Life Lessons for Neurogenic Fever Management

Leave It To Fever

Presented by:
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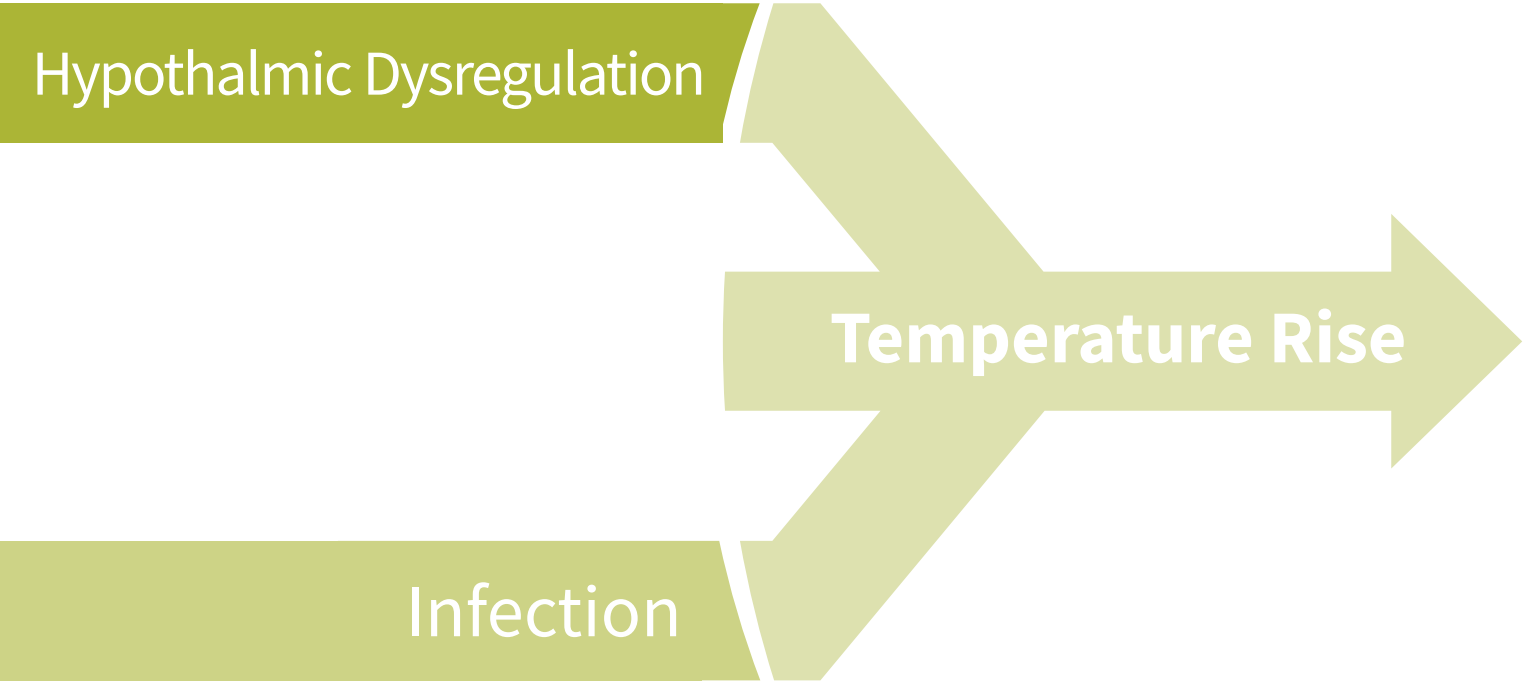
Fever: Core Body Temperature Exceeding 38.3°C[1]



Fever is linked to:

- Increased mortality
- Disability
- Length of stay[5,6]

Outcomes are independent of the underlying cause[7]

Hyperthermia in Brain Injured Patients

Fever Incidence

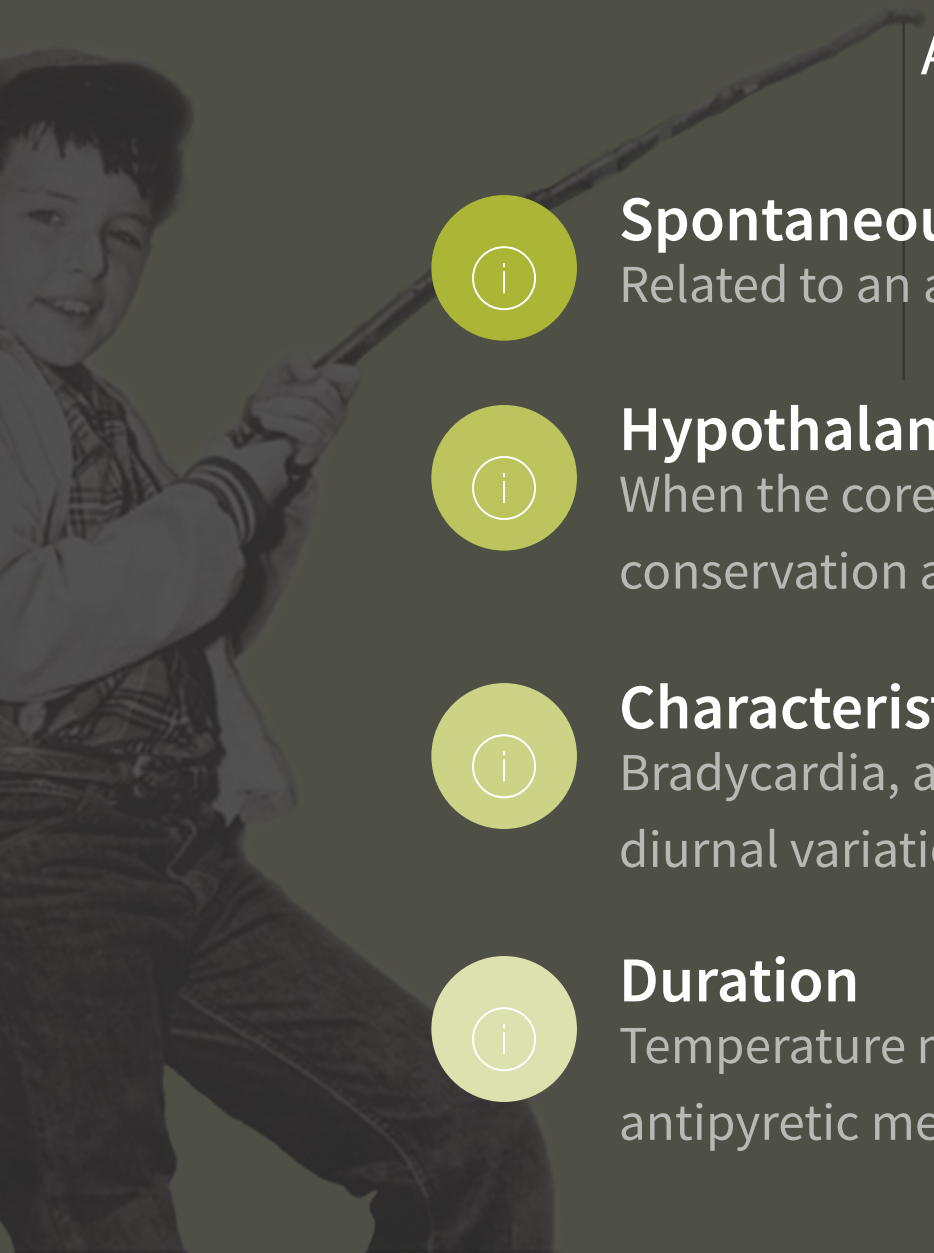
Occurs in up to 70% in neurologically impaired patients[1]

Direct Injury

To the thermoregulatory centers in the hypothalamus can lead to a rise in the core temperature setpoint[1]

Fever may Aggravate

Inflammatory cascades which are susceptible to mild increases in temperature after CNS injury, leading to worsened outcomes[2,3,5]



Neurogenic/Central Fever

A diagnosis of exclusion[3,6,7]

Spontaneous temperature elevation

Related to an acute brain injury[3]

Hypothalamus damage and setpoint elevation

When the core temperature setpoint is purposely elevated, mechanisms for heat conservation and generation are stimulated to attain the new setpoint[3]

Characteristics associated with neurogenic fever patients

Bradycardia, absence of perspiration, plateau-like temperature curve without diurnal variation[7]

Duration

Temperature may run very high and last for days to weeks, resistant to antipyretic medications[7]

Fever Research by Diagnosis

Ischemic Stroke

SAH

TBI

Fever
in Ischemic Stroke

Temperature Elevation

Of a degree Celsius is associated with double the relative risk for poor functional outcome[3]

Independent Association

A robust independent association between stroke severity, outcome and fever at admission and in first 24 hours has been established through clinical studies[1]

Greer, *et. al.*, 2008[8]

Patient Population

14,431 patients in 39 studies (stroke and other brain injuries)

Research Design

- Meta-analysis

Results

- Fever or elevated body temperature was significantly linked to worse outcome in every measure studied: mortality, GOS, Barthel Index, modified Rankin Scale, Canadian Stroke Scale, ICU LOS, hospital LOS

Saini, *et. al.*, 2009[9]

Patient Population

5305 patients in acute ischemic stroke trials

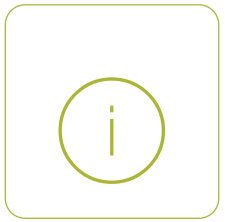
Research Design

- Pooled analysis from VISTA database

Results

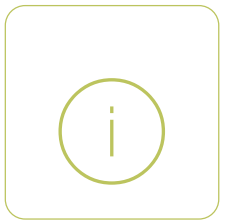
- Hyperthermia found to be a statistically significant predictor of poor outcome, with a delay in hyperthermia more predicative than in early hours post stroke

Fever
and Subarachnoid Hemorrhage



Fever Onset

Fever may occur in up to 70% of SAH patients during the first 10 days after the insult [1,2]



SAH Severity

Recurrent and elevated temperature has been seen in patients with more severe grades of SAH over 10 days[2]

Fever and Vasospasm

Fever has been correlated with vasospasm regardless of severity of SAH[10]

Fever
and Traumatic Brain Injury

Fever may lead to secondary injury in the injured brain[11]

Cerebral Metabolism

Every 1^{0}C increase in core body temperature raises cerebral metabolism by 7-13%[7,11]

Ischemia Threshold

is diminished in the injured brain, broadening the divergence between cerebral blood flow and metabolic demand[11]

Secondary Injury

Linked to longer ICU and hospital days, decrease in survival and quality of life[11]

Treatment Modalities

The effectiveness of antipyretic agents is linked to intact thermoregulatory responses; therefore, they may be ineffective in patients with hypothalamic injuries[3,5]

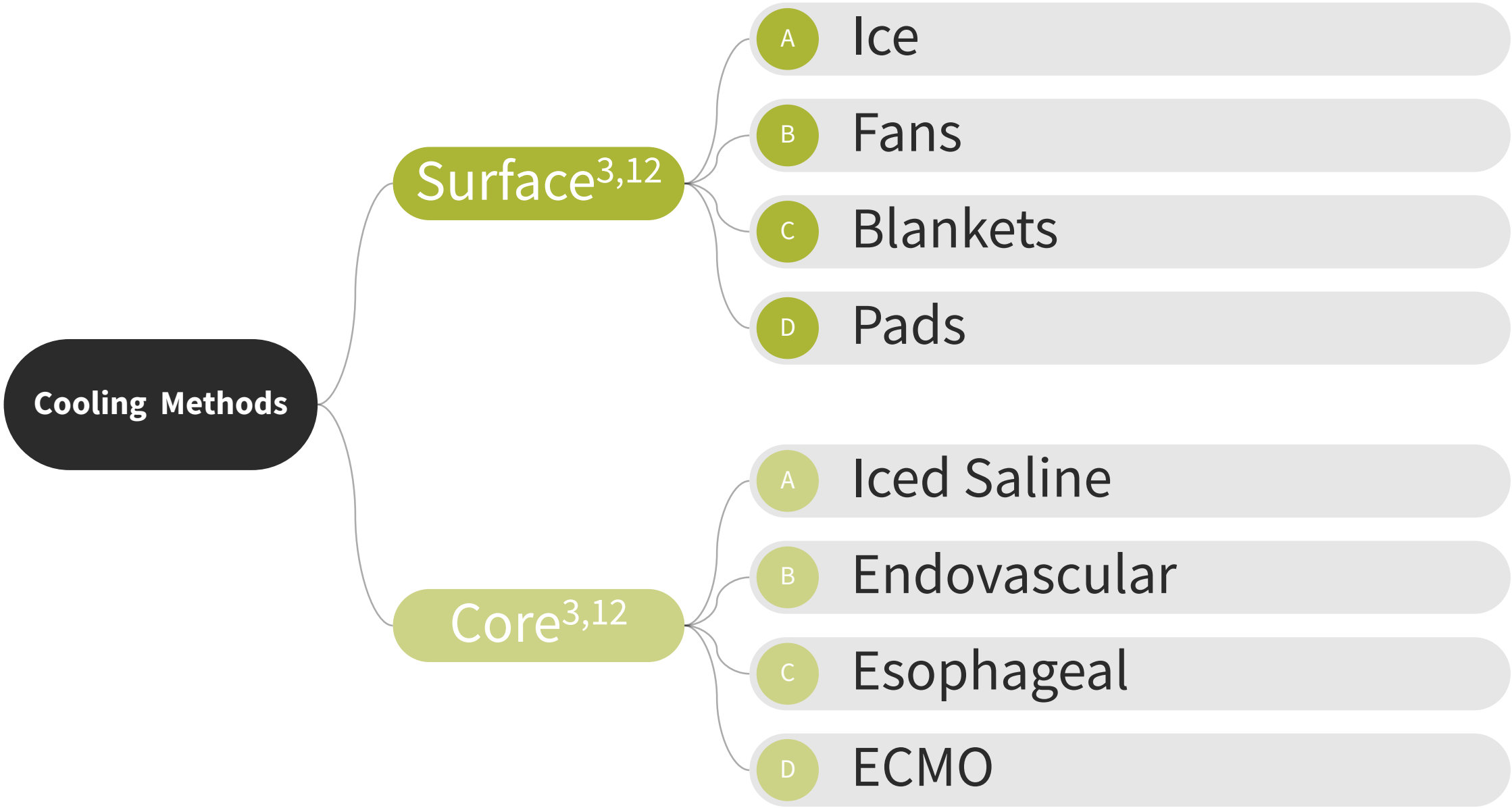
Ibuprofen[3]

Aspirin[3]

Acetominophen[3]

Mechanical cooling methodologies[3]

- Promotes heat loss without affecting hypothalamic set point
- Timing for treatment initiation remains contentious


Cooling Methods
Surface[3,12]
A Ice
B Fans
C Blankets
D Pads
Core[3,12]
A Iced Saline
B Endovascular
C Esophageal
D ECMO

Side Effects Associated with Fever Management

Shivering

Up to 40% incidence in patients undergoing NT[3]

Body is trying to generate heat and counteract cooling process

Vasoconstriction

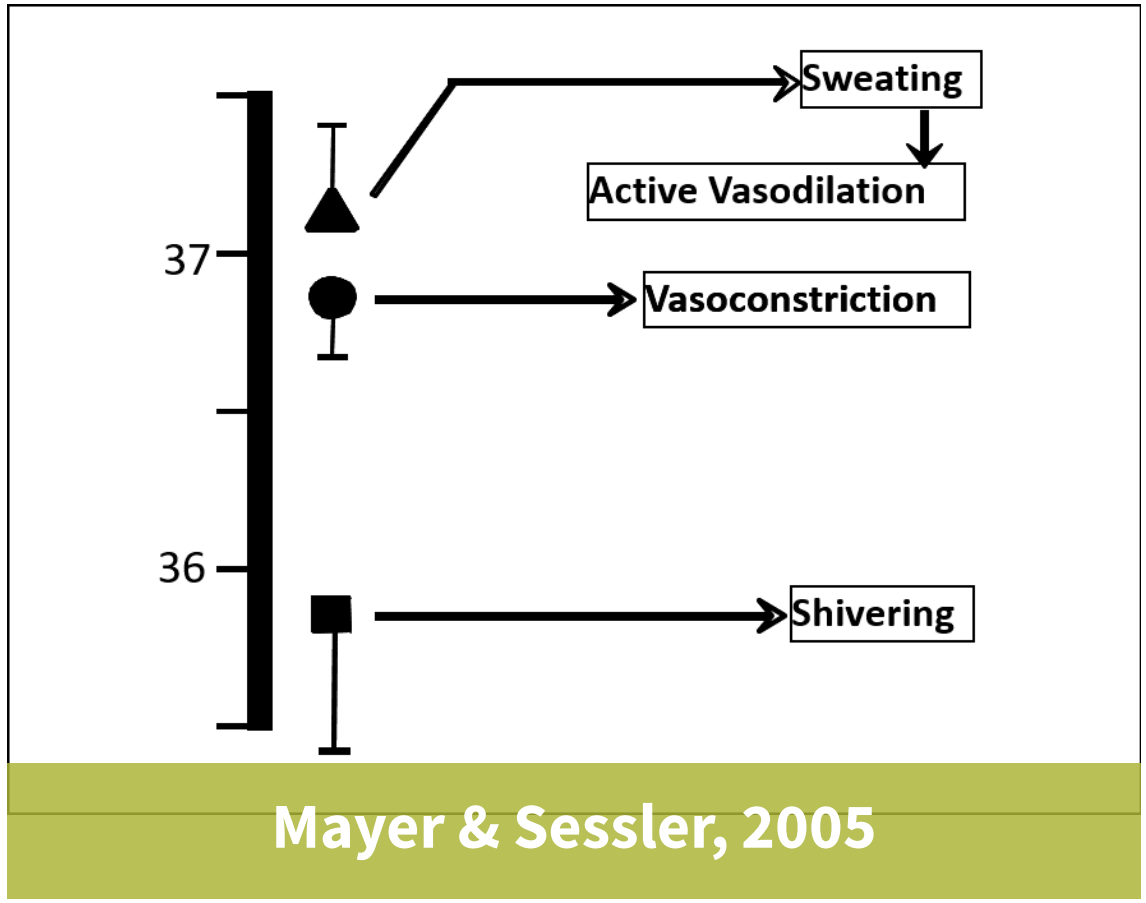
The body attempts to preserve heat and counteract the cooling process[3]

Impaired Febrile Response

Induced normothermia may impair the febrile response, which enhances body's ability to fight infection[1]

- Important to continue routine infection surveillance
- No standard approach exists
- May trend WBC counts or water temperature on cooling device

Normal Thresholds for Thermoregulatory Responses



Mayer & Sessler, 2005

01 Sweat at 37.3°C

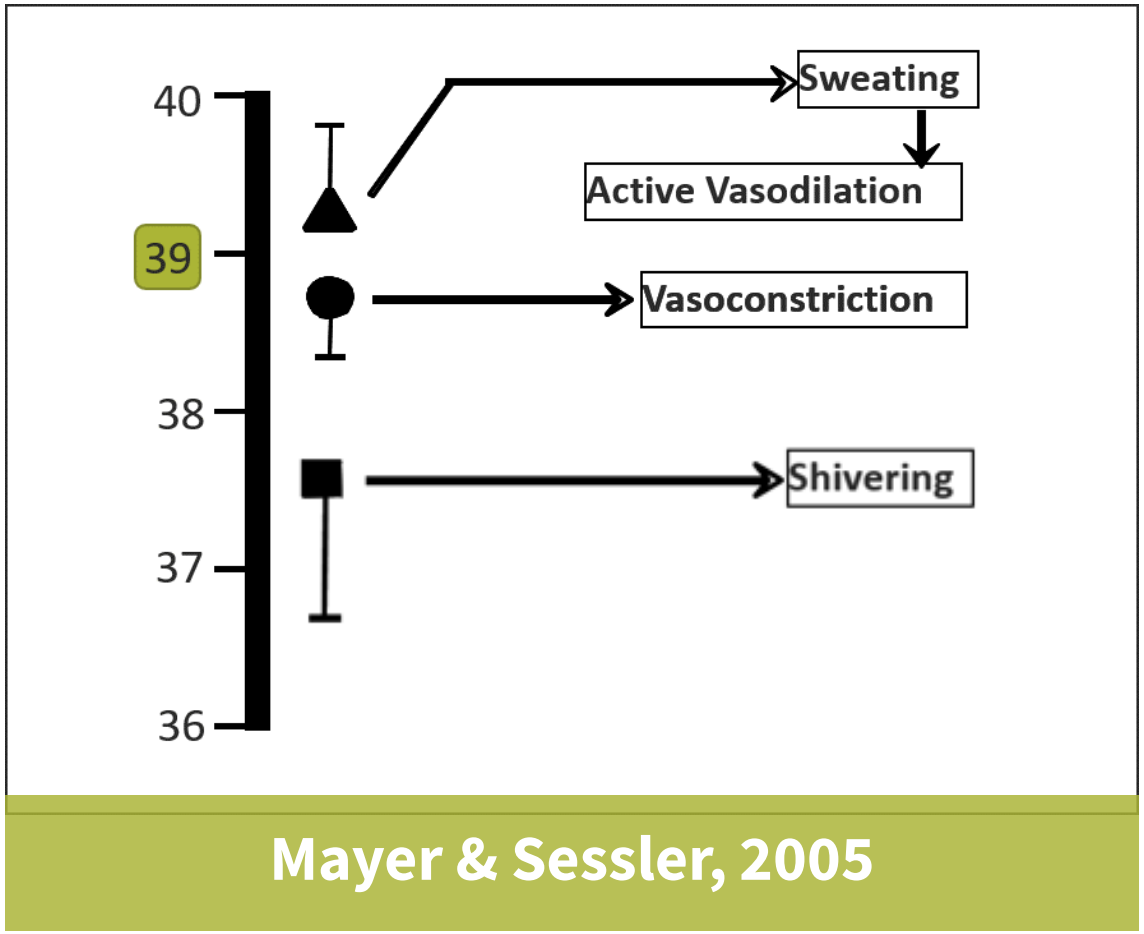
02 Vasoconstrict at 36.7°C

03 Shiver at 35.7 °C

**shivering occurs one full degree below vasoconstriction*[13,14,15,16]

Abnormal Shivering Thresholds

Shivering may occur at normothermic or hyperthermic stages due to elevation in set point[13,21]



Mayer & Sessler, 2005

Threshold Range

There is an interthreshold range that is maintained with shifts in these responses[13,15]

Elevated Setpoint

For example, if the temperature setpoint is elevated to 39^{0}C[13,15,21], the patient will:

- sweat at 39.3^{0}C
- vasoconstrict at 38.7^{0}C
- shiver at 37.7^{0}C

Pathophysiology of Shivering

Normal Involuntary Response

Physiological response to changes between sensed temperature and patient's setpoint[13,14,17]

Involuntary Skeletal Muscular Activity

Motor neurons are recruited[13]; motor response is initiated in the trunk, and spreads to extremities[14]

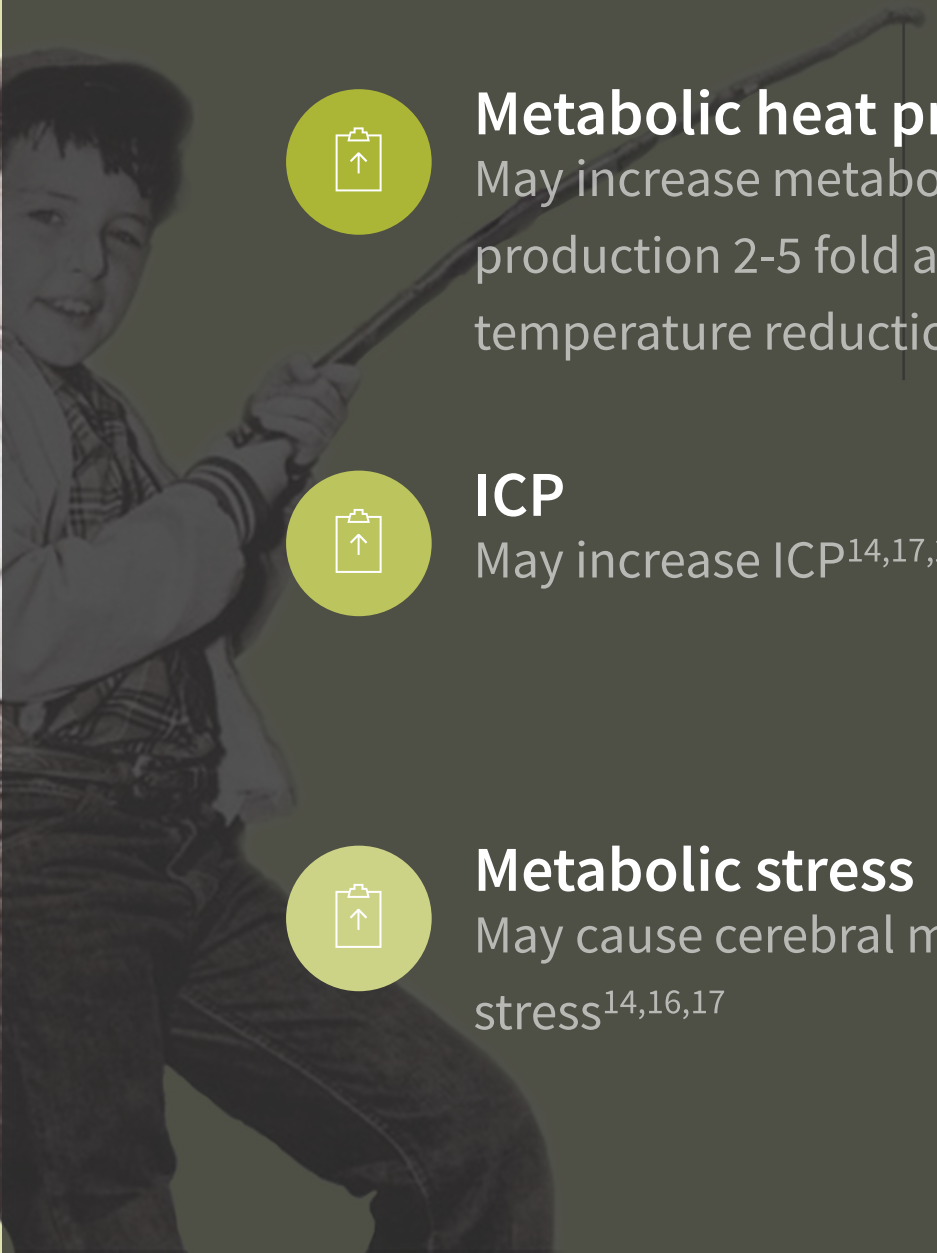
Highly Sensitive

Thermoreceptors near the skin surface are highly sensitive to temperature change and contribute to ~20% of thermoregulatory input and control of autonomic responses[13,17,18,19,20]

Increased Shivering

Shivering may be increased in younger patients, those with increased muscle tone, and low serum magnesium levels14,21

Impact of Shivering



Metabolic heat production
May increase metabolic heat production 2-5 fold and impede temperature reduction[14,16,18,19,21]

Cerebral oxygenation
May decrease cerebral oxygenation[17,22,23]

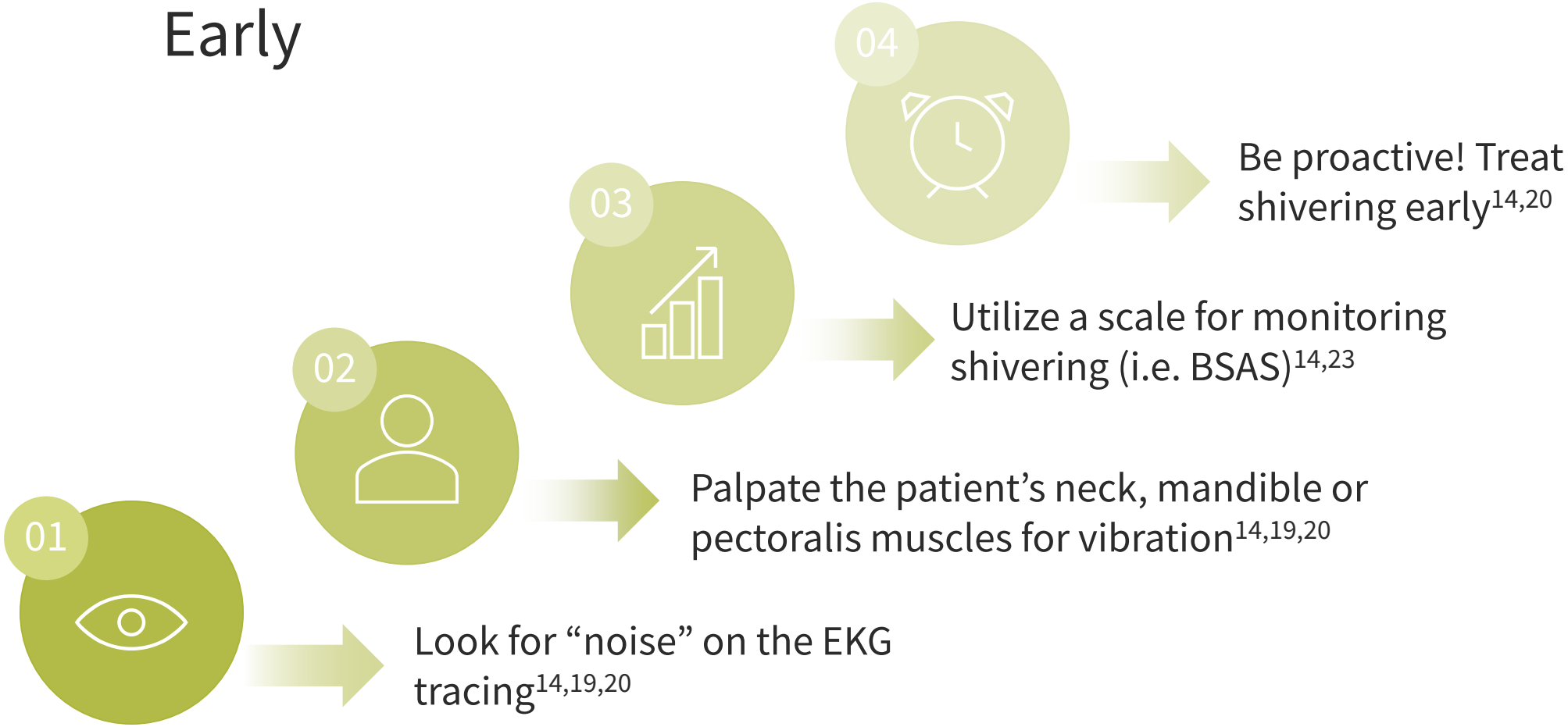
ICP
May increase ICP[14,17,22]

Metabolic demand
May increase metabolic demand which increases oxygen consumption 2-3 fold and respiration[14,16,21]

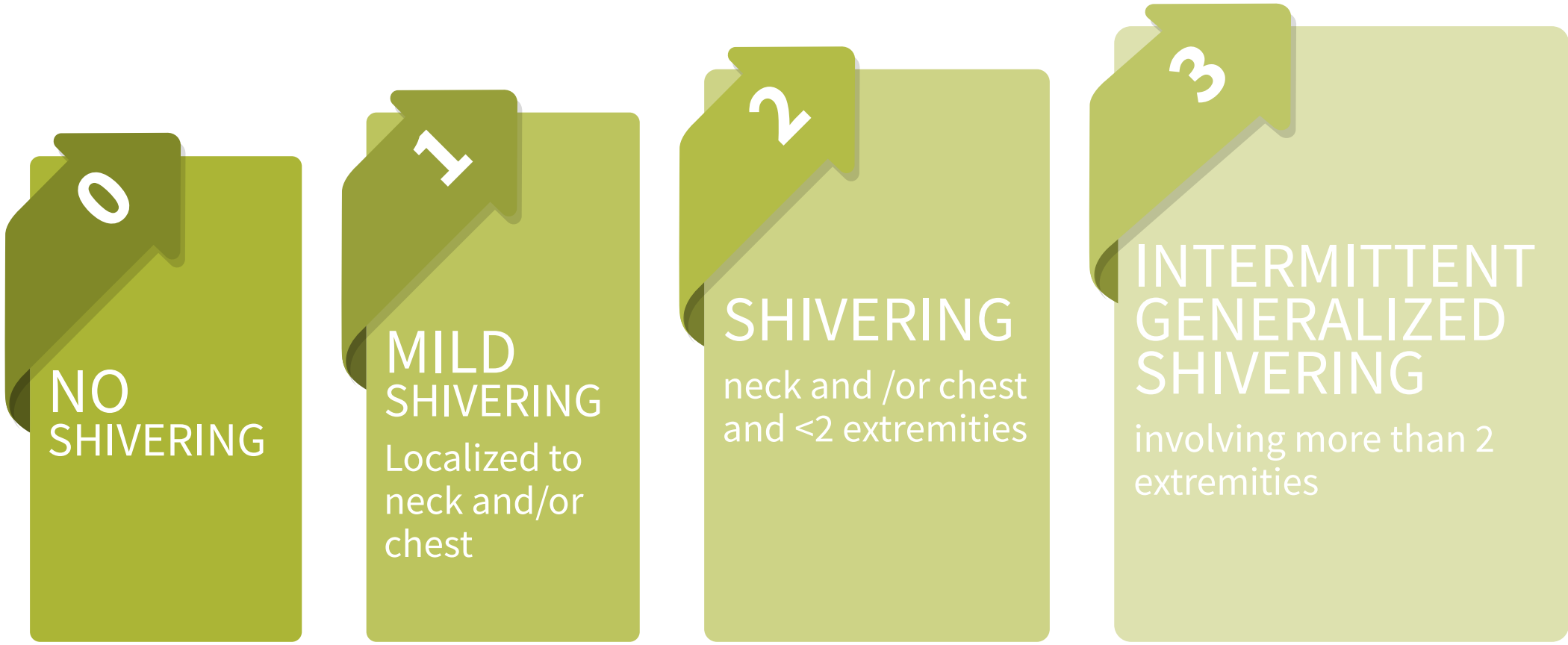
Metabolic stress
May cause cerebral metabolic stress[14,16,17]

TTM/fever control benefits
May eradicate any benefit of TTM/fever control[14,21]

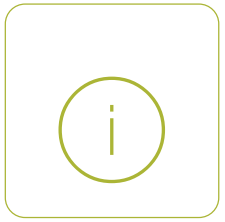
Detect Shivering Early



Bedside Shivering Assessment Scale (BSAS)[23]

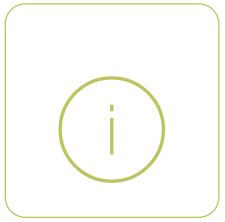


Shivering Management



Prevention

The goal is prevention[20]



Shivering Control

Aggressive shivering control is warranted in NT[14,17]

Stepwise Approach

Implementing a stepwise approach to address shivering which prioritizes the least sedating interventions and standardizes treatment may be beneficial[22]

Measure Efficacy

The efficacy of interventions can be measured through assessment with BSAS[14]

Non-Pharmacological Interventions

Counter-warming

- Surface counter-warming may be beneficial in lowering shivering threshold[18]

Shivering threshold

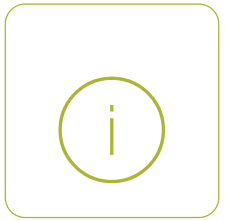
- There is a 1^0C decrease in shivering threshold with every 4^0C increase in mean skin temperature[13,18]

Prospective Study

- Badjatia, et. al. (2009) found the addition of a forced air warming blanket (43^0C) was effective in restricting the metabolic impact of shivering in the neurologically impaired patient[14]

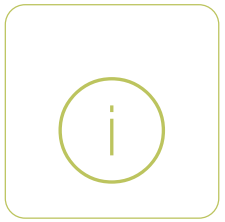
Conclusions

Final Thoughts



Worsened Outcomes

Hyperthermia has been associated with worsened outcomes in all forms of acute brain injury.[1,4,24]



Automatic Response

Shivering is an autonomic response to heat loss after vasoconstriction and requires aggressive treatment[16]

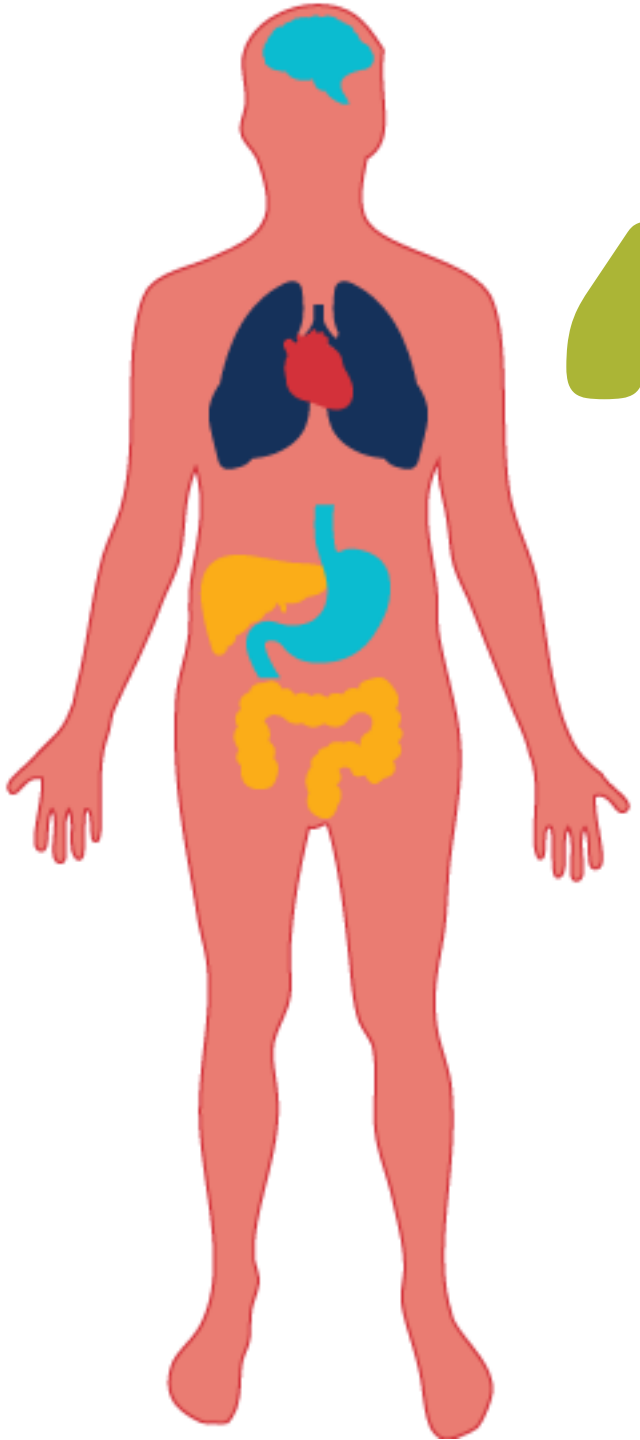
Shivering may Negate Benefits

Untreated shivering may negate any benefits of TTM[14,21]

Standardized Protocols

Ensuring standardized protocols are in place for shivering assessment and treatment is necessitated[22]

Badjatia[1] 2009



IN THE ABSENCE OF DEFINITIVE DATA, THE APPROACH TO FEVER MANAGEMENT SHOULD BE A BALANCE BETWEEN LIMITING SECONDARY INJURY AND IMPAIRING THE ABILITY TO FIGHT INFECTIONS

Questions remain

Best approach to achieve normothermia?[5]

Best practices for minimizing shivering?[5]

Who should receive therapeutic normothermia?[5]

What temperature should we cool to?[5]

What is the impact of NT on outcomes?[5]

Is there a causal relationship between fever and worsened outcomes or is fever a marker for more severe injury?[4]

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