recommendations for an abbreviated operation and accepting a damage control approach recognize 34 °C as a critical decision-making temperature.[22,23] We believe that a modification to standard classifications of hypothermia is appropriate, to recognize the importance of temperature in trauma: a temperature of less than 34 °C should not be labelled "mild" in any multiply injured patient; a body temperature warmer than 34 °C (class I) should be distinguished from more worrisome hypothermia of 34° to 32 °C (class II). This distinction is not recognized by the usual systems. Classes III and IV should refer to the generally accepted criteria for moderate and severe hypothermia (Table I).

Table I

Proposed Classification of Hypothermia in the Trauma Patient

General medical classification		Proposed trauma classification	
Category	°C	Class	°C
Mild	35–32	I	36–35
		II	34–32
Moderate	32–28	III	32–28
Severe	28	IV	28

Controversies

The currently accepted standard of care is to actively prevent and treat hypothermia. The current advanced trauma life support (ATLS) guidelines from the Committee on Trauma of the American College of Surgeons stress temperature control with aggressive efforts to avoid and to treat hypothermia.[17] Whether the increased death rate in trauma patients is causally related to hypothermia or merely an associated factor marking progressive metabolic failure is unclear. Ronco and colleagues[24] have documented that the process of dying is marked by a progressive failure of oxygen consumption and metabolic activity. Steinmann and colleagues[11] found no difference in outcome in hypothermic trauma patients when they were stratified by injury severity. A small, randomized, prospective trial that actively warmed patients showed a significantly lower early death rate and decreased overall fluid requirements to meet standardized hemodynamic goals, although there was no overall difference in the survival rate to discharge.[25] This was the first and only randomized, prospective, controlled trial to assess the effect of hypothermia as an independent variable in traumatic injury.

Physicians have often been intrigued by the potential benefits of hypothermia in critical illness. Dramatic survivals have been recorded with prolonged immersion in cold water.[26] Concerning head trauma, Hippocrates himself stated that "a man will survive longer in winter than in summer, whatever be the part of the head in which the wound is situated."[27] In both the Second World War and war in Indochina, physicians believed that cooled patients would survive longer.[25] French military physicians in Indochina even actively cooled combat casualties.[28] Without large well-performed clinical trials to guide us, an appreciation of the effects of hypothermia in the trauma patient warrants further background study.

General physiologic consequences of hypothermia

Hypothermia exhibits an array of deleterious physiologic consequences if maximal physiologic performance in health is the comparative standard. The main consequences are summarized in Table II.[4,10,12,14,16,29–34] In general, the effect is of a continuous overall slowing and depression of the functions of life. The patient may be in a state of agonal pulselessness without brain stem and deep tendon reflexes, yet remain viable. This state may not be distinguishable from death by any means other than rewarming.

Changes in the cardiac and coagulation systems are particularly pertinent to a discussion of wounding and injury. The ventricle becomes irritable below 30 °C, with 25° to 28 °C quoted as the threshold temperature for ventricular fibrillation.[14,29,35] Ventricular fibrillation in the setting of hypothermia below 28 °C is often refractory to defibrillation and pharmacologic intervention until the core temperature is raised.[12,29] Tracheal and gastric intubation, cardiopulmonary resuscitation or even injudicious movement can induce ventricular fibrillation.[10] This hazard, along with the ever-present risk of exacerbating spinal cord injuries, warrants careful handling and instrumentation of the hypothermic trauma patient. Perioperatively, maintenance of normothermia decreased the rate of hemodynamically important arrhythmias compared with patients who were allowed to cool to a mean of 34.5 °C in a nontrauma setting.[36]

Many of the early deaths in hypothermic trauma patients are believed to be due to exacerbation of acquired coagulopathies.[15,37] Hypothermia, especially that associated with transfusion of large volumes of blood and fluids produces an acquired coagulopathy and exacerbates bleeding.[38,39] Retrospective studies have suggested that hypothermia exacerbates intraoperative blood loss independent of the degree of injury[40] and that significant bleeding develops despite adequate blood, plasma and platelet replacement.[5] Factors thought to contribute to impaired coagulation in the trauma patient include the consumption and dilution of platelets and clotting factors, activation of the fibrinolytic cascade, alterations in electrolytes such as calcium, endothelial injury, endotoxin and other vasoactive mediator release, acidosis and hypothermia.[30,38,40–42] Clearly, a true appreciation of the isolated effects of hypothermia itself is difficult. With hypothermia, both the extrinsic and intrinsic pathways become dysfunctional with a prolongation of the prothrombin time (or international normalized ratio) and partial thromboplastin time when measured at the in-vivo temperature.[41,42] The fact that these laboratory determinations are made after warming the sample to 37 °C may lead to falsely reassuring values.[41,42] Normal laboratory indices in an obviously coagulopathic patient, obtained from such an ex-vivo measure-

Table II

Deleterious General Physiologic Consequences of Hypothermia

Category	Consequence
Immune	Possible decreased chemotaxis, phagocytosis, antibody production and oxidative killing
Hematopoetic	Hemoconcentration
	Cold-induced granulocytopenia
	Disseminated intravascular coagulation
	Right oxyhemoglobin shift (increased affinity)
	Decreased red cell deformability (microcirculation)
	Increased blood viscosity
Cardiac	Reduced cardiac output
	Depressed contractility
	Arrhythmias
	Delayed conduction (prolongation of EKG intervals), J wave or "Osborne" wave considered pathognomonic of hypothermia
	Vasoconstriction
Respiratory	Falsely increased Pa_{O_2} (if not temperature corrected)
	Decreased respiratory rate
Renal	Decreased renal tubular function (cold diuresis)
Gastrointestinal	Elevated serum amylase level
Hepatic	Reduced hepatic function
Metabolic	Falsely decreased pH (if not temperature corrected)
	Decreased adrenal activity
	Decreased metabolism of lactate and citrate
	Hyperkalemia
	Delayed wound healing
Neurologic	Decreased consciousness progressing to coma
	Absent motor and reflex functions

ment suggest that the best therapy is active rewarming.[15,42] Animal studies have demonstrated that platelet dysfunction is reversible solely by rewarming.[43]

Particular susceptibility of the trauma patient

In general, extremes of age, environmental stresses, chronic illnesses (especially those with hypometabolic features), alcoholism and drug abuse, impaired neurologic state, sepsis, dermal compromise, impaired mobility, socioeconomic disadvantages and previous exposures all contribute to the likelihood of injury with cold exposure.[14,31,44–46] The elderly and infirm are particularly susceptible because they cannot increase heat production and decrease heat loss by vasoconstriction, and children are susceptible because of their increased body surface area-to-mass ratios and limited energy resources.[17] Urban hypothermia describes a subset of hypothermic victims who are commonly alcoholic, homeless, elderly or disabled.[12] Submersion imparts marked cooling stresses with the thermal conductivity of water being 32 times greater than that of air.[12,45]

Of all the critically ill patients that the surgical intensivist is required to treat, the severely traumatized patient may be the most prone to hypothermic complications. The patient may be admitted after prolonged exposure, especially if discovery or extrication is delayed or difficult.[10,45] Hypothermic stresses continue in the prehospital care phase and are often propagated throughout the early hospital phase. Standard ATLS guidelines warrant the infusion of 3 volumes of crystalloid fluids for every 1 volume of shed blood, often administered many degrees below that of body temperature. Banked blood being stored at 4 °C will act as a heat sink, actually absorbing heat from the patient at a time when the patient can least afford it.[32] Standard care also stresses complete exposure of the patient to avoid missing injuries. Coincident head injuries (typically present in 60% of severe blunt trauma in a Canadian setting),[47] well-meaning spinal precautions (restraints), and analgesia and sedation will prevent activity-induced thermogenesis. The frequent presence of alcohol and drugs can prevent appropriate conscious behavioural responses, cause hypothalamic dysfunction and blunt protective vasoconstrictive and shivering responses.[4,10,45] If a laparotomy is required, further heat losses may be extensive and impossible to reverse until the abdominal cavity is closed.[48] Anesthetic agents can promote further heat loss and impair thermoregulatory mechanisms.[37–39,49] Hypotension itself may reset the hypothalamic set-point for shivering, which can further compound these problems.[50,51]

Prevention and treatment of hypothermia

The prevention and treatment of hypothermia in the traumatized patient are effectively different points on the continuum of good patient care. It is crucial to accurately measure the body temperature. Special thermometers capable of reading low temperatures must be used, as standard thermometers may not have low enough values to accurately reflect the degree of hypothermia.[17] Rectal temperatures may lag behind actual core changes, especially if the rectum is loaded with cold stool.[10]

Prevention

Effective prevention is facilitated by attention to the etiologic factors that promote hypothermia in trauma patients. The victim should be initially removed or protected from environmental extremes. Passive warming should be applied at the scene, extrication and transportation times minimized, and resuscitative measures that create a negative heat flux such as the administration of cold fluids, should be either avoided or minimized. Once the patient is transported, one of the cornerstones of prevention and treatment of hypothermia has been the use of devices or practices to allow the use of heated resuscitation fluids.[37] Fluids may be successfully rewarmed by microwave techniques. Fluid warmers are usually more convenient and are generally of 2 types: dry-wall fluid warmers and water-bath fluid exchangers. Both types of devices will allow the infusion of large volumes (more than 3L/h) of heated intravenous fluids. The water-bath type heating systems have a wider range of effective flow rates.[52]

The lungs are a normal area of heat exchange and can simply and effectively be used to heat the victim with warm inspired gases. Humidification also increases the heat flux, besides being generally beneficial in liquidizing secretions and decreasing insensible fluid losses.[53] Inspired gas temperatures up to 44° to 46 °C have been used safely.[53–56] Ambient room temperature may need to be elevated even if this is uncomfortable for the medical staff.[57]

Treatment

To reverse hypothermia and its deleterious effects, a rewarming method should be implemented, guided by the degree of hypothermia and hemodynamic stability. Potential methods can be classified as passive external, active external or surface, and active internal or core rewarming (Table III[2,13,25,29,46,58]). Physiologically, categories of hypothermia severity reflect the degree of protective reflexes remaining. Mild cases reflect preserved heat production and compensatory reflexes. There is loss of heat production but preservation of vasoconstriction in moderate cases. In severe hypothermia both vasoconstrictive and endocrinologic reflexes are lost

and the human responds to hypothermia essentially as a poikilothermic being.[10] Hemodynamically stable, mildly hypothermic patients generate a positive heat flux and are suitable for passive rewarming techniques.[12,16,31,38] Essentially all trauma patients should be treated with measures that provide passive external rewarming (e.g., warm environment, covering). Patients who are moderately or severely hypothermic must be actively rewarmed.[10,31,59]

The optimal rewarming method for the trauma patient is not clear. The method used will be partly guided by the specific injuries, physiological features of the patient, the phase of care and the institutional resources available. For example, pleural lavage in the setting of severe chest injuries, or peritoneal lavage in severe abdominal trauma would not be appropriate in the initial resuscitation phases. During operative intervention though, warm mediastinal lavage during a thoracotomy or warm peritoneal irrigation during a laparotomy would be intuitive. Postoperatively, introduction of fluids into any thoracoabdominal compartment would be detrimental in patients at risk of an abdominal compartment syndrome. Many, if not most, polytraumatized patients are not candidates for systemic heparinization and therefore are not suitable for standard cardiopulmonary bypass.

Active external rewarming

Active external rewarming may increase peripheral cellular metabolic activity, especially if shivering occurs, diverting blood flow to the periphery and exacerbating hypoperfusion of the core.[31] The use of active external rewarming may also induce "rewarming shock" caused by peripheral vasodilatation when the periphery is rewarmed in the setting of an overall reduced intravascular volume.[38,53,54] Volume and circulatory status must therefore be closely monitored.[31,44,45] A further theoretical concern is premature inhibition of shivering by preferential warming of the skin and muscles since cold stress responses can be evoked by both central and peripheral thermoreceptors.[49,53,60] This may contribute to further cooling of the thermal core despite the initiation of peripheral warming, an effect known as "after-drop."[54]

Active internal (core) rewarming

Core rewarming takes advantage of the introduction of heat to large surface areas such as the lungs, peritoneum, pleural cavities or endothelium (vasculature). Invasive core rewarming has the potential advantage of rewarming the brain, heart, lungs and other core organs simultaneously so that, theoretically, perfusion should increase in concert with the increasing metabolic demands of the vital organs.[31] Besides the vascular and respiratory routes, heat can be transmitted through the peritoneal, pleural or pericardial cavities with dialysate warmed up to 45 °C.[53,61] Other potential routes of organ irrigation such as the bladder and colon may not be particularly effective owing to the limited surface area warmed.[53]

Extracorporeal blood rewarming

In accidental hypothermia, cardiopulmonary bypass has been recommended as the resuscitative method of choice for any severely hypothermic patient or any moderately hypothermic patient with hemodynamic insta-

Table III

Methods of Warming Hypothermic Patients

Type of warming	Method
Passive external	Warm environment
	Blankets
	Shivering
Active external	Immersion in warm water
	Electric blankets
	Environmental heaters
	Convective air blankets
Active core	Warm gastric lavage
	Warm pleural lavage
	Colonic lavage
	Peritoneal lavage
	Mediastinal lavage
	Inhalational gases
	Intravenous fluids
	Regional radiowaves
	Hemodialysis
	Extracorporeal circulation
Extracorporeal methods	Hemodialysis
	Centrifugal vortex pumps: venovenous circulation, arteriovenous circulation
	Standard cardiopulmonary bypass
	Continuous arteriovenous rewarming

bility.[16,29,61] Standard renal hemodialysis circuits with heated dialysate may also be used.[13] The requirement for systemic heparinization generally limits these strategies in multiply injured patients. Recent technical advances have brought a new generation of extracorporeal warming devices that may obviate the need for systemic heparinization.

Gentilello and associates[25] adopted a simple technique to provide extracorporeal blood warming using percutaneous femoral access, known as continuous arteriovenous rewarming. No pump is needed with this system as perfusion of the heparin-bonded heat exchanger is dependent on the patient's blood pressure. If the patient's hemodynamic status is suspect, extracorporeal pumps with heat exchangers may avoid the need for formal cardiopulmonary bypass. Gregory and colleagues[58] described the clinical use of an effective venovenous circulatory system that did not require heparinization, presumably due to coincident coagulopathies in their hypothermic trauma population. Animal studies have documented deaths due to in-situ thrombosis of the extracorporeal circulation; therefore, standard monitors such as foam, clot and low-pressure detectors should be used with this technique.[61] The development of centrifugal vortex bio-pumps that permit either partial cardiac bypass[62] or venovenous bypass[63] without heparinization will allow extracorporeal blood warming and cardiovascular support without systemic heparinization. If only warming not hemodynamic support is required, vascular access for the centrifugal vortex pump can be obtained percutaneously or through an open approach in both venous and arterial vessels. Further study will be required before widespread application can be recommended, but these techniques may bring extracorporeal strategies into the hands of a greater number of physicians.

Recommendations for treatment

Despite varying opinions, no differences in survival with the various treatment methods have been reported for hypothermic patients in general[31,55,64] or for hypothermic trauma patients.[25] As with so many other critical care situations, prevention of hypothermia is simpler and more effective than the treatment. Most of the preventive measures can be simply and cheaply instituted as part of a standard resuscitation. If faced with an unstable, moderately or severely hypothermic trauma patient, we would individualize the rewarming, based on the injuries present and body cavities involved. We would also critically evaluate what surgical interventions are urgently required and limit the duration of any operative procedure with a ready acceptance of an abbreviated, damage control approach. We would strongly advocate the avoidance of initiating or prolonging any surgical intervention in a class III or class IV hypothermic trauma patient. A suggested algorithm for our approach to the hypothermic trauma patient is given in Table IV.

Pronouncement of death

In general, no hypothermic patient should be pronounced dead without rewarming.[17] The axiom is that hypothermic patients should not be considered dead until they are "warm and dead." This has resulted in logistic strains beyond reason in many critical care settings. Exceptions to this axiom obviously include catastrophic injuries such as decapitations, significant anoxic

Table IV

Suggested Management of Hypothermia

	Hypothermia type/class			
	Mild		Moderate	Severe
Phase of care	Class I	Class II	Class III	Class IV
Prehospital/Emergency Department/Critical Care Unit	Standard measures ± active external warming	Active external warming	Extracorporeal measures	Extracorporeal measures
Intraoperative	Standard measures ± active external warming	Active internal warming (intracavitary irrigation)	Extracorporeal measures ± intracavitary methods	Extracorporeal measures ± intracavitary methods
Permissibility of further surgery?	Completion of definitive surgery	Damage control	Damage control	Damage control ? consider DHCA

Standard measures to be instituted in all serious trauma patients encompass but are not limited to measures recognized as passive external methods (warm environment, blankets, covers), warmed intravenous fluids, warmed inspired gases if intubated, convective warming blankets.
Extracorporeal methods to be utilized with appropriate personnel and institutional support: continuous arteriovenous rewarming, venovenous rewarming with centrifugal vortex pump, arteriovenous rewarming with centrifugal vortex pump, standard cardiopulmonary bypass, hemodialysis circuits with heated dialysate.
DHCA = deep hypothermic circulatory arrest (only with severe injuries and appropriate support).

events in normothermic patients who are subsequently without pulse or respiratory effort, and a serum potassium level greater than 10 mmol/L.[17,33,65]

Specific therapeutic applications of hypothermia in trauma

For select clinical settings, hypothermia may benefit the trauma patient and may be deliberately induced by caregivers. Resuscitative hypothermia constitutes post-insult therapeutic hypothermia and is distinguished from protective (pre-treatment) or preservation (intra-insult) hypothermia.[66] A promising area of current clinical application is the use of moderate hypothermia in traumatic brain injury. Some deeply hypothermic trauma patients have survived remarkably long periods of pulselessness when purposely not rewarmed before definitive control of the cardiorespiratory system with extracorporeal resuscitation. Large animal studies of novel methods of resuscitation suggest potential means of extending a patient's viability until emergency surgical interventions can be undertaken. It must be remembered that these are controversial or experimental uses of hypothermia used in select patients.

Hypothermia in traumatic brain injury

In 1993, 3 small preliminary studies documented decreased intracranial pressures (ICPs) and cerebral metabolic requirements, and found trends toward an improved neurologic outcome with cooling in severe traumatic brain injury.[67–69] These studies targeted patients with nonpenetrating brain injury and excluded those with major systemic trauma associated with hypotension or hypoxia.[67,68] They were followed by a larger study by Marion and colleagues from Pittsburgh.[70] This study showed improved outcomes in a group of severely brain injured patients with Glasgow Coma Scale (GCS) scores of 5 to 7 on admission, who were cooled to 33 °C within a mean of 10 hours after injury and were maintained at this temperature for 24 hours.[70] This treatment provided little benefit in patients with initial GCS scores of 3 or 4.[70] Seventy-three out of 155 patients with severe head injuries were excluded, including those with prolonged hypotension or hypoxia, who would presumably be those with the most severe extracranial systemic injuries. Although one of the early studies[67] suggested a nonsignificant trend toward increased sepsis in the hypothermia group, this trend was not replicated in the study of Marion and colleagues.[70]

Hypothermia has long been known to decrease cerebral blood flow by 6% to 7% per degree Celsius as well as to decrease brain volume, cerebral venous pressure and cerebrospinal fluid volume.[10,14,35,71] Although hypothermia is known to reduce the cerebral requirements for oxygen, the true benefits of hypothermia are likely more complicated.[66] Hypothermia may reduce the actual basal cerebral metabolic rate rather than just electrophysiologic activity.[70] The attenuation of brain injury is believed to be from reducing cerebral ischemia, edema and tissue injury, and in helping to preserve the blood–brain barrier, reducing extracellular excitatory neurotransmitters, or by suppressing the post-traumatic inflammatory response.[66,68,70] Significantly lower concentrations of the excitatory neurotransmitter glutamate and the cytokine interleukin-1β have been detected in the cerebrospinal fluid of cooled versus non-cooled patients having severe head injury.[68,70]

We currently use therapeutic hypothermia in the treatment of severe head injuries. This technique is not applied to patients until other systemic injuries are identified and managed, and hemodynamic stability is assured. Our severely head injured patients are treated in accordance with standard therapies, including the prompt surgical evacuation of intracranial hemorrhage, maintenance of cerebral perfusion as reflected by jugular bulb venous extractions (CvO_2ER), and following and treating increased ICPs with sedation, neuromuscular blocking agents, osmotic diuretics and cerebrospinal fluid drainage as appropriate.[72] All severely head-injured patients are considered potential candidates for resuscitative (post-insult) cooling in the early post-injury period when the patient's neurologic course is uncertain. If the patient's ICP and CvO_2ER are stable at 24 hours, hypothermia will be cautiously withdrawn. Cooling is instituted as long as there is difficulty in controlling ICP and maintaining an adequate CvO_2ER. Mild infections are not considered a contraindication to cooling, although the patient is warmed if there are concerns regarding systemic effects of infection. There is no arbitrary time to which the application of hypothermia is limited.

Utilizing resuscitative hypothermia in patients with traumatic brain injuries is an admittedly controversial clinical area. Animal models that have specifically included the secondary injury that frequently occurs clinically have failed to show a benefit of post-injury hypothermia.[73] Animal work also raises the possibility that post-insult hypothermia only delays rather than prevents the loss of selectively vulnerable neurons.[66,74] Some authors caution against the routine use of hypothermia for head injuries and are particularly concerned about core after-drop, cardiovascular instability, shivering and increased ICPs that may be seen upon rewarming.[34] Future clarification of the role of hypothermia from a randomized multicentre trial is anticipated.

Accidental deep hypothermia with circulatory arrest

Trauma patients who are deeply hypothermic and have no vital signs are a rare but fascinating subset for whom attempts at early prehospital rewarming may be counterproductive. If these same patients were normothermic or mildly hypothermic, no resuscitative efforts would be justified, recognizing the universally dismal results of out-of-hospital arrest in blunt trauma.

Walpoth and colleagues[75] have recently reported a remarkable series documenting 15 of 32 long-term survivors of accidental deep hypothermia who suffered prolonged circulatory arrest before rewarming. Nine of these 15 patients were environmentally exposed after traumatic injuries. Nine were without vital signs on discovery, and 6 suffered arrest a mean of 14 minutes into transport. The initiation of a perfusing circulation on cardiac bypass required an average time of 141 minutes. The patients were intubated and cardiopulmonary resuscitation was instituted, but no attempt at rewarming was made before the initiation of bypass. At long-term follow-up (mean 6.7 years), there were no hypothermia-related sequelae that impaired the quality of life of these patients, and all had resumed their former lifestyles. Based on these findings, the authors recommended against attempts at rewarming outside the hospital,[76] a departure from the standard recommendations.[29,31,77] The data are limited and patients in this subset are few. The high survival in a usually dismal situation is intriguing and deserves further evaluation.

Deep hypothermic circulatory arrest

Case reports are beginning to report the successful salvage of patients whose injuries were repaired in bloodless fields, with the patient protected by profound hypothermia. This technique may permit repair of what are considered to be technically irreparable injuries (in a perfused operative field), such as retrohepatic caval injuries due to either blunt or penetrating causes.[78,79] The patient's blood volume is effectively drained into the venous reservoir of the heart–lung machine to be reinfused once vascular integrity is re-established. At present, these techniques are only appropriate when there are no other extensive injuries (especially head injuries) that preclude systemic heparinization, and the appropriate personnel and cardiac bypass equipment are available and rapidly accessible.

Suspended animation

Controlled profound hypothermia as an adjunct to a state of suspended animation has recently been entertained as a realistic goal by reputable investigators.[18–21] Suspended animation has been defined as the protection and preservation of the whole organism during prolonged clinical death, for transport and repair (resuscitative surgery) without a pulse, followed by delayed resuscitation to complete recovery.[19] Dogs have survived normothermic hemorrhagic shock followed by up to 60 minutes of suspended animation with ultraprofound hypothermic circulatory arrest at 5 °C and have recovered completely with histologically "clean" brains.[80] This has required the use of extracorporeal circulation to induce and reverse the state of suspended animation, utilizing heparin-bonded circuitry, obviating the need for systemic heparinization.[21,81] It is certain that induction of suspended animation independent of cardiopulmonary bypass and extension of the tolerance of total circulatory arrest beyond 1 hour after normothermic shock will require further developments in basic and clinical science. With a committed and active scientific community currently exploring these issues, this approach may one day provide the ultimate damage control approach to allow evacuation and delayed surgical repair of otherwise lethal injuries.

At present, the technical limitations of quickly accessing the central circulation and instituting extracorporeal circulatory support with which to cool an injured patient are logistically limiting. Japanese investigators have developed a portable manual extracorporeal circulatory system, but this is in its technical infancy.[82] A more practical strategy may be that of emergency hypothermic aortic arch flushing.[65] This simpler approach has provided a remarkably normal neurologic outcome despite 15 minutes of exsanguinated cardiac arrest in large animal studies. The technique involves the early institution of mild cerebral hypothermia (35.8 °C) by a single flush of cooled saline administered through a catheter directed into the aortic arch through a femoral artery.[83] Such a technique might facilitate evacuation to a definitive care centre with a pulseless but potentially viable victim.

Conclusions

Much remains to be learned about hypothermia in the trauma patient. Currently accepted surgical principles mandate the avoidance of hypothermia, which is seen as a predictor of a poor prognosis. We believe that the acutely traumatized patient must be evacuated promptly and resuscitated aggressively. Simple measures of avoiding prolonged exposure, warming blood, administering warm fluids, applying warmed gases, and using convective air blankets *should* be standard protocol through all phases of care (prehospital, Emergency Department, operative and critical care phases). Hypothermia must be suspected, prevented and treated early if it occurs in the polytraumatized patient. A modification to the classification of hypother-

mia in trauma patients and a management approach to these patients is suggested. Treatment must be individualized, based on the patient's physiology and anatomy, the phase of care and the institution's resources. Ongoing developments may one day radically alter our perspectives allowing modifications to our approaches to the most severely injured patients.

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References

1. Stedman TL, editor. *Stedman's medical dictionary.* 24th ed. Baltimore: Williams & Wilkins; 1982.
2. Fischer RP, Souba WW, Ford EG. Temperature-associated injuries and syndromes. In: Felician DV, Moore EE, Mattox KL, editors. *Trauma.* 3rd ed. Stamford (Conn): Appleton & Lange; 1996. p. 951-2.
3. Jurkovich GJ, Greiser WB, Luterman A, Curreri PW. Hypothermia in trauma victims: an ominous predictor of survival. *J Trauma* 1987;27(9): 1019-24.
4. 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.
5. Ferrara A, MacArthur JD, Wright HK, Modlin IM, McMillen MA. Hypothermia and acidosis worsen coagulation in the patient requiring massive transfusion. *Am J Surg* 1990;160:515-8.
6. Stone H, Strom P, Mullins R. Management of the major coagulopathy with onset during laparotomy. *Ann Surg* 1983;197:532-5.
7. Burch JM, Ortiz VB, Richardson J, Martin RR, Mattox KL, Jordan GL. Abbreviated laparotomy and planned reoperation for critically ill patients. *Ann Surg* 1992;215:476-83.
8. Hirshberg A, Walden R. Damage control for abdominal trauma. *Surg Clin North Am* 1997;77:813-9.
9. Mattox KL. Introduction, background, and future projections of surgery. *Surg Clin North Am* 1997;77:753-9.
10. Best R, Syverud S, Nowak RM. Trauma and hypothermia. *Am J Emerg Med* 1985;3:48-55.
11. Steinmann S, Shackford SR, Davis JW. Implications of admission hypothermia in trauma patients. *J Trauma* 1990; 30:200-2.
12. Britt LD, Dascombe WH, Rodriguez A. New horizons in management of hypothermia and frostbite injury. *Surg Clin North Am* 1991;71:345-70.
13. Hernandez E, Praga M, Alcazar JM, Morales JM, Montejo JC, Jimenez MJ, et al. Hemodialysis for treatment of accidental hypothermia. *Nephron* 1993; 63:214-6.
14. Orkin FK. Physiologic disturbances associated with induced hypothermia. In: Gravenstein N, Kirby RR, editors. *Complications in anesthesiology.* Philadelphia: Lippincott-Raven; 1996. p. 131-40.
15. Cullinane DC, Bass JG, Nunn CR. Hypothermia: impact on the trauma victim. *Tenn Med* 1997;90:323-6.
16. Lazar HL. The treatment of hypothermia [editorial]. *N Engl J Med* 1997; 337:1545-7.
17. Committee on Trauma, American College of Surgeons. *Advanced trauma life support program for doctors. Instructor manual.* Chicago: American College of Surgeons; 1997.
18. Tisherman SA, Safar P, Radovsky A, Peitzman A, Marrone G, Kuboyama K, et al. Profound hypothermia (< 10 °C) compared with deep hypothermia (15 °C) improves neurologic outcome in dogs after two hours' circulatory arrest induced to enable resuscitative surgery. *J Trauma* 1991;31(8):1051-62.
19. Bellamy R, Safar P, Tisherman SA, Basford R, Bruttig SP, Capone A, et al. Suspended animation for delayed resuscitation. *Crit Care Med* 1996;24: S24-47.
20. Safar P. Novel ultra-advanced resuscitation methods — introduction of topic, speakers, problems, and potential solutions. In: *Critical Care Symposium — 1999. Technique & Technology.* Course syllabus. San Francisco: Society of Critical Care Medicine; January 23–27, 1999. p. 473-6.
21. Tisherman SA. Suspended animation research. In: *Critical Care Symposium — 1999. Technique & Technology.* Course syllabus. San Francisco: Society of Critical Care Medicine; January 23–27, 1999. p. 485-6.
22. Carrillo C, Fogler RJ, Shaftan GW. Delayed gastrointestinal reconstruction following massive abdominal trauma. *J Trauma* 1993;34(2):233-5.
23. Moore EE. Staged laparotomy for the hypothermia, acidosis, and coagulopathy syndrome. *Am J Surg* 1996;172: 405-10.
24. Ronco JJ, Fenwick JC, Tweedale MG, Wiggs BR, Phang PT, Cooper DJ, et al. Identification of the critical oxygen delivery for anaerobic metabolism in critically ill septic and non-septic humans. *JAMA* 1993;270:1724-30.
25. Gentilello LM, Jurkovich GJ, Stark MS, Hassantash SA, O'Keefe GE. Is hypothermia in the victim of major trauma protective or harmful? *Ann Surg* 1997;226:439-49.
26. Antretter H, Dapunt OE, Meuller LC. Survival after prolonged hypothermia [letter]. *N Engl J Med* 1994;330:219.
27. On injuries of the head. In: Adams F, editor. *The genuine works of Hippocrates.* Baltimore: Williams & Wilkins; 1939. p.145-160.
28. Chippaux C. Application of artificial hibernation to war surgery in Indochina. *Int Rec Med Gen Pract Clin* 1954;167:328-32.
29. Zell SC, Kurtz KJ. Severe exposure hypothermia: a resuscitation protocol. *Ann Emerg Med* 1985;14:339-45.
30. Wilson RF, Dulchavsky SA, Soullier G, Beckman B. Problems with 20 or more blood transfusions in 24 hours. *Am Surg* 1987;53:410-7.
31. Farmer JC. Temperature-related injuries. In: Civetta JM, Taylor RW, Kirby RR, editors. *Critical care.* 3rd ed. Philadelphia: Lippincott-Raven; 1997. p. 1451-62.
32. Collins JA. Problems associated with the massive transfusion of stored blood. *Surgery* 1974;75:274-95.
33. Schaller MD, Fischer AP, Perret CH. Hyperkalemia: a prognostic factor during acute severe hypothermia. *JAMA* 1990;264:1842-5.
34. Schubert A. Side effects of mild hy-

pothermia. *J Neurosurg Anesthesiol* 1995;7:139-47.
35. Sessler DI. Temperature monitoring. In: Miller RD, editor. *Anesthesia*. 4th ed. New York: Churchill Livingstone; 1994. p. 1363-82.
36. Frank SM, Fleisher LA, Brslow MJ, Higgins MS, Olson KF, Kelly S, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events. A randomized clinical trial. *JAMA* 1997;277(14):1127-34.
37. Gentilello LM, Moujaes S. Treatment of hypothermia in trauma victims: thermodynamic considerations. *J Intensive Care Med* 1995;10:5-14.
38. Patt A, McCroskey BL, Moore EE. Hypothermia-induced coagulopathies in trauma. *Surg Clin North Am* 1988; 68:775-85.
39. Mendez C, Jurkovich GJ. Blunt abdominal trauma. In: Cameron JL, editor. *Current surgical therapy*. 6th ed. St. Louis: Mosby; 1998. p. 928-33.
40. Bernabei AF, Levison MA, Bender JS. The effect of hypothermia and injury severity on blood loss during trauma laparotomy. *J Trauma* 1992;33:835-9.
41. Rohrer MJ, Natale AM. Effect of hypothermia on the coagulation cascade. *Crit Care Med* 1992;20:1402-5.
42. Gubler KD, Gentillelo LM, Hassantash SA, Maier RV. The impact of hypothermia on dilutional coagulopathy. *J Trauma* 1994;36:847-51.
43. Valeri CR, Cassidy G, Khuri S, Feingold H, Ragno G, Altschule MD. Hypothermia-induced reversible platelet dysfunction. *Ann Surg* 1987;205:175-81.
44. Ledingham IM, Mone JG. Treatment of accidental hypothermia: a prospective clinical study. *BMJ* 1980;280:1102-5.
45. Helm M, Hauke J, Lampl L, Bock KH. Accidental hypothermia in trauma patients. *Acta Anaesthesiol Scand Suppl* 1997;111:S44-6.
46. Bickel KD. Cold injury. In: Cameron JL, editor. *Current surgical therapy*. 6th ed. St. Louis: Mosby; 1998. p. 1018-20.
47. Hamilton SM, Breakey P. Fluid resuscitation of the trauma patient: How much is enough? *Can J Surg* 1996;39(1):11-6.
48. Burch JM, Denton JR, Noble RD. Physiologic rationale for abbreviated laparotomy. *Surg Clin North Am* 1997; 77:779-82.
49. Grahn D, Brock-Utne JG, Watenpaugh DE, Heller HC. Recovery from mild hypothermia can be accelerated by mechanically distending blood vessels in the hand. *J Appl Physiol* 1998; 85(5):1643-8.
50. Stoner HB. The impairment of thermoregulation by trauma. *Br J Exp Pathol* 1969;50:125-38.
51. Sori AJ, el-Assuooty A, Rush BF Jr, Engler P. The effect of temperature on survival in hemorrhagic shock. *Am Surg* 1987;53(12):706-10.
52. Block EFJ, Cheatham ML, Safcsak K, Nelson LD. Dry-wall fluid warmers fail to heat crystalloid fluids at low to moderate infusion rates [abstract]. *Crit Care Med* 1999;27:S172.
53. Sedlak SK. Hypothermia in trauma: the nurse's role in recognition, prevention, and management. *Int J Trauma Nurs* 1995;1:199-226.
54. Hayward JS, Steinman AM. Accidental hypothermia: an experimental study of inhalational rewarming. *Aviat Space Environ Med* 1975;46(10):1236-40.
55. Harnett RM, Pruitt JR, Sias FR. A review of the literature concerning resuscitation from hypothermia: part II — selected rewarming protocols. *Aviat Space Environ Med* 1983;54(6):487-95.
56. Weinberg AD. The role of inhalation rewarming in the early management of hypothermia. *Resuscitation* 1998;36:101-4.
57. Martin RR, Byrne M. Postoperative care and complications of surgery. *Surg Clin North Am* 1997;77:929-42.
58. Gregory JS, Bergstein JM, Aprahamian C, Wittmann DH, Quebbeman EJ. Comparison of three methods of rewarming from hypothermia: advantages of extracorporeal blood warming. *J Trauma* 1991;31:1247-52.
59. Josephs JD, Farmer JC. Hypothermia and extracorporeal rewarming: the journey towards a less invasive, more invasive methodology. *Crit Care Med* 1998;26:1944-5.
60. Terndrup TE. An appraisal of temperature assessment by infrared emission detection tympanic thermometry. *Ann Emerg Med* 1992;21:1483-92.
61. Seigler RS, Golding E, Blackhurst DW. Continuous venovenous rewarming: results from a juvenile animal model. *Crit Care Med* 1998;26:2016-20.
62. Grosso MA, Brown JM, Moore EE, Moore FA. Repair of the torn descending thoracic aorta using the centrifugal pump with partial left heart bypass: technical note. *J Trauma* 1991;31:395-400.
63. Baumgartner F, Scudamore C, Nair C, Karusseit O, Hemming A. Venovenous bypass for major hepatic and caval trauma. *J Trauma* 1995;39:671-3.
64. Giesbrecht GG, Paton B. Review article on inhalational rewarming [letter]. *Resuscitation* 1998;38:59-60.
65. Antretter H, Dapunt OE, Mueller LC. Portable cardiopulmonary bypass: resuscitation from prolonged ice-water submersion and asystole [letter]. *Ann Thorac Surg* 1994;58:1786-7.
66. Marion DW, Leonov Y, Ginsberg M, Katz LM, Kochanek PM, Lechleuthner A, et al. Resuscitative hypothermia. *Crit Care Med* 1996;24(2 Suppl):S81-9.
67. Clifton GL, Allen S, Barrodale P, Plenger P, Berry J, Koch S, et al. A phase II study of moderate hypothermia in severe brain injury. *J Neurotrauma* 1993;10:263-71.
68. Marion DW, Obrist WD, Carlier PM, Penrod LE, Darby JM. The use of moderate therapeutic hypothermia for patients with severe head injuries: a preliminary report. *J Neurosurg* 1993; 79:354-62.
69. Shiozaki T, Sugimoto H, Taneda M, Yoshida H, Iwai A, Yoshioka T, et al. Effect of mild hypothermia on uncontrollable intracranial hypertension after severe head injury. *J Neurosurg* 1993; 79:363-8.
70. Marion DW, Penrod LE, Kelsey SF, Obrist WD, Kochanek PM, Palmer AM, et al. Treatment of traumatic brain injury with moderate hypothermia. *N Engl J Med* 1997;335:540-6.
71. Rosomoff HL, Gilbert R. Brain volume and cerebrospinal fluid pressure during hypothermia. *Am J Physiol* 1955;183:19-22.
72. Bullock R, Chestnut RM, Clifton G, Ghajar J, Marion DW, Narayan RK, et al. Guidelines for the management of severe head injury. *J Neurotrauma* 1996; 13:639-734.
73. Robertson CL, Clark R, Dixon CE, Graham S, Alexander H, Wisniewski S, et al. No long-term benefit from hypothermia after severe traumatic brain injury with secondary hypoxemia in

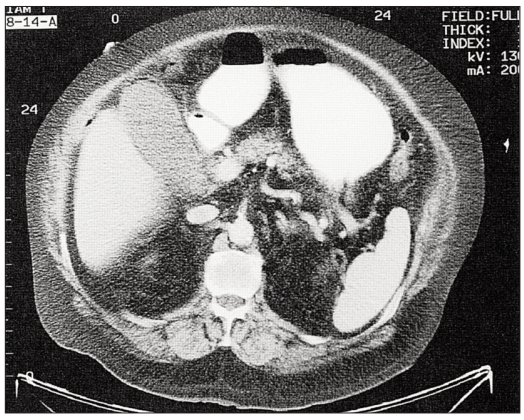
rats [abstract]. *Crit Care Med* 1999; 27:S52.
74. Dietrich WD, Busto R, Alonso O, Globus MY, Ginsberg MD. Intraischemic but not postischemic brain hypothermia protects chronically following global ischemia in rats. *J Cereb Blood Flow Metab* 1993;13:541-9.
75. Walpoth BH, Walpoth-Aslan BN, Mattle HP, Radanov BP, Schroth GS, Schaeffler L, et al. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. *N Engl J Med* 1997;337:1500-5.
76. Walpoth BH, Mattle HP, Althaus U. Accidental deep hypothermia [letter]. *N Engl J Med* 1998;338:1161.
77. Grant P, Snadden D, Syme D, Walker T. Freezing to death — the treatment of accidental hypothermia in the Scottish mountains. *Scot Med J* 1998;43:36-7.
78. Hartman AR, Yunis J, Frei LW, Pinard BE. Profound hypothermic circulatory arrest for the management of a penetrating retrohepatic venous injury: case report. *J Trauma* 1991;31:1310-1.
79. Marelli D, Tchervenkov CI, Metrakos P, Barayan S, Haesel R, Brown RA. Deep hypothermic circulatory arrest for blunt retrohepatic venous injury: a case report. *J Trauma* 1995;38:609-11.
80. Capone A, Safar P, Radovsky A, Yuanfan W, Peitzman A, Tisherman SA. Complete recovery after normothermic hemorrhagic shock and profound hypothermic arrest of 60 minutes in dogs. *J Trauma* 1996;40:388-95.
81. Tisherman T, Safar P, Radovsky A, Kuboyama K, Marrone G, Peitzman A. Cardiopulmonary bypass without anticoagulation for therapeutic hypothermic arrest during hemorrhagic shock in dogs [abstract]. *Crit Care Med* 1992;4:S41.
82. Terasaki H. Emergency (portable) cardiopulmonary bypass for resuscitation. In: *Critical Care Symposium — 1999. Technique & Technology*. Course syllabus. San Francisco: Society of Critical Care Medicine; January 23–27, 1999. p. 481-3.
83. Pruecker S, Woods RJ, Safar P, Takasu A, Stezoski SW, Radovsky A, et al. Mild normothermic aortic arch flush improves neurologic outcome from exsanguination cardiac arrest in dogs [abstract]. *Crit Care Med* 1999;27:S105.

Section Editor's comments: Hypothermia in trauma patients continues to be a significant ongoing problem with most cases being iatrogenic. In the acute situation, there always seem to be more pressing, and sometimes overwhelming, issues to deal with than remembering to keep the patient warm and covered. By repeatedly bringing up this issue, we will remind ourselves to give it the priority it deserves.

SESAP Question / Question SESAP

Category 4 Item 10

A 72-year-old man with insulin-dependent diabetes mellitus has epigastric pain and moderate upper abdominal tenderness with nausea and vomiting. The computed tomographic (CT) scan shown is obtained.



The most appropriate management includes intravenous hydration and

(A) bowel rest
(B) systemic antibiotics and observation
(C) systemic antibiotics and urgent operation
(D) systemic antibiotics and operation in 48 to 72 hours
(E) systemic antibiotics and elective esophagogastroduonenoscopy.

For the incomplete statement above select the one answer that is best of the five given.
For the critique of this item see page 370.