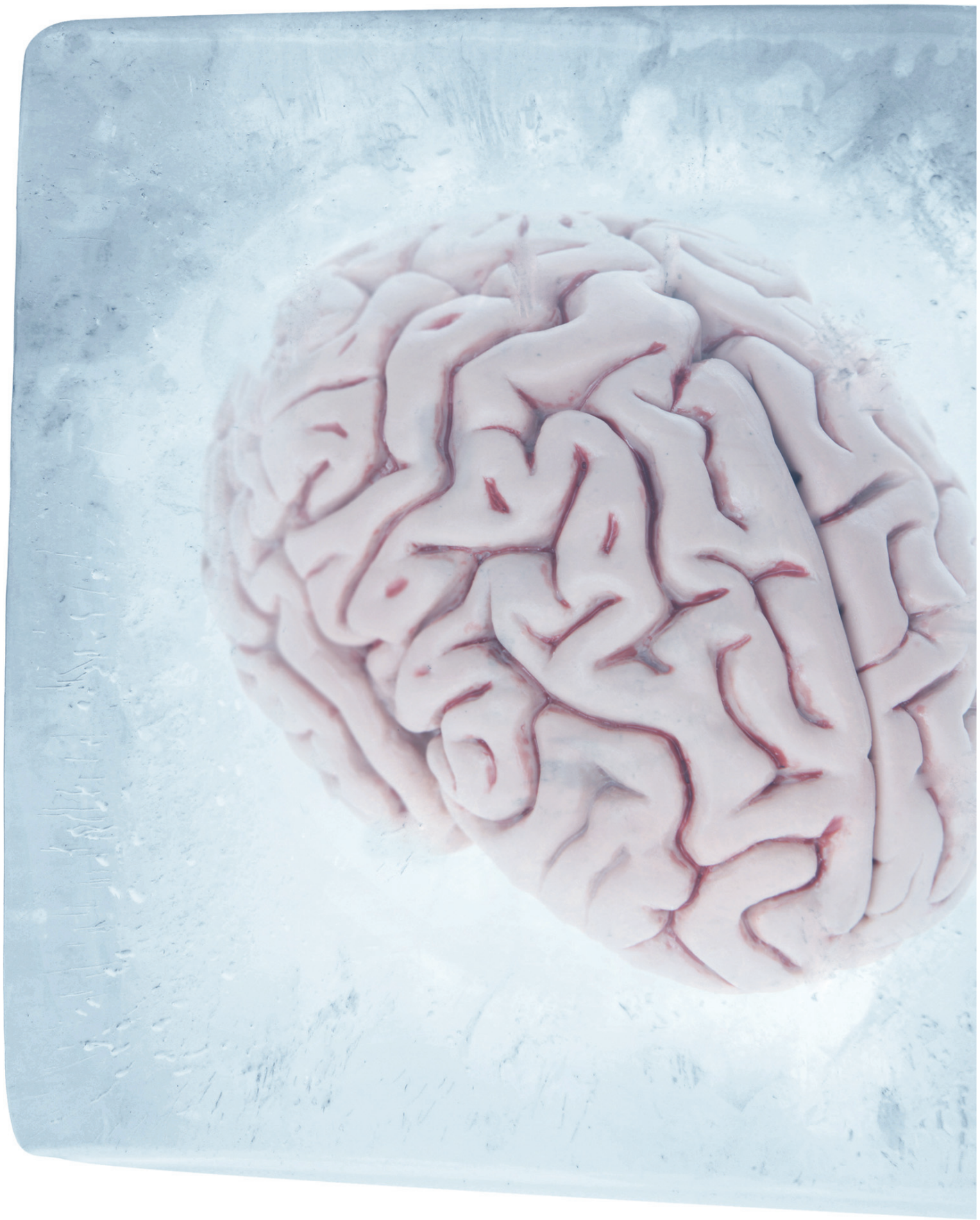

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The cold truth about postcardiac arrest targeted temperature management: 33°C vs. 36°C

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Abstract: This article provides nurses with up-to-date evidence to empower them in contributing to the 33°C versus 36°C discussion in postcardiac arrest targeted temperature management (TTM). Presented in debate format, this article addresses the pros and cons of various target temperatures, examines the evidence around TTM, and applies it to clinical scenarios.

Keywords: cardiac arrest, comatose, cooling, rewarming, targeted temperature management, therapeutic hypothermia, TTM

AN ADULT PATIENT is admitted to the ICU. He was resuscitated from a cardiac arrest in the ED 20 minutes ago after presenting with chest pain. His vital signs are stable, but he does not have a meaningful response to verbal commands.[1] The resident physician suggests targeted temperature management (TTM) for neuroprotection, and a conversation begins about the relative risks and benefits of the various target temperatures.

This article provides nurses with the latest TTM evidence and empowers them to discuss the 33°C (91.4°F) versus 36°C (96.8°F) question. Additionally, it weighs the pros and cons of target temperatures, examines the evidence surrounding TTM, and applies it to clinical scenarios.

Evidence supporting TTM

TTM is an evidence-based therapy for patients who have experienced a cardiac arrest and who are unresponsive, or comatose, without purposeful movements or the ability to follow simple verbal commands.[1] The therapy has been associated with improved neurologic outcomes after cardiac arrest, and national and international guidelines currently reinforce the use of TTM in patients who experience neurologic injury after cardiac arrest.[1-6]

Comatose survivors of cardiac arrest have a high risk of death and poor neurologic function. TTM provides neuroprotection for hypoxic-ischemic brain injuries sustained during cardiac arrest to improve survival with good neurologic function.[7] The American Heart Association's (AHA) 2015 Cardiopulmonary Resuscitation (CPR) Guidelines state that any comatose patient who achieves return of spontaneous circulation (ROSC) after a cardiac arrest should be treated with TTM and maintained at a constant temperature between 32°C and 36°C for at least 24 hours.[1] Although the AHA strengthened its recommendation for the use of temperature management, the guidelines still allow for provider preference when choosing a temperature. So, why is there a range of target temperatures to choose from?

In 2002, two randomized controlled trials were published, showing that patients who experienced a cardiac arrest and were cooled to a target temperature of 32°C to 34°C had better survival with good neurologic recovery than those not in the intervention arm.[2,3] In these studies, neurologic outcomes were evaluated in addition to survival because neurologic recovery is a more patient-centered outcome and a more desired result of therapy than simply surviving.

The Cerebral Performance Category (CPC) is commonly used to define neurologic function after cardiac arrest. The CPC is used to differentiate "good" neurologic outcomes from "poor" neurologic outcomes. A "good" neurologic outcome is characterized by a CPC score of 1 (no or mild disability, such as in patients who are able to lead normal lives with mild deficits) or 2 (moderate disability, such as in patients who can perform activities of daily life independently and work part-time in a sheltered environment). A "poor" neurologic outcome is described by CPC categories 3 (severe disability), 4 (persistent vegetative state), and 5 (brain death).[8,9] Based on the significant benefit for patients shown in the two 2002 trials, "mild hypothermia" therapy became the standard of practice.

In 2013, another randomized controlled trial was published showing that neurologic outcomes were the same whether patients were cooled to target temperatures of 33°C or 36°C, regardless of the initial rhythm during the cardiac arrest.[4] After this trial, many hospitals worldwide quickly adopted the new findings by targeting 36°C in what would become known as TTM.[10,11] The current evidence appears to support two temperatures for patients after cardiac arrest, and practice is known to be variable. So which is the better target temperature?

The case for 33°C

The aforementioned 2002 landmark studies used 33°C as the target temperature.[2,3] The near twofold increase in survival with good neurologic recovery observed in these two studies was groundbreaking with respect to postcardiac arrest care. Researchers found a protocol that would achieve better neurologic recovery, with a CPC of 1 or 2 in patients who experienced an out-of-hospital cardiac arrest, a group that previously had dismal outcomes.

After the 2002 trials, early adopters applied TTM internationally to patients who had experienced a cardiac arrest, resulting in a body of literature that further supported the initial findings.[12,13] One before-and-after study by Oddo and colleagues found that implemening TTM at 33°C resulted in a 55.6% survival rate with good neurologic recovery in patients with out-of-hospital ventricular fibrillation (VF) cardiac arrest, compared with 25.6% of the historical cohort treated with standard therapy (no TTM).[12] In a review of studies that addressed the efficacy of TTM in clinical environments, Sagalyn and colleagues found that, in six studies with an overall total of 1,004 patients, the therapy achieved a higher rate of good neurologic recovery compared with standard therapy (odds ratio [OR] 2.5, 95% confidence interval [CI]: 1.9–3.4).[13]

Though initial studies specifically targeted the utility of therapeutic hypothermia in patients who initially presented with shockable rhythms, such as VF or pulseless ventricular tachycardia, the evidence supports this therapy for patients with all initial rhythms. In the first randomized controlled trial to test the utility of hypothermia (33°C) in patients with cardiac arrest and nonshockable rhythms (pulseless electrical activity and asystole), Lascarrou and colleagues found that 10.2% of patients receiving TTM at 33°C survived with a good neurologic recovery, compared with 5.7% of patients receiving targeted normothermia at 37°C (P = .04).[5] Some observational studies have also found that patients with nonshockable initial rhythms benefit from therapeutic hypothermia between 32°C and 34°C.[14-16]

Given the early randomized controlled trials and the wealth of observational data, multiple studies have demonstrated that TTM at 33°C benefits neurologic recovery for comatose survivors of cardiac arrest. This places temperature management as a mainstay of therapy within the recommended guidelines for postcardiac arrest care.[1] Because healthcare professionals are unable to measure the extent of hypoxic-ischemic brain injury after cardiac arrest, why reduce the dose of therapy by using a warmer target temperature? Clinicians have only one chance to impart neurologic protection to this patient population. Is it fact or misperception to opt for warmer temperatures for patients who have experienced a cardiac arrest?

The case for 36°C

The concept of using 36°C as an alternative target for TTM was introduced in November 2013 with the publication of the TTM Trial.[4] Nielsen and colleagues randomized 950 patients who had out-of-hospital cardiac arrests to be cooled to either 33°C or 36°C. In the 2002 Mild Therapeutic Hypothermia to Improve the Neurologic Outcome after Cardiac Arrest (HACA) Trial, many patients in the standard treatment (normothermia) group developed fevers, so it was unclear whether the outcome benefits were due to the hypothermia (33°C) or to the prevention of fever.[2] The TTM Trial was designed to prevent fever in both groups and isolate the effects of the temperature.

The important finding from this study was that there was no difference in survival or neurologic outcomes between patients maintained at 33°C and those maintained at 36°C. Further, the patients' outcomes were similar to the those achieved in the hypothermia group in the 2002 trial.[4] This randomized controlled trial provided evidence that 36°C is a valid choice as a target temperature in patients eligible for TTM, with equivalent outcomes to the 33°C target temperature.

So, why consider targeting 36°C instead of 33°C? The rationale is that it can provide neurologic and survival benefits while avoiding some of the adverse reactions that occur during hypothermia. In the TTM Trial, patients maintained at 36°C experienced significantly less hypokalemia. Hypothermia drives potassium from the blood into cells, and the reverse happens when a patient is rewarmed. Hypo- and hyperkalemia can each contribute to dysrhythmias, so fewer electrolyte shifts may be safer for patients who are at risk for dysrhythmias.

Using a target temperature of 36°C may allow patients experiencing cardiac arrest to receive TTM who may otherwise have been disqualified from the therapy, such as those with major bleeding. Although randomized controlled trials indicate no difference in the rate of bleeding between the two temperature targets, patients with bleeding or known coagulopathy were not included in several major studies.[4] Clinicians may hesitate to initiate TTM in patients who are bleeding. One observational study revealed less bleeding at a warmer target temperature, indicating that a target of 36°C may be a safer choice in this patient population.[17]

Another adverse reaction of hypothermia is bradycardia. In most patients receiving TTM, the bradycardia is not considered severe. However, a warmer target temperature of 36°C could be safer in patients for whom bradycardia poses independent risk factors, such as those with a prolonged corrected QT (QTc) interval. Prolongation of the QTc interval places patients at risk for torsades de pointes, a lethal ventricular dysrhythmia. Both hypokalemia and bradycardia can further prolong the QTc interval and increase the risk of torsades de pointes, making 33°C potentially unsafe for this patient population.

Clinicians may also consider a goal temperature of 36°C in patients with profound hemodynamic instability, such as those with refractory septic shock requiring multiple vasopressors. To date, no definitive data support that patients with profound hemodynamic instability fare better at 36°C, but patients with this degree of instability may concern clinicians as they discuss temperature selection. These clinicians may opt to avoid possible temperature fluctuation and fluid shifts that could be more detrimental to an already unstable patient.

The introduction of a 36°C option for TTM has opened the door to neuroprotection for patients with cardiac arrest who otherwise may not have received the therapy. Patients who are candidates for TTM after a cardiac arrest from a neurologic standpoint but who have other comorbidities such as bleeding, bradycardia, or dysrhythmias that may have initially excluded them, may receive TTM more safely at 36°C, increasing their chances of a good neurologic recovery (a CPC score of 1 or 2).

Common misperceptions

Several misunderstandings are related to both target temperatures. Many of those surrounding 33°C focus on adverse reactions; for example, concern that patients may encounter fatal dysrhythmias at such a cold temperature. This, in fact, is not true. Studies have shown no difference in the occurrence of malignant dysrhythmias between the two temperatures, and concerning dysrhythmias become prevalent only when the temperature of the myocardium is lower than temperatures recommended for TTM.[2-5,18] Bleeding risk is also cited as a concern for dropping the core temperature to 33°C, but there was no difference in bleeding risk between the two temperatures in the patients included in the TTM Trial. Further, most bleeding risks associated with TTM in patients with a cardiac etiology are minimal and not fatal, such as bleeding from line insertion sites.[4]

Another misperception is that 36°C will be easier to maintain than 33°C, but this is simply not true.[19] The evidence shows that 36°C is safe, but that does not mean it is any less intense as a protocol. For example, clinicians may claim they do not need a device or drugs to maintain 36°C as long as no fever occurs. The TTM Trial does not support this claim, and the 36°C group received active temperature management, not just fever prevention. In the trial, both temperature groups received active temperature management at their target temperature for 24 hours, followed

by gradual controlled rewarming, and then normothermia below 37.5°C for 72 hours.[4] Targeting 36°C does not make the protocol faster or reduce length of stay compared with a target of 33°C.

Additionally, targeting 36°C does not reduce the amount of shivering. Shivering, even microshivering—which may be difficult to detect without specialized monitoring—can have a detrimental impact on brain oxygenation and should be avoided.[20,21] In the TTM Trial, there was no significant difference in the percentage of patients who experienced shivering: 30% of patients at 33°C and 34% of patients at 36°C.[4] Patients may need more aggressive shivering management at 36°C because the average shiver threshold has been identified between 34°C and 36°C (mean 35.5°C).[22] Patients at 36°C may be more prone to shivering, and those at 33°C are at the lower end of the shiver threshold, so assessment and treatment of shivering remain essential.[23]

Data from Australia underscore the importance of adequate sedation when targeting a temperature of 36°C. After changing the target temperature from 33°C to 36°C, researchers found that patients spent less time within their target temperature range and had higher rates of fever.[11] Fever contributes to poor neurologic outcomes, so these trends make it clear that it is crucial to manage the temperature closely if 36°C is the target.[24]

Another misperception is that a target temperature of 36°C will require fewer sedatives for the patient, but the TTM Trial does not support this either. Both temperature groups received the same pharmacologic protocol in the trial, which included mandatory sedation for 36 hours during cooling and rewarming. The quantity of sedatives used was not reported, so there is no evidence that targeting 36°C does or does not require fewer sedatives. The trial data showed no difference between groups in the number of days that sedation affected neurologic evaluation.[4]

Additionally, the notion that clinicians can conduct ongoing neuroprognostication earlier if the patient is at 36°C instead of at 33°C is an error. A patient on any TTM protocol, no matter the target temperature, should be sedated and may be receiving neuromuscular blockade. Patients cannot undergo valid accurate neuroprognostication until 72 hours after arrival at normothermia, and some might suggest even later given the observation that many patients have delayed awakening.[1,25,26]

The most dangerous misperception surrounding the 33°C versus 36°C debate would be not using TTM at all or replacing it with passive temperature strategies. The introduction of 36°C as a target temperature brought with it a statistically significant decrease in the use of active cooling methods for patients with cardiac arrest in the US, possibly reflecting the misperception that 36°C is not active TTM.[27] Active temperature management remains key in giving patients a better chance for a good neurologic outcome after cardiac arrest.

Case studies

Evaluate the patients in the following two clinical scenarios, and decide if the patient needs TTM. If so, would they benefit most from a target temperature of 33°C or 36°C?

Case study 1. A 56-year-old man found unresponsive at home was just admitted to the ICU. Upon EMS arrival, he was in pulseless electrical activity with agonal respirations. EMS initiated CPR and intubated the patient in the field. In the ED, the patient received 20 minutes of CPR and four rounds of epinephrine. ROSC was achieved with an irregular wide complex tachycardia that was defibrillated to sinus rhythm. Now in the ICU, the patient has a Glasgow Coma Scale score of 4 and is not following simple verbal commands. His initial temperature is 31.5°C.

Considering the evidence-based guidelines, which of the three options below would be most appropriate and why?

- Option 1: Do not initiate TTM.
- Option 2: Initiate TTM at 33°C.
- Option 3: Initiate TTM at 36°C.

Option 1: Do not initiate TTM. This would not be an evidence-based choice. The patient has ROSC, but he is lacking meaningful response to verbal commands. To comply with the 2015 AHA Guidelines, TTM should be implemented. Also, new evidence in patients presenting with nonshockable initial rhythms indicates TTM is beneficial.[5]

Option 2: Initiate TTM at 33°C. This would be a good option, as current evidence supports the use of 33°C for patients presenting with nonshockable rhythms.[5] Also, the patient is already at 31.5°C, making it more efficient to get him to 33°C than 36°C. This patient does not have any contraindications to 33°C; for example, his QTc interval is not prolonged and he has no reported bleeding issues, so remaining at 33°C is a good option.

Option 3: Initiate TTM at 36°C. This could be a more difficult target temperature to choose because clinicians would technically have to rewarm the patient to "cool" him. Guidelines suggest rewarming at a rate of 0.25°C to 0.5°C per hour to avoid electrolyte shifts and cerebral edema, so it would take 9 to 18 hours before the patient safely reaches the target temperature.[1,6] The patient would be going through the shiver zone (34°C to 36°C), so more sedation and paralytics may be needed to control shivering. Although this target temperature choice would be suboptimal, it is still better than not cooling at all.

Case study 2. A 42-year-old man is receiving a nitroglycerin infusion in the ICU. He has construction

gear with him and says he was at work when he began experiencing chest pain. He becomes diaphoretic, clammy, and disoriented. The ECG shows acute ST-elevation myocardial infarction. The patient stops talking and VF shows on the monitor. CPR is initiated, and the patient is defibrillated. After 10 minutes of advanced cardiac life support, including epinephrine administration, the patient achieves ROSC. He is unresponsive, and the ECG now shows bradycardia with prolonged QTc. His initial temperature is 35°C.

Considering the evidence-based guidelines, which of the three options below would be most appropriate and why?

- Option 1: Do not initiate TTM.
- Option 2: Initiate TTM at 33°C.
- Option 3: Initiate TTM at 36°C.

Option 1: Do not initiate TTM. Patients with in-hospital cardiac arrest are included in the recommendations to receive TTM if they are unresponsive after ROSC.[1] The evidence for this patient population is of lower quality, but the pathophysiology of a hypoxic-ischemic brain injury would be the same no matter the location of the cardiac arrest.[1]

Option 2: Initiate TTM at 33°C. This would be a higher-risk option. Bradycardia with a prolonged QTc puts the patient at risk for torsades de pointes and cooling him to 33°C could potentially worsen the bradycardia. A target temperature of 33°C has a higher incidence of hypokalemia, which puts the patient at greater risk for dysrhythmias.

Option 3: Initiate TTM at 36°C. This is the optimal choice because of the risks outlined in Option 2. Evidence shows that 36°C provides equivalent outcomes to 33°C for patients whose arrest has a presumed cardiac cause.[4] Because this patient presented at 35°C, it is important to increase the patient's temperature by only 0.25°C to 0.5°C per hour until 36°C is reached. This slow controlled rewarm helps prevent cerebral edema and electrolyte shifts.

Conclusion

There are many variables to consider when selecting 33°C or 36°C as a target temperature after cardiac arrest, and numerous misperceptions surround both temperatures. Although some patients may be better managed at one temperature over the other, evidence supports each as safe and effective therapies.

Although there is variation in practice when choosing a target temperature of 33°C or 36°C for patients after cardiac arrest, they should still receive TTM, which requires active temperature management using a cooling device and tight temperature control. A target temperature of 33°C or 36°C should be chosen and maintained for at least 24 hours.[1] Patients at either temperature could experience shivering or seizures. Use a shivering protocol with medications to prevent and treat shivering and microshivering, ensuring that shivering is assessed frequently and treated aggressively during TTM.[20] Additionally, evaluate and treat seizure at either temperature, as this could also be detrimental to recovery. Meticulous nursing care, including skin care (especially skin in contact with surface cooling devices), positioning, and infection prevention, is important regardless of the target temperature.

After maintaining a goal temperature of 33°C or 36°C for 24 hours, the patient should be slowly rewarmed to normothermia.[1] When normothermia is achieved, it should be maintained and fever prevented for 72 hours after ROSC.[1,4,6] Fever after cardiac arrest may have adverse neurologic outcomes.[24] Neuroprognostication should occur no earlier than 72 hours after returning to normothermia.[1] This time frame could be longer if sedation or neuromuscular blocking agents confound the neurologic exam.

Questions related to optimal temperature for TTM continue to be investigated. Future studies are planned to further elucidate the appropriate temperature, the best way to deliver therapy, the optimal duration, and how to best rewarm patients. Research continues to focus on advancing postcardiac arrest care to ensure improved outcomes from this devastating event.

Nurses are instrumental in executing TTM therapy effectively. Regardless of the chosen target temperature, the keys to success are tight temperature control, the use of a shivering protocol, a slow controlled rewarm, and fever prevention. Clinicians have one chance to protect the brain after cardiac arrest, so evidence-based practices must be followed to achieve optimal patient outcomes. ■

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