Targeted Temperature Management

Delia Russell RN

1

Faculty Disclosure

- I am currently employed as a Clinical Education Specialist with Medivance, Inc®

Objectives

- Understand the importance of targeted temperature management
- Review current strategies to address fever
- Identify signs of shivering and means to help control shivering

3

The goal of targeted temperature management in the neurologically impaired patient is neuroprotection

4

Neuroprotection

- Protecting neurons from apoptosis (programmed cellular death) or degeneration after a brain injury

5

Two Documented Facts:

1. **Hypothermia** offers neuroprotective benefits.
   - ⇩ tissue oxygen demand for the brain
   - ⇩ temp by 1ºC = 6-8% decrease in cerebral metabolic rate
   - ⇩ Decrease Negative cascade of events due to SCA
2. Mild to moderate **hyperthermia** is detrimental in ischemic brain injury and traumatic brain injury.

Ischemia

"…ischaemia has a key role in all forms of brain injury and preventing ischaemic injury is central to all neuroprotective strategies." (Dr. Kees Polderman)

Lancet, 2009

7

Neuronal Damage from Ischemia

- Negative cascade of reactions occur at cellular level
  - May continue for hours to days after initial insult
- Temperature dependent
  - Increased by fever
  - Inhibited by mild hypothermia

Polderman, Lancet, 2009

8

Ischemia

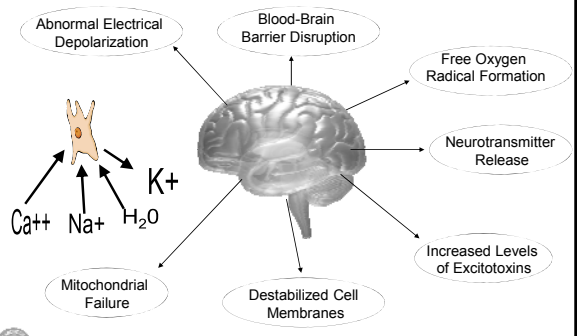
- Global ischemia: no perfusion to brain
- Focal ischemia: lack of perfusion to one area of brain

9

Pathophysiology

- Brain loses oxygen stores within 20 seconds
- Damage starts 4-6 minutes after the heart stops
  - Glucose and ATP stores are deplete (brain energy)
  - Membrane depolarization
  - Calcium influxes
  - Glutamine is released
  - Acidosis and edema develop
- Ischemia may persist for several hours after resuscitation (re-perfusion injury)

Neuronal Damage



11

Reperfusion Injury

- Occurs after blood flow is restored
- Secondary wave of excitotoxicity and free radical formation
- May exacerbate initial effects of blood deprivation
- Leads to blood brain barrier breakdown, cerebral edema and hemorrhage

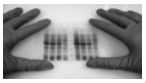
12

Hypothermia

13

History of Cooling

- Moderate Hypothermia used early 1900's: had poor outcomes
- 1940-1960's: TH revisited as a neuroprotective intervention: again poor outcomes
- Researchers learned a lot from cooling during coronary artery bypass grafting (CABG) on bypass
- In the mid-1980's and early 1990's: Animal studies revealed protective benefits from **mild hypothermia** and researchers realized that moderate or deep hypothermia was not necessary
- 1997: first human study by Dr. Bernard with mild hypothermia



14

Research on Hypothermia

Two Landmark studies were published in New England Journal of Medicine in February, 2002

The New England Journal of Medicine

Induced hypothermia in critical care medicine: A review

15

Research Outcomes

Pittsburgh Cerebral Performance Scale:

1. Good recovery
2. Moderate disability
3. Severe disability
4. A vegetative state
5. Death

16

Two Landmark Studies

- HACA Study (Europe)
- 9 facilities; 5 countries
- Randomized 275 pts
- Cooled 32-34 X 24 hrs

- Results:
  TH 55% vs Normo 39%

- Australian Study
- (Dr Bernard)
- 4 hospitals in Melbourne
- Randomized 77 pts
- Cooled 33 X 12 hrs

- Results:
  TH 49% vs Normo 26%

ACC/AHA Guidelines 2005

Circulation

American Heart Association. Learn and Live.

JOURNAL OF THE AMERICAN HEART ASSOCIATION

Thus, unconscious adult patients with ROSC after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C (89.6°F to 93.2°F) for 12 to 24 hours when the initial rhythm was VF (Class IIa). Similar therapy may be beneficial for patients with non-VF arrest out of hospital or for in-hospital arrest (Class IIb).

Part 7.5: Postresuscitation Support

Circulation 2005;112;IV-84-IV-88; originally published online Nov 28, 2005; DOI: 10.1161/CIRCULATIONAHA.105.166560

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

IIa. Weight of evidence/opinion is in favor of usefulness/efficacy

IIb. Usefulness/efficacy is less well established by evidence/opinion.

2010 AHA Update

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION
American Heart Association
Learn and Live

Part 9: Post Cardiac Arrest Care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Mary Ann Peberdy, Clifton W. Callaway, Robert W. Neumar, Romergryko G. Geocadin, Janice L. Zimmerman, Michael Donnino, Andrea Gabrielli, Scott M. Silvers, Arno L. Zaritsky, Raina Merchant, Terry L. Vanden Hoek and Steven L. Kronick

Circulation 2010;122;S768-S786
DOI: 10.1161/CIRCULATIONAHA.110.971002
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

19

2010 Updated AHA Guidelines

In summary, we recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32°C to 34°C (89.6°F to 93.2°F) for 12 to 24 hours (Class I, LOE B). Induced hypothermia also may be considered for comatose adult patients with ROSC after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of pulseless electric activity or asystole (Class IIb, LOE B). Active rewarming should be avoided in comatose patients who spontaneously develop a mild degree of hypothermia (>32°C [89.6°F]) after resuscitation from cardiac arrest during the first 48 hours after ROSC. (Class III, LOE C).

- Core Temperature Measurement If Comatose
- Rationale: Minimize brain injury and improve outcome
- Prevent hyperpyrexia >37.7°C
- Induce therapeutic hypothermia if no contraindications
- Cold IV fluid bolus 30 mL/kg if no contraindication
- Surface or endovascular cooling for 32°C–34°C×24 hours
- After 24 hours, slow rewarming 0.25°C/hr
- Consider Non-enhanced CT Scan
- Rationale: Exclude primary intracranial process

20

Hypothermia

- Definitions of hypothermia:
  - Mild: 33-36°C (91.4- 96.8°F)
  - Moderate: 26°-32°C
  - Deep: 20°-25°C
  - Profound: < 20°C

Hammer, *Clev Clinic J Med*, 2002

21

Effects of Hypothermia: Neuroprotection

- Inhibit neurotransmitter release
- Inhibit free radical production
- Reduces tissue oxygen demand
- Decrease cerebral metabolic rate (8% for every 1°C)
- Suppress inflammatory cells and factors
- Preserve blood brain barrier integrity
- ICP is decreased

Sessler, *CCM*, 2009

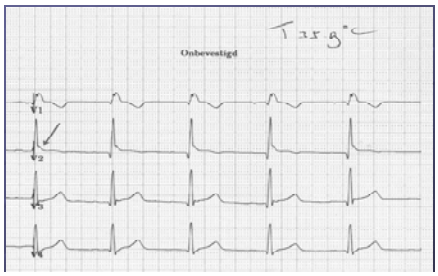
22

Physiological Effects of TH 32-35 C

- ***Cardiovascular:***
  - Initially HR, CO & BP
    - Then ↓ HR, CO, BP
  - EKG Changes
    - Prolonged PR interval
    - Widening QRS complex
    - Increased QT interval
    - Osborne wave
- ***Hematological:***
  - Thrombocytopenia
  - Impaired clotting cascade
  - Impaired platelet function
  - Decreased WBC Count
- ***Gastrointestinal:***
  - Impaired bowel function/motility
  - Mild pancreatitis
  - Increased liver enzymes

23

Osborne Wave



Lancet, 2009

24

Physiological Effects of TH 32-35 C

Systemic:
- –↓ O2 Consumption
- –Left shift on the oxyhemoglobin curve
- –↓ CO2 production
- –↑ Lactate levels

Immune suppression:
- –↓Neutrophil and macrophage function
- –↑Infection (wound infections and pneumonia)

Endocrine
- –↓ Insulin secretion

Renal:
- –Diuresis
- –Electrolyte Loss

25

Physiological Effects of TH 32-35 C

Seizures:
- TH may have anti-epileptic properties
- Seizure activity may be missed during paralysis
- "Continuous EEG monitoring should be considered if convulsive or nonconvulsive seizures are suspected" (Seder)
- When continuous EEG unavailable, consider utilizing antiepileptic sedatives for anti-shivering protocol (Propofol, Benzodiazepines)

Seder, *CCM*, 2009

26

Pharmacokinetics and TH

Evidence for prolonged drug clearance:
- Neuromuscular Blockade
- Morphine
- Fentanyl
- Propofol
- Volatile Anesthetics
- Barbituates
- Midazolam
- Phenytoin
- Nitrates

Polderman, *CCM*, 2009

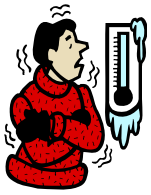
27

Physiological Effects of TH 32-35 C

Coagulopathies:
- No serious bleeding noted with the administration of aspirin, clopidogrel and abciximab (Hovdenes 2007)
- Mild hypothermia does not augment abciximab-induced inhibition of platelet aggregation (Frelinger 2003)
- Coagulopathies – rare to non-existent (Polderman 2008)

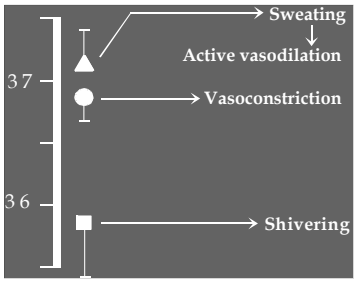
28

SHIVERING



29

Normal Thresholds for Thermoregulatory Responses



Sessler, *TH*, 2005

30

Shivering

- Involuntary Sympathetic Response
  - Vasoconstriction
  - Muscle contraction and twitching
  - Shiver to produce heat
- Increased in younger patients and increased muscle mass
- Inhibited by neuromuscular disease, muscle relaxants and decrease in muscle tone
- May increase oxygen consumption by 40-100%

31

Shivering Management

- ***Demerol***: will decrease shivering threshold
- ***Buspirone***: will potentiate Demerol and Fentanyl
- ***Sedatives*** (i.e. Fentanyl, Versed): may sedate enough to decrease shivering, but will not decrease shivering threshold
- ***Anesthetics*** (i.e. Propofol, Precedex, Pentobarbital): may decrease shivering threshold and sedate

Sessler, TH, 2005

Shivering Management…cont

- ***Magnesium***: vasodilitation
- ***Neuromuscular Blockade***: alleviate muscle movement and shivering
- ***Non-Pharmacological***: hand, foot and face warming provides "feeling" of warmth

Sessler, TH, 2005

33

Steps for Success: Phases of TH Treatment

Induction Phase:

- Initiate quickly
- Careful monitoring of fluid balance
- Prevention of hypovolemia and hypotension
- Tight glucose control
- Electrolyte management
- Prevention of infectious complications
- Adjustment of various medications
- Adjustment of ventilator settings
- Prevention of shivering

34

Steps for Success: Phases of TH Treatment

Maintenance Phase:

- Tightly controlled core temperature with minor fluctuations
- Prevent and manage potential side effects:
  - Bradycardia
  - Glucose management
  - Wound infections
  - Pulmonary infections (especially pneumonia)
  - Skin breakdown
  - Electrolyte imbalance

35

Steps for Success: Phases of TH Treatment

Re-warming Phase:

- Slow and controlled re-warming
  - 0.1-0.5°C/h
- Slower re-warming preserves the benefits of TH
- Rapid re-warming may lead to:
  - Rapid electrolyte shifts (hyperkalemia)
  - Increased ICP
  - Sudden vasodilatation
  - Hypoglycemia

Polderman, *ICM*, 2002

36

They're Warm…Now What?

Controlled Normothermia (4th Phase of Cooling):

- "It may be reasonable to conclude that Controlled Normothermia is beneficial for at least 48 hours after 24 hours of therapeutic hypothermia."
- Badjatia, CCM, 2009

37

Patience is a Virtue

"Durations of observation greater than 72 hours …should be considered before predicting poor outcome in patients treated with hypothermia"

Circulation 2010;122;S768-S786

38

Hypothermia and the Injured Brain

39

Traumatic Brain Injury

- There are no conclusive human studies that therapeutic hypothermia improves neurological outcomes
- Clinical Trials: Too Much Variation
  - Various patients populations
  - Many types of injuries
  - Diverging treatment protocols
  - Duration of cooling (24h to 5 days)
  - Duration of rewarming

Polderman, *Lancet*, 2008

40

TTM™ in TBI: ICP control

Reference	PRCT	N	Length of cooling	Results
Shiozaki et al, 1993	Y	33	48h	Decrease
Clifton et al, 1993	Y	46	48h	No change
Metz et al, 1996	N	10	25H	Decrease
Marion et al, 1997	Y	82	24h	Decrease
Nara et al, 1998	N	23	?	Decrease
Tateishi et al, 1998	N	9	Max of 6 days	Decrease
Takahashi et al, 2000	N	9	3-21 days	Decrease
Jiang et al, 2000	Y	87	3-14 days	Decrease
Clifton et al, 2001	Y	392	48h	Decrease
Biswas et al, 2002	Y	21	48h	Decrease
Gal et al, 2002	Y	30	72h	Decrease
Tokutomi et al, 2003	N	31	48-72h	Decrease
Zhi et al, 2003	Y	396	1-7 days	Decrease
Qiu et al, 2005	Y	86	3-5 days	Decrease
Qiu et al, 2007	Y	80	5 days	Decrease

41

Effects of therapeutic hypothermia on intracranial pressure

- Study included 136 patients admitted to teaching hospital in The Netherlands
  - Severe head injury
  - GCS </= 8
  - ICP > 20mmHg after conventional therapy
    - Control group (Barbiturate coma): 72
    - Moderate hypothermia (32-34°C): 64

Polderman, *ICM*, 2002

42

Effects of therapeutic hypothermia on intracranial pressure

- MAP maintained > 90mmHg
- CPP maintained > 70mmHg
- Hypothermia continued until ICP remained below 20mmHg for 24hrs
  - Rewarmed at 1°C/12h
  - If ICP increased above 20mmHg, patient target temperature was decreased again
  - Average duration of hypothermia = 4.8 days

Polderman, *ICM*, 2002

43

Effects of therapeutic hypothermia on intracranial pressure

	Hypothermia	Control
Mortality	62%	72%
Good neurological outcome	15.7%	9.7%
Subgroup of patients with admission GCS 5 or 6:		
• Mortality	52%	76%
• Good neurological outcome	29%	8%

Polderman, *ICM*, 2002

44

Managing ICP with TH

Most evidence reveals:

- TH can be effective in patients with severe TBI and elevated ICP when:
  1. treatment is initiated early,
  2. continued for sufficient time (2-5 days),
  3. and when patients are rewarmed slowly (at least over 24 hours)
- Management of side effects (hypotension and hypovolemia) is imperative

Polderman, *Lancet*, 2008

45

Optimal Temperature for the Management of Severe Traumatic Brain Injury: Effect of Hypothermia on Intracranial Pressure, Systemic and Intracranial Hemodynamics, and Metabolism

- Cerebral perfusion pressure peaked at 35.0 to 35.9°C and decreased with further decreases in temperature.
- Intracranial pressure decreased significantly at brain temperatures below 37°C and decreased more sharply at temperatures 35 to 36°C, but no differences were observed at temperatures below 35°C.
- Jugular venous oxygen saturation and mixed venous oxygen saturation remained in the normal range during hypothermia.

Neurosurgery, Jan 2003; Vol 52, Number 1; 102-112

46

What Do the Guidelines Say?

Guidelines for the Management of Severe Traumatic Brain Injury

3rd Edition

Brain Trauma Foundation
and
American Association of Neurological Surgeons (AANS)
Congress of Neurological Surgeons (CNS)
AANS/CNS Joint Section on Neurotrauma & Critical Care

47

TBI Guidelines

Prophylactic Hypothermia

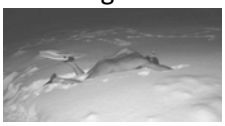
Recommendations

Level III: Pooled data indicate that prophylactic HT is not significantly associated with decreased mortality when compared with normothermic controls.

Preliminary findings suggest that a greater decrease in mortality risk is observed when target temperatures are maintained for more than 48 hours. Prophylactic HT is associated with significantly higher GOS scores when compared to scores for normothermic controls."

48

What is the best method for inducing hypothermia?



- External
  - Ice packs
  - Mechanical cooling devices: Blankets, vests etc
- Internal
  - Iced fluids
  - Femoral vein catheters

GOAL: to be able to control pt's temperature through a continuous feedback loop

Infusion of ice-cold IV Fluids to Induce Hypothermia

Bernard
- 22 patients (paralyzed)
- 30ml/kg of 4°C LR over 30 minutes
- Used peripheral or femoral venous access
- Median difference of 1.6°C
- Improvements seen in MAP, renal function and acid-base balance
- No pulmonary edema

Bernard et al. *Resuscitation*. 2003

Kim
- 17 patients
  - Paralyzed or sedated
- 2L of 4°C NS over 20-30 minutes
- Used peripheral vein
- Paralyzed patients had mean difference of 1.7°C
- Sedated patients had mean difference of 1.1°C within 30 min
- No pulmonary edema

Kim et al. *Circulation*. 2005

50

Controlled Normothermia

51

Fever in Neurological Injuries

"Fever is a common complication in patients with various types of neurological injury and is independently associated with an increased risk of adverse outcome. This link persists after multivariate analysis (correcting for factors such as presence of infection), and applies to infectious and non-infectious causes." (Polderman, *Lancet*, 2008)

52

Fever is Bad…

"Moreover, increasing evidence suggests that fever (irrespective of its cause) can directly and adversely affect neurological outcome in various types of neurological injury" (Dr Kees Polderman)

Lancet, 2009

53

Pathophysiology of Fever

- Rise in temperature due to regulated increase in patient's hypothalamic set point
  - ↑ oxygen consumption
  - ↑ metabolic rate
  - ↑ heart rate
  - ↑ cardiac output
  - ↑ leukocyte count
  - ↑ level of C-Reactive Protein
- May be detrimental to a critically ill patient

54

Brain Temperature

- Local brain temperature may exceed core temperature by up to 2°C
  - This variance may increase in the injured brain due to hyper-metabolism in injured areas

55

Prevalence of Hyperthermia

- Defined as temperature increase of > 38.3 C or one full degree above baseline
- Occurs in up to 70% of all neurologically-injuried patients
- Typically not an isolated event but a sustained response for as long as 2 weeks
- Risk of hyperthermia may increase by 32% for each additional day in ICU
- Evidence suggests that fever worsens injury after ischemia or trauma

McIlvoy, *AACN Clin Issues*, 2006
Badjatia, *Current Neur and Neurosc Reports*, 2009

56

Elevated Body Temperature & Increased LOS

- Retrospective study
- Reviewed 4,295 admissions to a 20-bed neurology/neurosurgery ICU over a period of 6 years
- Measurements included APACHE scores, GCS scores, daily maximum temperature, complications, length of stay, mortality rate and discharge disposition
- Controlled for age, diagnosis, severity of illness and complications

Diringer, CCM 2004

57

Results

Patient Temperature	No Fever <37.5° C n=1268	Low Fever 37.5-38.4° C n=1591	Medium Fever 38.5-39.0° C n=719	High Fever >39.0° C n=717
Mortality	9.1%	7.8%	16.3%	28.7%
Discharge to Home	59%	49%	24%	17%

•Elevated body temperature was associated with 3.2 additional IC U days and 4.3 additional hospital days.

Diringer, *CCM* 2004

58

Neurogenic Fever

- Occurs in patients with acute brain injury
- Intraventricular catheterization is a risk factor

Thompson et al, *J Neurol Neurosurg Psychiatry*, 2003

59

Neurogenic Fever

- Non-infectious source of fever
- Results from disruption in the hypothalamic set point
- Injury to hypothalamus; unclear mechanism
- Patient response
  - Increased temperature (may plateau), resistant to antipyretics
  - Absence of perspiration

60

Diagnosis of Fever

- "Diagnosis of exclusion"
- Work up required includes:
  - Chest X-Ray
  - Blood cultures (anaerobic and aerobic)
  - Sputum culture
  - Urine culture
  - Cerebral spinal fluid culture
  - Bronchial aspirate
  - Invasive lines cultured

Thompson et al. *J Neurol Neurosurg Psychiatry*, 2003

61

What do the Guidelines Say?


Stroke
American Stroke Association.

62

Ischemic Stroke

"Because of the negative effects of fever, lowering an acutely elevated body temperature might improve the prognosis of patients with stroke"

"It is generally agreed that sources of fever should be treated and antipyretic medications should be administered to lower temperature in febrile patients with stroke" Class I, Level C

Stroke, 2007

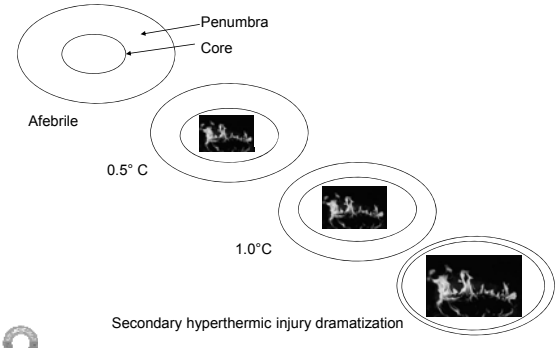
63

Implications of Fever

- Fever on admission has been correlated with larger infarct sizes and increased mortality
  - Each 1 C increase in temperature "doubles the relative risk for poor functional outcome" (Dr Badjatia)
  - Neuronal loss may be increased even when occurring 24 hours after original insult
- Maintaining normothermia after ischemia stabilizes the blood-brain barrier and reduces cerebral metabolism

64

Temp elevations aggravate ischemic neuronal injury and exacerbate brain edema



Adapted from Ginsberg, 2002

65

TTM in TBI: Normothermia


neurocritical care society Neurocrit Care (2009) 11:82–87
DOI 10.1007/s12028-009-9213-0

ORIGINAL ARTICLE

Induced Normothermia Attenuates Intracranial Hypertension and Reduces Fever Burden after Severe Traumatic Brain Injury

Ava M. Puccio · Michael R. Fischer · Brian T. Jankowitz · Howard Yonas · Joseph M. Darby · David O. Okonkwo

66

Hyperthermia in the Injured Brain

- Intracranial blood volume increases with rise in temperature
  - Causes a reduced compliance and increases ICP
  - Elevates risk for further neuronal injury
- 13% increase in metabolic rate associated with every 1°C increase in body temperature
  - Increases risk of secondary injury

Thompson et al, *J Neurol Neurosurg Psychiatry*, 2003

67

Hyperthermia in the Injured Brain

- Fever occurring within first week of injury has been associated with:
  - Increased ICP
  - Cerebral Vasospasm
  - Neurologic impairment
  - Prolonged ICU stay
  - Long-term poor outcome
- Recommendation is to treat fever aggressively in this patient population

Badjatia, *CCM*, 2009

68

TTM in TBI: Normothermia

neurocritical care society Neurocrit Care (2009) 11:82–87
DOI 10.1007/s12028-009-9213-0

ORIGINAL ARTICLE

Induced Normothermia Attenuates Intracranial Hypertension and Reduces Fever Burden after Severe Traumatic Brain Injury

Ava M. Puccio · Michael R. Fischer · Brian T. Jankowitz · Howard Yonas · Joseph M. Darby · David O. Okonkwo

69

What do the Guidelines Say?

70

Management of Cerebral Vasospasm after SAH

"Delayed narrowing of large capacitance arteries at the base of the brain after SAH, often associated with diminished perfusion in the distal area of the affected artery."

- Imperative to avoid systemic & metabolic insults:
  - Hyperglycemia, acidosis, electrolyte fluctuations, hypoxia, and **hyperthermia**
  - **Maintain Normothermia**

Stroke, 2007

71

Continuing Medical Education Article

Intensive care of patients with acute liver failure: Recommendations of the U.S. Acute Liver Failure Study Group

R. Todd Stravitz, MD; Andreas H. Kramer, MD, MSc; Timothy Davern, MD; A. Obaid S. Shaikh, MD; Stephen H. Caldwell, MD; Ravindra L. Mehta, MD; Andres T. Blei, MD; Robert J. Fontana, MD; Brendan M. McGuire, MD; Lorenzo Rossaro, MD; Alastair D. Smith, MD; William M. Lee, MD; the Acute Liver Failure Study Group

- Generally, **maintenance of euthermia (36.5–37.5°C)** is recommended in patients with ALF, because fever exacerbates intracranial hypertension and is independently associated with worse outcome in patients admitted to neurologic intensive care units.
- **Fever should be treated aggressively with cooling blankets, fans, or other noninvasive devices, but nonsteroidal anti-inflammatory drugs and acetaminophen are not recommended because of possible nephro- and gastric mucosal toxicity, and possible potentiation of liver injury.**

MT08168 Rev D

72

Temperature Control

- What the brain likes
- What the brain does NOT like

73

Treating Fevers

- Pharmacological (Tylenol):
  - Linked to intact thermoregulatory mechanisms
- Surface Cooling
  - Water blankets
  - Hydrogel pads
- Intravascular
  - Endovascular catheters
  - Iced saline

74

Treatment: Controlled Normothermia

- "Traditional therapy" has consisted of water blankets and Tylenol, which have been proven to have <40% success rates
- Advanced technology
  - 75% reduction in fever

Carhuapoma, *J Neurosurg Anesth*, 2003
Mayer, *CCM*, 2004

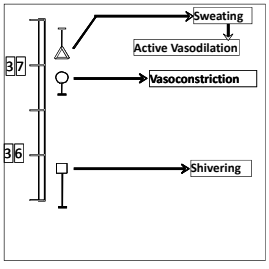
75

Shivering

- Setpoint has been reset
- Patients will shiver even when goal is normothermia
- Therefore, shivering must be addressed with this pt population

76

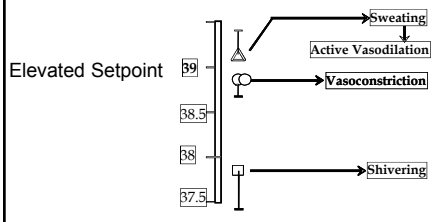
Normal Thresholds for Thermoregulatory Responses



Sessler, 2005

77

Thresholds for Thermoregulatory Responses During Neurogenic Fever



Interthreshold range remains constant when **setpoints are shifted**

78

Bedside Shivering Assessment Scale BSAS

0. No Shivering

1. Mild Shivering, localized to neck and/or Chest.

2. Shivering, neck and /or chest and <2 extremities.

3. Intermittent generalized shivering involving more than 2 extremities.

Badjatia, *Stroke*, 2008

79

Shivering Management

Badjatia et al; *Critical Care Med* 2006

Found that surface counter warming provides beneficial control of shivering and improves metabolic profile in patients undergoing therapeutic temperature modulation.

80

Program Summary

- It is important to properly manage patients receiving targeted temperature management
- One must be dedicated to shivering control to effectively cool patients to normothermia or therapeutic hypothermia
- Fever in the average patient indicates a good host response
- Fever in an acute brain injury could cause secondary hyperthermic brain injury

81

Thanks for your time. Have a great day!

82

MT08168 Rev C