Considerations for Neuroprotection in the Traumatic Brain Injury Population

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KEYWORDS

• Trauma • Brain injury • Neuroprotection • Cooling • Craniectomy

KEY POINTS

- The brain undergoes complex pathophysiologic changes following a traumatic injury, and efforts should be made to decrease the amount of secondary injury.
- There is evidence to support the use of cooling, craniectomy, and medications as neuroprotective measures to save the brain following traumatic injury.
- Cooling for all persons with severe traumatic brain injury is not supported in the evidence; however, cooling may be beneficial to reduce intracranial pressure.
- Craniectomy may be beneficial in the management of increased intracranial pressure; however, current research does not support improved outcomes for patients, and further research is needed.
- Severe traumatic brain injury is associated with a high rate of death or disability; nurses are key in monitoring and treating secondary brain injury in efforts to save the brain.
- Further research is needed to improve knowledge of neuroprotection following traumatic brain injury.

INTRODUCTION

Traumatic brain injury (TBI) is a contributing factor in approximately 30% of all injury-related deaths in the United States.[1] The Centers for Disease Control and Prevention estimated the direct and indirect cost of TBI in 2010 to be $76.5 billion, with the medical care for those with severe TBI accounting for 90% of those costs. Approximately 5.3 million Americans are living with a disability as a result of TBI, affecting all aspects of their life and their ability to function as contributing members of society.[1]

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TBI damages the neurons of the brain in both direct and indirect ways. Direct damage is caused by the initial injury compressing, twisting, or otherwise damaging the neuron, resulting in neuronal dysfunction or neuronal death. The aims of medical and nursing therapies for management of brain injury are to limit the effects of secondary brain injury, which is caused by a myriad of cellular and pathophysiologic mechanisms. Secondary injury is triggered by the initial injury and then leads to further brain damage as a result of altered cerebral blood flow, altered cerebrovascular autoregulation, changes in cerebral metabolism, and low cerebral oxygenation. Further damage to neurons can occur by excessive release of glutamate, an excitatory neurotransmitter, and the influx of calcium and sodium into the intracellular space.[2] Minimizing the effects of the secondary injury to the brain following the initial injury is the focus of nursing care in the days to weeks following the trauma.

Neuroprotection is a concept that health care providers use to optimize treatments in such a way that the neuronal damage or loss is minimized, and patient outcomes are improved. This article summarizes areas in which research has previously supported or refuted the brain-protecting efforts of procedures or medications.

COOLING

Therapeutic hypothermia has been studied for more than a decade as a neuroprotective strategy for patients after cardiac arrest.[3] The 2011 National Institute for Health and Clinical Excellence guidelines support the use of hypothermia to 32°C to 34°C for 12 to 24 hours following cardiac arrest with slow rewarming to improve neurologic outcome for comatose patients with return of spontaneous circulation.[4]

The pathophysiology of cooling as a neuroprotective mechanism is complex and involves a multitude of responses at the cellular level to the ischemia or injury. This article provide an overview of the pathophysiology in order for nurses to better understand the background of why cooling brain-injured patients may or may not be beneficial. Ischemia or injury to the brain causes the release of excitatory amino acids and glutamate. Neuronal exposure to the excess levels of these substances causes inflammation, edema, and accelerated cell death. In addition, ischemia or injury to the neuron causes a deficit in the oxygen, ATP, and glucose needed for normal cellular function. Hypothermia is able to decrease cerebral metabolic rate as well as reduce the inflammatory response, thus protecting the brain by decreasing the secondary injury.[5,6]

With the promising trials supporting the use of therapeutic hypothermia for the post-arrest population, it became an area of interest to determine whether the TBI population would also have improved outcomes with the use of cooling. Several factors come into play when assessing the quality of studies supporting or refuting the effects of therapeutic hypothermia in the brain-injured population, such as when to cool (prophylactic vs targeted management if intracranial pressure [ICP] is increased), what methods to use to cool (superficial vs intravascular), how long to cool, what temperature is best, how to rewarm, and which combinations of these factors produce the best outcomes. This article uses the therapeutic moderate hypothermia range of 32°C to 35°C, because most studies are within this range.

Two large multicenter trials were conducted to determine the effect on early hypothermia after severe brain injury, targeting overall hypothermia as a neuroprotectant and not as a treatment of ICP. These trials were the North American Brain Injury Study: Hypothermia I and II.[7,8] Patients were randomized to cooling to 35°C, 33°C, or normothermia with standard of care for severe TBI. Patients were cooled quickly (<6 hours to

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