



The impact of hypothermia on trauma care at the 31st combat support hospital

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Abstract

Background: The primary objective of this study was to review the incidence of hypothermia, and its effect on surgical management, resource utilization, and survival at the 31st Combat Support Hospital (CSH).

Methods: This study was a retrospective analysis of all combat trauma injuries treated at the 31st CSH over a 12-month period. All trauma admissions were included. Descriptive and inferential analysis were performed using SPSS 11.0 software package (SPSS Inc., Chicago, IL).

Results: A cohort of 2848 patients was identified; 18% were hypothermic (temperature $<36^{\circ}\text{C}$). Hypothermia was significantly ($P < .05$) correlated with admission Glasgow Coma Scale (GCS), tachycardia, hypotension, lower hematocrit, and acidosis. Hypothermic patients had a significantly higher blood product and factor VIIa requirement. Hypothermia was an independent predictor of operative management of injuries, damage control laparotomy, factor VIIa use, and overall mortality ($P < .05$).

Conclusion: Combat trauma patients have a high percentage of penetrating injuries with variable evacuation times. Hypothermia was a pre-hospital physiologic marker, and independent contributor to overall mortality. Prevention of hypothermia could reduce resource utilization and improve survival in the combat setting. © 2006 Excerpta Medica Inc. All rights reserved.

Keywords: Hypothermia; Trauma; Military; Battlefield; Outcomes

Hypothermia dramatically impacts the outcome of trauma patients, and has been linked to systemic shock, volume of resuscitation, and severity of the injuries. The critical temperature of 32°C results in 100% mortality independent of injuries [1]. This is in contrast to accidental hypothermia (not trauma-associated) where severe hypothermia is defined as temperature less than 28°C , which is associated with a mortality rate as low as 10% [2]. In trauma patients, milder degrees of hypothermia result in devastating consequences; therefore, a separate classification scheme has been defined: mild hypothermia ($34\text{--}36^{\circ}\text{C}$), moderate hypothermia ($32\text{--}34^{\circ}\text{C}$), and severe hypothermia ($<32^{\circ}\text{C}$) [3].

As hemorrhagic shock overwhelms the body's compensatory responses, inadequate oxygen delivery results in anaerobic metabolism. The acidosis leads to coagulopathy that

is further compounded by the effects of hypothermia. The "deadly triad," hypothermia, acidosis, coagulopathy, has been feared by surgeons as the mark of a patient that has exhausted his or her physiologic reserve [4,5].

The civilian traumatized patient is susceptible to hypothermia from environmental exposures, extrication and transport, polytrauma, intoxication, and extremes of age [6,7]. In the combat arena, patients tend to be primarily young males who sustain multiple penetrating injuries, and the time for extrication and evacuation may be situation-dependent. This report will review the surgeons' experience at the 31st Combat Support Hospital (CSH) with respect to hypothermia and its impact on surgical management, resource utilization, and survival of combat casualties.

Methods

This is a retrospective review of the 31st CSH Institutional Review Board-approved trauma database. Data were

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Table 1
Univariate analysis among hypothermia subcategories

Variable	Normothermic (T >36°C)	Mild hypothermia (T = 34–36°C)	Moderate-severe hypothermia (T <34°C)	P*	r†
Air evacuation	495/528 (94%)	81/89 (91%)	26/26 (100%)	.594	—
Mechanism				<.01	.092
Penetrating	1783/2328 (77)	391/446 (88)	54/63 (86)		
Blunt	262/2328 (11)	22/446 (5)	6/63 (10)		
Other	279/2328 (12)	31/446 (7)	2/63 (4)		
Operative management	1463/2315 (64)	364/445 (82)	50/63 (80)	<.01	.149
Laparotomy	174/1686 (10)	70/377 (19)	14/50 (28)	<.01	.187
DCS at FST	25/382 (9)	7/57 (12)	7/57 (12)	<.01	.171
DCS at CSH	28/1811 (2)	19/396 (5)	10/57 (18)	<.01	.133
Factor VIIa used	13/2259 (1)	23/440 (5)	3/59 (5)	<.01	.152
Mortality	46/2334 (2)	28/447 (6)	17/62 (27)	<.01	.160

T = temperature; MVC = motor vehicle crash; DCS = damage control laparotomy; FST = forward surgical team; CSH = combat surgical hospital.

* Pearson chi-square test with significance $P < .05$.

† Spearman correlation coefficients.

collected from January 1, 2004 to December 20, 2004. Inclusion criteria were patients with an initial temperature recording on arrival at the CSH. This subset of patients was queried for all data fields to include care delivered at lower echelons of care, evacuation method, age, nationality, mechanism of injury, vital signs, laboratory data, operative management, blood product requirement, factor VIIa use, blood loss, intensive care unit (ICU) days, Injury Severity Score (ISS), and mortality.

Descriptive and inferential statistics were performed using the SPSS 11.0 Statistical Software Package (SPSS, Inc., Chicago, IL). Nonparametric variables were subjected to chi-square analysis and Pearson's correlations. Parametric

variables of grouped data underwent analysis of variance with post-hoc Tukey testing for specific significance between groups. Differences that were found to be significant were then subjected to stepwise logistic regression for dichotomous dependent variables. Significance was set at $P < .05$.

Results

Our study population consisted of 2848 trauma admissions that had an initial temperature recording among the 3387 total patients. The mean age was 28 ± 10 years, and 97% (2762) were male. Sixty-five percent (1851) were US/Coalition

Table 2
Univariate analysis of parametric variables among hypothermia subcategories

Variable	Normothermic (T >36°C)	Mild hypothermia (T = 34–36°C)	Moderate-severe hypothermia (T <34°C)	Significance within groups (P)	Correlation (r)
FST					
Operative time (min)	125 (± 58)	125 (± 25)	93 (± 68)	.550	—
Time to evacuation (min)	308 (± 272)	201 (± 39)	204 (± 82)	.151	—
Arrival vitals/labs					
SBP (mm Hg)	128.5 (± 18.4)*	122.4 (± 25.5)*	113.1 (± 30.6)*	<.01	-.145
HR	91 (± 21)	93 (± 25)	99 (± 25)*	<.01	.057
GCS	13.9 (± 3.1)*	12.6 (± 4.4)*	7.7 (± 5.6)*	<.01	-.234
HCT (mg/dL)	39.5 (± 7.6)*	36.7 (± 8.5)*	31.5 (± 9)*	<.01	-.193
pH	7.34 (± 0.1)*	7.3 (± 0.1)*	7.2 (± 0.2)*	<.01	-.280
BD	4.5 (± 3.8)*	6.3 (± 5.2)*	10.4 (± 7.3)*	<.01	.288
Perioperative data					
EBL (mL)	370 (± 910)*	806 (± 1206)*	1317 (± 2581)*	<.01	.215
PRBC (units)	4.8 (± 5)	6.5 (± 5)	9.6 (± 9)*	<.01	.227
Whole blood (units)	5.6 (± 5)	5.5 (± 5)	3.8 (± 2)	.490	-.106
FFP (units)	4.9 (± 5)	5.5 (± 4)	6.4 (± 4)	.214	.105
Operative time (min)	133 (± 89)*	173 (± 94)	173 (± 86)	<.01	.173
Postoperative data					
ICU (days)	3.7 (± 5)	3.9 (± 5)	8.7 (± 11)*	<.01	.118
ISS	11.2 (± 9)*	18.2 (± 15)*	29.7 (± 15)*	<.01	.296

SBP = systolic blood pressure; HR = heart rate; GCS = Glasgow Coma Scale; HCT = hematocrit; BD = base deficit; EBL = estimated blood loss; ISS = Injury Severity Score.

* Group is significantly different ($P < .05$) than the other 2 groups by Tukey post hoc analysis.

Table 3
Independent predictors of hypothermia (temperature <36°C) on admission

Variables (significant)	Odds ratio (95% confidence interval)	P
Penetrating mechanism	2.1 (1.6–2.8)	<.01
GCS <8.0	3.4 (2.6–4.3)	<.01
Shock (SBP <90 mm Hg)	5.7 (4.0–8.0)	<.01

GCS = Glasgow Coma Score; SBP = systolic blood pressure.

forces, and the remainder were local nationals or opposition forces. Eighty percent (2279) suffered penetrating wounds; these included high-velocity gunshot wounds (43%) and blast fragmentation wounds (37%). Ten percent (284) sustained blunt trauma, and the remainder were the result of falls, crush injuries, assaults, and burns. The mean ISS was 14 (± 12), and the overall mortality for this cohort was 5% (163).

Within this cohort, 82% (2335) were normothermic (temperature >36°C), 16% (455) mildly hypothermic (temperature <36°C), 2% (57) moderately hypothermic (temperature <34°C), and 0.2% (5) severely hypothermic (temperature <32°C). Secondary to the low numbers within the severe hypothermia group, these patients were grouped as a moderate-severe hypothermia group (temperature <34°C). The groups were then subjected to univariate analysis. No difference was found in the degree of hypothermia and evacuation method (Table 1). There was a correlation ($r = .092$, $P < .01$) in mechanism of injury with worsening degree of hypothermia. Hypothermic patients tended to present with lower blood pressures, tachycardic, and lower scores on the Glasgow Coma Scale (GCS) (Table 2). Hypothermic patients had lower hematocrits, lower pH, and higher base deficits on arrival.

A patient that underwent a damage control laparotomy at the Forward Surgical Team (FST), a mobile surgical unit, and was then transferred to the CSH correlated with degree of hypothermia ($r = .171$, $P < .01$). Seven of the 49 patients with

Table 4
Independent predictors of operative outcomes

Outcome variable	Odds ratio (95% confidence interval)	P
Operative management		
Hypothermia (T <36°C)	4.4 (2.5–7.8)	<.01
Penetrating mechanism	2.2 (1.6–3.1)	<.01
Shock (SBP <90 mm Hg)	7.9 (1.9–31)	<.01
GCS <8	4.4 (1.8–11)	<.01
Damage control laparotomy		
Hypothermia	5.2 (3.0–8.9)	<.01
Penetrating mechanism	3.4 (1.1–10.8)	<.05
Shock	9.9 (5.6–17.5)	<.01
Blood loss >2 L	10.7 (5.8–20)	<.01
Factor VIIa use		
Hypothermia	3.4 (1.5–7.7)	<.05
Blood loss >2 L	32.8 (14–75)	<.01
Blast mechanism	2.8 (1.2–6.5)	<.05

Table 5
Independent predictors of mortality

Variables	Odds ratio (95% confidence interval)	P
Significant		
Hypothermia (T <36°C)	3.8 (2.1–6.9)	<.05
GCS <8.0	23 (14.8–35.9)	<.01
Shock (SBP <90 mm Hg)	6.1 (3.1–12.1)	<.01
ISS >25	13.9 (7.3–26)	<.01
Not significant		
Age	—	.705
Mechanism	—	.808
Evacuation method	—	.246
Heart rate	—	.613
Hematocrit	—	.870
Base deficit	—	.150
Quantity of blood products	—	.725
Operative procedure	—	.966

GCS = Glasgow Coma Score; SBP = systolic blood pressure; ISS = Injury Severity Score.

FST damage control laparotomies had an initial temperature recording on arrival at the FST and at the CSH. A paired-sample *t* test revealed no difference in temperature at the FST ($35.2 \pm 1.4^\circ\text{C}$) and at arrival to the CSH ($36.2 \pm 1.8^\circ\text{C}$), $P = .276$. In addition, there was no significant difference in the time spent in the operating room at the FST, nor was there a difference in times waiting for evacuation from FST among hypothermic groups on arrival to the CSH.

Hypothermia correlated with need for operative management, laparotomy, and damage control laparotomy at the CSH (Table 1) for treatment of injuries. Hypothermic patients had higher blood loss in the operative theater, as well as required higher packed red blood cell, whole red blood cell, and fresh frozen plasma transfusions (Table 2). Operative times were longer for the mild and moderately-severe hypothermia groups. Factor VIIa use also correlated with hypothermia. ISS had the highest correlation ($r = .296$, $P < .01$) of all variables. The hypothermic patients spent more days in the ICU, and their overall mortality was significantly higher than normothermic patients.

After univariate analysis, all variables were subjected to stepwise logistic regression analysis. Three pre-hospital variables—penetrating mechanism, GCS less than 8, and shock, were independently predictive of the patient arriving hypothermic to the CSH (Table 3). Specific independent predictors were identified for requiring operative management, requiring damage control laparotomy, and receiving factor VIIa (Table 4). Hypothermia proved to be a marker for all 3 events.

Hypothermia was also identified as a predictor of mortality in this cohort (Table 5). Shock, GCS <8, and ISS >25 also were found as independent predictors. Temperature correlated with both GCS and requiring craniotomy as the primary procedure; therefore, the logistic regression analysis were repeated excluding the patients that went to emergent craniotomy. After excluding craniotomies from the

analysis, the odds ratios were identical for hypothermia, shock, and ISS, and while the GCS was still significant, the odds ratio decreased from 23 to 13.

Comments

Results from this study demonstrate a hypothermia rate of 18% in a cohort of young males that primarily sustained penetrating trauma (80%). There were very few severely hypothermic patients ($n = 5$); the majority of patients were mildly hypothermic, 34–36°C (16%). The majority of patients were evacuated by air, which would decrease transport time, but also has the potential to expose the patient to an environment that would exacerbate hypothermia. In this series, evacuation did not correlate with hypothermia. There was a subset of patients who were received at the FST, received operative treatment, and were then transferred to the CSH. These patients did not become hypothermic, and their operative times and times until evacuation did not significantly correlate with hypothermia on arrival to the CSH. The patients who underwent damage control laparotomy at the FST correlated with hypothermia on admission to the CSH. However, the patients' temperatures at the FST were not significantly different from their temperatures on arrival at the CSH.

Hypothermia subgroups correlated with patients' presenting physiologic profiles. With worsening hypothermia, patients were more likely to be acidotic and in shock, and notably, they were more likely to have a lower GCS. The response of a patient in hemorrhagic shock can account for hypothermia and mental status changes, but head injuries could also exacerbate thermoregulatory dysfunction. Attempting to control for the confounding potential of head injuries, the cohort was analyzed excluding those patients that first underwent emergent craniotomy. GCS in the field still correlated with temperature on arrival as an independent predictor.

Once a hypothermic patient was received, he was more likely to require operative intervention and more specifically damage control laparotomy for management of his injuries. The hypothermic subgroup experienced longer operative times, and blood products administered were greater among hypothermic patients. Hypothermic patients accounted for 50% of the pack red blood cell, 56% of the fresh frozen plasma, 60% of the whole red blood, and 63% of the factor VII transfusions. In this study, hypothermia independently predicted the events of operative management, damage control, and need for factor VIIa.

Within this cohort, four variables were found to predict mortality: hypothermia, injury severity score, shock, and GCS less than 8. Each variable independently impacted the mortality rate. The critical temperature for trauma patients has been previously described as 32°C; this is significantly higher than that for accidental hypothermia, which is reported at 28°C. The compounding effects of hypothermia, shock, and injury severity have been described by Jurkovich

et al [1], and evaluating those 3 variables our mortality rate is similar to that reported in civilian trauma. When the patient also presents with a GCS less than 8, the mortality rate is again compounded at each incremental increase in temperature such that no patient survived with temperature less than 33°C. The patients with temperature greater than 33°C, shock, ISS less than 25, and GCS less than 8 had a 40% mortality rate.

In this cohort of primarily penetrating trauma in young patients, the patients who were in shock, had a lower GCS, higher ISS, and were also hypothermic had the worst prognosis. Using rapid rewarming techniques, mortality, fluid requirements, and morbidity can be altered in hypothermic patients [8]. The time and threshold at which to begin rewarming is not known, but the threshold should be lower for trauma patients who are hypotensive, with probable high injury severity, and low GCS on arrival. Continuous arteriovenous rewarming devices are now available for the theater. Their use has the potential to reduce blood product requirements in a combat setting and is currently being reviewed.

This study identified 3 pre-hospital markers as predictors of the patient arriving hypothermic: low GCS, penetrating trauma, and shock. These 3 variables are important for the pre-hospital provider to identify those patients who are at risk for hypothermia. The merits of prevention in the pre-hospital combat setting have not been studied, but combat medics now have mobile in-line fluid warming devices in the field. Commercial fluid warming devices are being evaluated for use in lower echelons of care to help prevent the development of hypothermia [9].

This study has the limitations of a retrospective review. All patients were included, but there is a potential subgroup of patients that could have been treated in the field or at a lower level of care and who died in route to the CSH. The study consists of a fairly homogenous cohort of penetrating trauma in young male patients, limiting the generalizations of these conclusions.

There are unmeasured events that may influence some of our conclusions. In order to protect patient confidentiality, the time of year to determine ambient temperatures could not be recorded as part of the database. Time of injury, time for extrication, and time to evacuation are not available in this database. There is also no information about patients' pre-hospital crystalloid or colloid resuscitations. Several patients arrived intubated and some patients were paralyzed in the field. We were unable to stratify the patients based on receipt of paralytic therapy in the pre-hospital phase, due to the frequent absence of these pre-hospital data. Hence, we could not account for these effects on the patients' thermoregulatory systems, potentially blocking compensatory mechanisms to hypothermia.

While coagulation profiles were performed, they were not available for this analysis. The thromboelastogram has become available in the operating theater for combat support hospitals to utilize since these data were collected. Their use may better guide correction of coagulopathy in

trauma patients [10]. This will enable a comparison of hypothermia with temperature-dependent coagulation analysis. There were only 5 patients with temperature less than 32 C; therefore, very few conclusions could be made for this population of patients. Unlike civilian medicine, resources are often limited in the combat arena. Blood products are especially limited; therefore, the surgeon may have given the patients larger blood product resuscitations had they been available at that time. Additionally, factor VIIa may have been available and given in situations when blood products were not readily available.

Conclusions

Results from this study demonstrate a dramatic impact of hypothermia on the combat trauma patient. Pre-hospital markers, GCS, shock, and penetrating trauma identify those patients at risk of being hypothermic on arrival. Once the trauma patient becomes hypothermic, he or she is more likely to require operative treatment of injuries and will require significant resources to maintain physiologic support. Paralleling civilian literature, hypothermia was an independent predictor of mortality. Prevention in the pre-hospital setting has implications for reduced resource utilization and possible improvement in mortality.

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Discussion

Hugh Foy (Seattle, WA): The authors describe their 1-year experience with 3387 trauma patients in Iraq. This represents more than 9 admissions per day, which is just about 60% of what we admit at Harborview. In stark contrast, however, the mechanism of injury in their patients was 80% penetrating and 10% blunt, which is a reciprocal of what we typically see in our civilian population in Seattle. A greater contrast resides in that these penetrating injuries were mostly high-velocity rifle injuries and blast fragmentation wounds, both rare in civilian trauma centers. So, we are wise to learn from their experience, as the tragic events in Oklahoma City, New York, Madrid, London, and just days ago in Jordan underscore the unfortunate reality that any of us might be called upon to treat similar patients with little advance notice.

Their paper focuses on the relationship between hypothermia and operative management, transfusion practice, and mortality. Elaborate statistical methods are employed in an attempt to help predict and, hence, improve treatment and better plan logistics. Patients received an average of 12 units of blood and had a 5% overall mortality. The average Injury Severity Score (ISS) was 14, which is low compared to unstable blunt trauma patients and illustrates the limitations of ISS when applied to penetrating and closed head injury. As ISS calculation is based on the most severe injury in each anatomic region, it underestimates the physiologic derangement typical of high-velocity penetrating torso and head injury. Did the authors consider using the “New ISS” or “NISS” as a better physiologic indicator?

The authors state “hypothermia correlated with requiring operative treatment, laparotomy, and damage control . . .” I would offer that hypothermia is surely associated with these factors, but likely is a result of the patients shock and, in and of itself, is not a contributing cause. Merely correcting the hypothermia is an adjunct to control of hemorrhage, adequate resuscitation, prevention of further heat loss, and rewarming. The authors clearly state that their resources were limited and that they recently deployed in-line rewarming devices to the field medics. It would be of interest to know whether rewarming was done in any of these patients.

Likely, some of the sickest patients were among the 49 who underwent damage-control laparotomy at the Forward Surgical Team. It is unfortunate that temperature data was only available in 7 of the 49 and that field IV fluid volumes are also unavailable. Understandably, there is often no time to collect additional data when faced with a critically injured patient. The use of factor VIIa in 39 patients is a significant experience. It was most often used in the mild hypothermic group. Our hematologists tell us that factor VIIa is ineffective in severe hypothermia and acidosis. With a price tag of \$3000 per dose, an analysis of its effectiveness would be helpful in this era of scarce resources.