Targeted Temperature Modulation in the Neuroscience Patient

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KEYWORDS

• Targeted temperature management • Induced hypothermia • Normothermia
• Shivering

KEY POINTS

- Both induced hypothermia and normothermia means of improving outcomes in neurologically challenged patients have been a continued topic of discussion in the critical care literature.
- The need for a collaborative approach to best facilitate targeted temperature management (TTM) strategies and to minimize potential complications is warranted.
- Evidence-based standardized protocols are lacking; thus, initiation of treatment might be delayed by nurses' knowledge of fever management as well as individual physician orders.

INTRODUCTION

The American Heart Association's supported practice of therapeutic hypothermia (32°C–34°C for 12–24 hours) in out-of-hospital adult post–cardiac arrest comatose patients, as well as research on controlled hypothermia for neonates with hypoxic/ischemic encephalopathy, provides for key positioning in improving neurologic outcomes based on controlling temperature postinsult.[1,2] Based on the premise that brain cells die due to complex biochemical processes and postinflammatory cascade, utilization of TTM protocols, both induced hypothermia (32°C–34°C) and controlled normothermia (36°C–37°C),[3] as a means of improving outcomes in neurologically challenged patients has been a continued topic of discussion.

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FEVER

Fever is a normal cytokine-mediated immunologic reaction to infection or inflammation; however, hyperthermia in brain-injured patients can also be as the result of dysregulation within the hypothalamus aberrantly shifting the set point, which can also lead to secondary brain injury.[4,5] Healthy adult brains have the ability to tolerate fluctuations in temperature; however, the same variations in temperatures in compromised brains have been shown to increase ischemia and injury.[6]

There are 3 major reasons for elevated temperature in critically ill patients: infectious fever, noninfectious fever (including neurogenic fever, thrombophlebotic events, transfusion reactions, and drug fevers), and hyperthermic events (including malignant hyperthermia), with identification of a majority of fevers in patients with brain injury attributable to a pulmonary source.[4] In 1 of 5 to 1 of 3 of cases, the cause of fever remains unexplained despite aggressive work-up, leading to a belief that the cause may be central in origin.[7]

The underlying mechanism of these phenomena is thought due to several coexisting processes. Acute stress situations (such as response to injury), which in turn elevate body temperature, lead to stimulation of the autonomic centers within the right insular cortex, amygdala, and hypothalamus, which increases sympathetic outflow.[8] This heightened sympathetic response leads to increased heart rate, minute ventilation, oxygen consumption, and resting energy consumption. This concept can also be linked to increasing cerebral metabolic demand, which further stresses the injured brain, predisposing it to secondary injury. It has been hypothesized that for each 1°C increase in core body temperature, a corresponding 7% to 13% increase in cerebral metabolism may be experienced.[9] This increased metabolic demand at the cellular level coupled with diminished blood flow from injury further exacerbates the situation.[6] Fever is also known to increase the inflammatory process, which compromises the blood-brain barrier, leading to cerebral edema and neuronal death, as well as increasing production of free radicals due to excessive catecholamine release, inducing calcium influx and prolonged cellular excitation as a result of glutamate release.[10]

On a fundamental level, much debate has surrounded defining temperature measurement methodology. Normothermia has been described as 37°C, factoring in diurnal variations up to 1°C and acknowledging that axillary and oral temperatures are slightly less than core temperature but more easily obtainable than rectal, bladder, esophageal, or pulmonary artery catheter temperatures.[10,11] Definitions of "fever" include temperatures varying between 37.1°C and 38.5°C, with many suggesting intervention for any temperature greater than 37.5°C,[5] noting the importance of acknowledging that brain temperature is often higher than core body temperature and, as patients experience a febrile condition, the disparity between brain and core body temperatures increases.[12] This leads to belief that the diagnosis of fever in neurologically impaired populations may be underestimated and the treatment underused.

Neuroscience critical care research has suggested that after controlling for diagnosis, severity of illness, age, and complications, elevation in body temperature consistently can be linked to longer length of stay, higher mortality, and worse economic and functional outcomes.[13] Controlling fever can lead toward establishment of normal body physiology and lead toward a goal of recovery.

HISTORICAL PERSPECTIVE

The history of cooling patients for medical treatment was initiated by several early clinicians. In 1766, John Hunter, a physiologist, investigated animals exposed to extreme