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Analgesia, sedation, and neuromuscular blockade during targeted temperature management after cardiac arrest

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The approach to sedation, analgesia, and neuromuscular blockade during targeted temperature management (TTM) remains largely unstudied, forcing clinicians to adapt previous research from other patient environments. During TTM, very little data guide drug selection, doses, and specific therapeutic goals. Sedation should be deep enough to prevent awareness during neuromuscular blockade, but titration is complex as metabolism and clearance are delayed for almost all drugs during hypothermia. Deeper sedation is associated with prolonged intensive care unit (ICU) and ventilator therapy, increased delirium and infection, and delayed wakening which can confound early critical neurological assessments, potentially resulting in erroneous prognostication and inappropriate withdrawal of life support. We review the potential therapeutic goals for sedation, analgesia, and neuromuscular blockade during TTM; the adverse events associated with that treatment; data suggesting that TTM and organ dysfunction impair drug metabolism; and controversies and potential benefits of specific monitoring.

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We also highlight the areas needing better research to guide our therapy.

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Targeted temperature management (TTM) is now a standard treatment for hypoxic–ischemic encephalopathy after cardiac arrest, but many components, including the approach to sedation, analgesia, and neuromuscular blockade (NMB), remain unproven or untested. Providing comfort and safety for any intensive care unit (ICU) patient can be challenging because therapeutic goals vary over time, by disease state, and by patient context with individualized goals; patients treated with TTM pose even greater challenges. The goals of care shift dramatically over time during TTM, from amnesia and shivering control with NMB therapy requiring moderate or deep sedation, to calm wakefulness and ability to assess unsedated neurologic status after rewarming, all within a short period of time. Furthermore, derangements in drug clearance resulting from organ dysfunction and TTM itself can greatly prolong medication effects, making these therapeutic choices even more complex. Several observational studies have suggested that neurologic prognostication and decisions to withdraw life support due to presumed severe hypoxic–ischemic encephalopathy may take place too soon after TTM, and raised concerns that lingering sedative effects may confound these critical examinations [1,2].

The first review of sedation, analgesia, and NMB during TTM concluded that few studies described their therapeutic strategies, and reported widely varying doses and approaches [3]. A 2015 report from the Institute of Medicine investigated the treatment of cardiac arrest, and, in more than 400 pages, mentioned sedation twice, and analgesia and NMB once each [4]. It is important to note that this document made no recommendations for this important aspect of treating cardiac arrest patients, nor the need to research better sedation protocols. Clearly, additional work is warranted to define the best approach to this challenging aspect of TTM.

This chapter focuses on four concepts for adult patients being treated with TTM after cardiac arrest:

- Review the goals for sedation, analgesia, and NMB during TTM.
- Identify the risks, controversies, and limited evidence to guide this practice.
- Review specific medications.
- Discuss the options available to minimize negative effects of therapy by monitoring.

In addition, it makes recommendations for two related areas:

- Suggest specific outcomes to include in future studies.
- Propose research needs and topics.

Therapeutic goals during TTM

Among general intensive care unit (ICU) patients, the primary goals when providing sedation vary based on patient severity of illness, particular injuries or diseases, and specific therapies being administered. In 2013, the most recent guidelines for treating ICU pain, agitation, and delirium (PAD) were published, including 472 references and >50 recommendations [5]; unfortunately, the practice of TTM was not included in these PAD guidelines. Light sedation was strongly recommended because of clearly improved outcomes in comparison to deeper sedation, but light sedation is not applicable to TTM patients because of shivering and frequent or continuous NMB. Many possible goals for sedation, analgesia, and NMB for TTM patients can be proposed, including preventing discomfort associated with TTM, reducing shivering and the associated systemic metabolic demands, reducing cerebral oxygen demands and brain metabolic activity, suppressing or preventing seizures, preventing awareness during NMB, minimizing drug-induced adverse effects (including hypotension, delayed ventilator weaning and mobilization, pneumonia, delirium), and confounding the critical post-TTM neurological examination due to prolonged sedative effects.

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