Hypothermia in Trauma
Friend or Foe?

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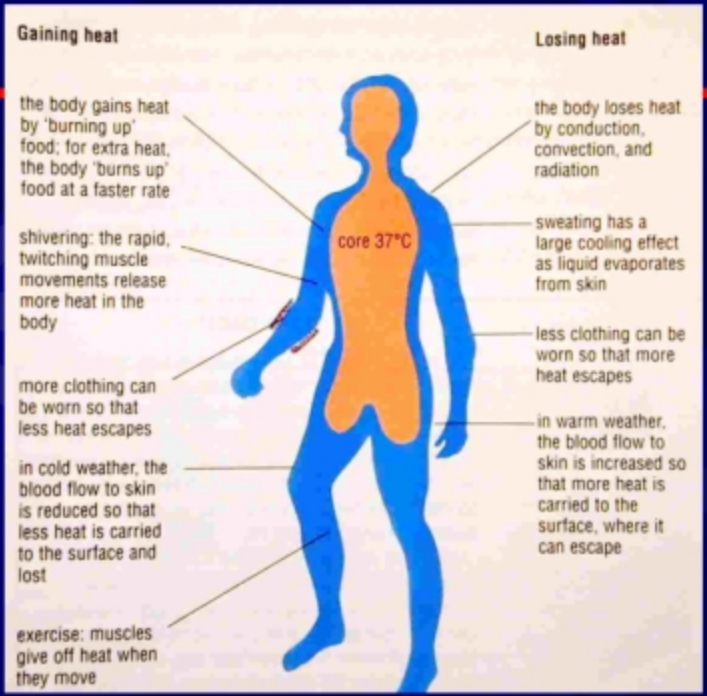
Is hypothermia beneficial or harmful?

- Ongoing controversy
  - Hypothermia can both prolong life, and contribute to death
  - Paucity of studies in trauma patients
    - Current management guidelines based on retrospective studies

Features of mammalian animals

- Warm-blooded
- Hair or fur, 2 pairs of limbs, feeds milk to young
  - **Homeotherms**
    - Active energy expenditure to maintain a constant internal temperature/environment
      - Many physiologic processes optimised to functions within a narrow temperature range
      - All enzymes are temperature-dependent
    - Allows adaptation to a wide range of climatic conditions

Earth by night
http://antwrp.gsfc.nasa.gov/apod/ap020810.html


Gaining heat
Losing heat
the body gains heat by 'burning up' food; for extra heat, the body 'burns up' food at a faster rate
shivering: the rapid, twitching muscle movements release more heat in the body
more clothing can be worn so that less heat escapes
in cold weather, the blood flow to skin is reduced so that less heat is carried to the surface and lost
exercise: muscles give off heat when they move
core 37°C
the body loses heat by conduction, convection, and radiation
sweating has a large cooling effect as liquid evaporates from skin
less clothing can be worn so that more heat escapes
in warm weather, the blood flow to skin is increased so that more heat is carried to the surface, where it can escape

- Heat Production
  - central control preoptic anterior hypothalamus
  - shivering
  - vasoconstriction
  - TSH and ACTH
- Heat Loss
  - convection (5x in 12 mph wind)
  - conduction (10x in wet clothing, 25x in water) [20%]*
  - radiation (head) [60%]*
  - evaporation [20%]*

*[%] normal loss at room temp

Hypothermia: definitions

- Hypothermia: Core temperature <35^{0}C
  - Mild
    - 35 – 32^{0}C
  - Moderate
    - 32 – 30^{0}C
  - Severe
    - <30^{0}C

For trauma patients, **T<32^{0}C** should be considered severe

Hypothermia and traumatic injury

- Components of the ‘lethal triad’
  - Hypothermia
  - Coagulopathy
  - Acidosis
  - A marker of the limits of physiological reserve
  - Concept of ‘damage control’ in trauma surgical management

Deleterious physiologic consequences of hypothermia

- stress response
- immune
  - possible decreased chemotaxis, phagocytosis, antibody production, and oxidative killing
- haemopoietic
  - haemoconcentration
  - cold induced granulocytopaenia
  - DIC
  - rightward oxyhemoglobin shift
  - decreased red cell deformability
  - increased blood viscosity
- cardiac
  - reduced cardiac output
  - depressed contractility
  - arrhythmias
  - delayed conduction
  - J wave or "Osborne" wave
  - vasoconstriction
- respiratory
  - falsely increased PaO_2
  - decreased respiratory rate
- renal
  - decreased renal tubular function (cold diuresis)
- gastrointestinal
  - elevated amylase
- hepatic
  - reduced hepatic function
- metabolic
  - falsely decreased pH (if not temperature corrected)
  - decreased adrenal activity
  - decreased metabolism of lactate and citrate
  - hyperkalaemia
  - delayed wound healing
- neurologic
  - decreased consciousness progressing to coma
  - absent motor and reflex functions

Hypothermia- pathophysiology

- Rate of heat loss a factor
- cardiac
  - initial increase CO then decrease, increase BP then decrease
  - EKG slowed depolarisation, arrhythmia, arrest
- vascular
  - impaired coagulation, DIC
- renal
  - diuresis, secondary to decreased resorption

Hypothermic coagulopathy

Watts et al, 1998

112 trauma patients: 40 normothermic
72 hypothermic (33-37^{0}C)

- thromboelastography
- PT, aPTT, platelets, CO_2, Hb, Hct, ISS

At T<34^{0}C: significant slowing of enzyme activity, platelet function
No effect on fibrinolysis

? Coagulopathy due to disruption of polymerisation process of platelets and fibrin

Hypothermia- pathophysiology

- pulmonary
  - depressed medullary respiratory centre
  - pulmonary oedema
- metabolic
  - slowed enzymatic reaction velocity--> pumps--> leaks--> lost gradients
  - hyperglycemia secondary to cold inactivation of insulin

Hypothermia- pathophysiology

- CNS
  - 3.3% decrease in cerebral blood flow per 0.5 degree drop
  - confusion-> decreased reflexes-> coma, fixed pupils, areflexia
- No One is Dead ……

Hypothermia and trauma

- Risk Factors
  - Environmental exposure
  - Extrication and transport time
  - Haemorrhage
  - Head injury
  - Drugs and alcohol

Hypothermia in the trauma victim: an ominous predictor of survival. Jurkovich et al, 1987

71 patients with ISS>25

mortality:

- T<34^{0}C: 40%
- T<33^{0}C: 69%
- T<32^{0}C: 100%

Controlled for ISS, BP, and fluid volume resuscitation, mortality significantly higher if patient also hypothermic

A temperature of 32^{0}C identified as being the critical temperature below which survival was zero

Incidence and effect of hypothermia in seriously injured patients. Luna et al, 1987

94 intubated trauma patients, average ISS 31

	normothermic	hypothermia	
		mild	severe
age (yr)	35	34	35
ISS	28	29	36
T (^{0}C)	36.9	35.1	32.2
survival (%)	78	59	41

Management of hypothermia

- ATLS/EMST guidelines
  - ABCD
  - E: Exposure and Environmental control
    - Prevent further heat loss
    - Initiate proactive measures for heat conservation

Modes of rewarming

- Passive
  - External
- Active
  - External
  - Internal

Continuous arteriovenous rewarming:experimental results and thermodynamic model simulation of treatment for hypothermia. Gentilello et al, 1990

Continuous arteriovenous rewarming (CAVR)

- Femoral arteriovenous bypass with percutaneous catheters placed in the groin of lab dogs
- Modified Level 1 rapid infuser with countercurrent heat exchanger
- No external pressure support
- No systemic heparinisation

Continuous arteriovenous rewarming:report of a new technique for treating hypothermia.

Gentilello and Rifley, 1991

Case report: 28 yr old multitrauma patient

Apnoeic pulseless GCS3 T31.5

Resuscitated, BP 80/- T29.5

CAVR via afferent subclavian vein, efferent femoral artery

Time to rewarm to T35, 85min

Spontaneous respirations, purposeful movt

Discharged independent and ambulatory

Is hypothermia in the victim of major trauma protective or harmful? Gentilello et al, 1997

Prospective randomised study

Patients: **57** hypothermic ($T_c \leq 34.5^0C$) trauma patients admitted to SICU and requiring PA catheter

Randomised to CAVR vs standard rewarming

Endpoints:
1. blood products and fluid requirements in first 24 hours
2. Coagulation and hemodynamics, LOS, mortality

Is hypothermia in the victim of major trauma protective or harmful? Gentilello et al, 1997

Table 1. DEMOGRAPHIC INFORMATION*

	SR	CAVR	p Value
n	28	29	—
Age (yr)	45.6	47.5	0.85
ED temperature (C)	33.92 (±1.4)	34.05 (±1.5)	0.76
ICU temperature (C)	33.3 (±1.3)	33.6 (±1.1)	0.10
Injury Severity Score	32 (±8.3)	31 (±9.3)	0.39
Gender (male)	18 (64%)	16 (55%)	0.48
Severe head injury	15 (52%)	15 (52%)	0.89
Severe chest injury	20 (71%)	17 (59%)	0.31
Severe abdominal injury	10 (36%)	10 (35%)	0.92
Severe extremity injury	16 (57%)	19 (66%)	0.52
Laparotomy	10 (36%)	20 (69%)	0.01
Systolic blood pressure at start of warming (mmHg)	112 (±43)	120 (±28)	0.18
Blunt mechanism (%)	86	83	0.75

SR = standard rewarming; CAVR = continuous arteriovenous rewarming.
* Severe injuries were those classified as having Abbreviated Injury Severity Score ≥3. Pearson chi square for categorical data and Mann-Whitney U test for continuous data.

Is hypothermia in the victim of major trauma protective or harmful? Gentilello et al, 1997

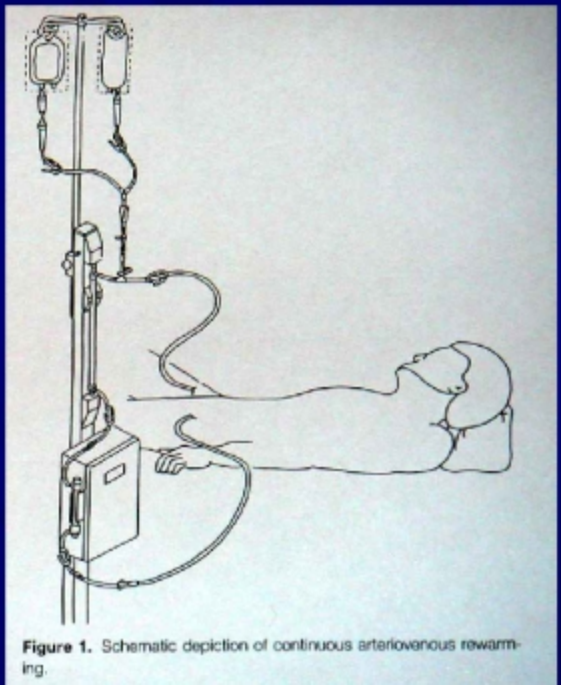


Figure 1. Schematic depiction of continuous arteriovenous rewarming.

Is hypothermia in the victim of major trauma protective or harmful? Gentilello et al, 1997

Table 2. VOLUME REQUIREMENTS

	SR	CAVR	p Value
Crystalloid 0–8 hr	9844 (±7039)	6289 (±4623)	0.04
Crystalloid 0–24 hr	23,916 (±14,446)	17,872 (±14,734)	0.08
Red blood cells 0–8 hr	5341 (±4824)	4179 (±4052)	0.34
Red blood cells 0–24 hr	6002 (±4935)	4941 (±4293)	0.48
FFP 0–8 hr	1412 (±1291)	1123 (±1507)	0.44
FFP 0–24 hr	2120 (±1622)	1669 (±1598)	0.23
Cryoprecipitate 0–8 hr	208 (±205)	139 (±194)	0.27
Cryoprecipitate 0–24 hr	387 (±419)	280 (±400)	0.16
Platelets 0–8 hr	449 (±514)	353 (±398)	0.49
Platelets 0–24 hr	535 (±537)	392 (±401)	0.32
Total volume 0–8 hr	12,548 (±9641)	8380 (±8391)	0.06
Total volume 0–24 hr	32,960 (±20,607)	25,154 (±20,723)	0.05

FFP = fresh frozen plasma; SR = standard rewarming; CAVR = continuous arteriovenous rewarming.

Is hypothermia in the victim of major trauma protective or harmful? Gentilello et al, 1997

Table 3. OUTCOMES*

	SR	CAVR	p Value
Time to 35 C (hr)	2.4 (±1.3)	1.4 (±1.0)	0.003
Time to 36 C (hr)	4.2 (±2.4)	2.3 (±2.5)	0.002
Survival to discharge	14 (50%)	19 (66%)	0.24
Hospital stay (days) (median)	7.5 (±17.1)	25 (±18.9)	0.018
Mechanical ventilation (days) (median)	4.5 (±10.3)	6.0 (±16.4)	0.031
ARDS	3 (11%)	9 (31%)	0.06
Sepsis	3 (11%)	4 (14%)	0.72
Pneumonia		8 (28%)	0.83
Acute renal failure	1 (4%)	1 (4%)	0.98
Hepatic failure	1 (4%)	1 (4%)	0.98

SR = standard rewarming; CAVR = continuous arteriovenous rewarming; ARDS = adult respiratory distress syndrome.

* Twelve of 28 control patients and two CAVR patients died before rewarming occurred, and are not included in rewarming times. Chi square for categorical data, Mann-Whitney-U test for continuous data.

Can hypothermia be beneficial?

- Accidental deep hypothermia
- Hypothermic cardiac surgery
- Organ transplantation
- Hypothermic induction in cardiac arrest

- Can hypothermia be beneficial in the trauma setting?

Hypothermia and traumatic brain injury

Moderate hypothermia (32-34^{0}C):

- reduction of cerebral ischaemia, oedema, and tissue injury; reduction in metabolic rate
- ?reduction of excitatory neurotransmitters eg.glutamate
- reduction of post-traumatic inflammatory response
  - preservation of blood-brain barrier
  - reduction of cytokines

Treatment of traumatic brain injury with moderate hypothermia. Marion et al, 1997

Prospective randomised trial

82 patients with severe CHI (GCS 3-7)

Randomised to hypothermia (cooled to 33^0C for 24 hours) or normothermia

Blinded assessment of outcome using the Glasgow Outcome Score at 3, 6, 12 months

GOS: 1-death; 2-vegetative state; 3-severe disability; 4-moderate disability; 5-mild or no disability

Treatment of traumatic brain injury with moderate hypothermia. Marion et al, 1997

Demographics similar between the two groups

No differences in LOS, morbidity or mortality

Neurologic outcomes:

- no difference for those who were GCS 3 or 4
- significantly greater proportion in hypothermia group with good outcome at 3, 6 and 12 months

Lack of effect of induction of hypothermia after acute brain injury. Clifton et al, 2001

Multicentre prospective randomised trial (National Acute Brain Injury Study)

392 patients with severe CHI (GCS 3-8)

Randomised within 6 hours of injury to moderate hypothermia (T 33^0C) for 48 hours, or normothermia

Blinded assessment of outcome using the Glasgow Outcome Score at 6 months

Lack of effect of induction of hypothermia after acute brain injury. Clifton et al, 2001

Demographics similar between the two groups

More complications in hypothermia group
No difference in mortality

Neurologic outcomes:
- no difference between the two groups

Noted that patients hypothermic on admission had:
- higher ISS
- more likely to be hypotensive
- received more IV fluids

Summary

- Is hypothermia a friend or foe in the injured patient?
  - Foe>friend, but not completely resolved
  - Injured organs and tissues seem unlikely to respond as well to hypothermia
- Prevention of hypothermia still the optimal goal
  - Treatment of hypothermic patients requires further study