



Nursing Management and Outcomes in Patients with Hypothermia: A Clinical Perspective

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Abstract

Background: Hypothermia, a dangerous drop in core body temperature below 35°C, is a significant clinical concern across diverse environments and patient populations. It results from an imbalance between heat loss and the body's capacity for thermogenesis, with risk factors including environmental exposure, extremes of age, comorbidities, and iatrogenic causes in healthcare settings.

Aim: This article provides a comprehensive clinical perspective on the nursing management and outcomes of patients with hypothermia, emphasizing the critical role of nurses in early recognition, pathophysiology-based intervention, and interprofessional coordination to optimize patient survival and recovery.

Methods: A detailed review of the etiology, epidemiology, and pathophysiology of hypothermia is presented. This foundation informs a systematic approach to evaluation (history, physical, accurate core temperature measurement, and diagnostics) and a staged management strategy, from passive external rewarming for mild cases to advanced extracorporeal techniques for severe, unstable patients.

Results: Successful outcomes are directly linked to prompt prevention of further heat loss, gentle handling to avoid arrhythmias, and the selection of appropriate, controlled rewarming modalities. Complications such as cardiac instability, coagulopathy, and metabolic derangements are common and must be anticipated. Mortality remains high in severe cases, though outcomes are significantly improved with rapid, protocol-driven care.

Conclusion: Effective management of hypothermia requires a rigorous, physiology-informed approach. Nurses are pivotal in early detection, initiating core interventions, and coordinating interprofessional care to mitigate organ dysfunction and improve survival across the care continuum.

Keywords: Hypothermia, Rewarming, Thermoregulation, Nursing Management, Core Temperature, Cardiac Arrhythmia, Extracorporeal Membrane Oxygenation, Patient Outcomes

Introduction

Hypothermia is a pathophysiological state defined by a core body temperature below 35 °C and remains a significant clinical concern across climates, with heightened incidence in colder geographic regions and during cooler seasons but with cases occurring even in temperate environments due to exposure, immersion, environmental disasters, or iatrogenic factors such as perioperative heat loss [1]. Its relevance to nursing practice is broad, encompassing prehospital care, emergency and critical care, perioperative services, neonatal and pediatric units, and community health. Because hypothermia affects virtually every organ system, clinical manifestations range from shivering and tachypnea to altered mental status, cardiac dysrhythmias, and coagulopathy, with presentations closely tied to both the absolute temperature and the rate of cooling [1]. Early recognition by nurses—often the first to observe subtle deviations in thermoregulation, sensorium, or skin findings—is essential for preventing progression

to more severe stages and for triggering rapid, protocolized interventions. Thermoregulation is principally coordinated by the hypothalamus, which integrates afferent input from central and peripheral thermoreceptors and orchestrates efferent autonomic responses to preserve core temperature [2]. Initial cold exposure elicits increased muscle tone and elevations in basal metabolic rate (BMR), effectively doubling heat production under sympathetic drive; the onset of shivering further augments thermogenesis, raising metabolic activity two- to fivefold above basal levels [2]. In neonates—who lack mature shivering mechanisms due to incomplete nervous system development—non-shivering thermogenesis in brown adipose tissue becomes paramount. Thermogenin-mediated uncoupling of oxidative phosphorylation increases mitochondrial membrane proton leak, curtails ATP synthesis, and dissipates energy as heat, thereby safeguarding core temperature during transitional and early postnatal periods [2]. Systemic endocrine responses, including rises in thyroid

hormones, catecholamines, and adrenal glucocorticoids, complement sympathetically mediated peripheral vasoconstriction to limit radiant and convective heat losses, while behavioral strategies such as adding insulation, seeking shelter, or exercising further assist in maintaining normothermia [2].

Clinically, hypothermia severity is categorized by core temperature thresholds that guide risk stratification and management priorities: mild hypothermia corresponds to 32–35 °C (90–95 °F), moderate to 28–32 °C (82–90 °F), and severe to temperatures below 28 °C (82 °F) [1]. For field and rescue operations, the Durrer staging paradigm provides an operational framework to identify patients most likely to benefit from rewarming and resuscitation, integrating both temperature-based and clinical criteria to inform triage and transport decisions [3]. From a nursing standpoint, accurate assessment of core temperature (preferably esophageal, rectal, or bladder in appropriate settings), vigilance for arrhythmias during rewarming, prevention of afterdrop through gentle handling, and the judicious selection of passive versus active rewarming modalities are central to safe care across the continuum. Importantly, escalating hypothermia severity correlates with higher rates of complications—ranging from aspiration and infection to refractory shock and malignant dysrhythmias—and with increased mortality, underscoring the imperative for early detection, meticulous thermoregulatory support, and interprofessional coordination to optimize outcomes [4][5]. In summary, a rigorous understanding of hypothermia’s epidemiology, neuroendocrine thermoregulation, age-specific physiology, and temperature-based staging equips nurses to implement timely, evidence-informed interventions that mitigate organ dysfunction and improve survival across diverse care environments [1][2][3][4][5].

Depth of hypothermia	Core body temperature	
	Classic hypothermia scale	Hypothermia scale in trauma patients
Mild	35–32°C	36–34°C
Moderate	32–28°C	34–32°C
Severe	28–20°C	<32°C
Profound	20–14°C	
Deep	<14°C	

Fig. 1: Core Body Temperature.

Etiology

Hypothermia results when total body heat loss exceeds endogenous heat production, a mismatch driven by environmental exposure, impaired thermoregulatory control, or diminished metabolic heat generation. Central and peripheral nervous system mechanisms normally coordinate vasoconstriction, shivering, and behavioral

adaptations such as adding clothing or seeking shelter to preserve core temperature; disruption of these responses—whether neurologic, metabolic, or psychosocial—predisposes individuals to pathologic cooling [6][7]. The balance is further influenced by surface area-to-mass ratio, ambient humidity and wind, and the presence of moisture on the skin, all of which accelerate conductive and convective heat loss when compensatory mechanisms are blunted [6]. Insufficient heat production is common at the extremes of age, where neonatal non-shivering thermogenesis may be inadequate and older adults exhibit reduced basal metabolic rate and attenuated vasomotor responses. Metabolic derangements such as hypoglycemia and malnutrition limit substrate availability for thermogenesis, while endocrine disorders—including hypothyroidism and adrenal insufficiency—decrease metabolic heat output and compromise cardiovascular responses to cold stress [6][7]. Neurologic conditions that impair hypothalamic set-point function or efferent pathways, notably cerebrovascular accidents and neurodegenerative diseases, reduce shivering and vasoconstriction, compounding the risk of precipitous core temperature decline [7]. Cutaneous and vascular factors also contribute; inflammatory dermatoses (e.g., psoriasis), extensive burns, and wounds augment radiant and evaporative losses, and peripheral vasodilation—whether from autonomic dysfunction or pharmacologic agents—facilitates rapid heat dissipation [6].

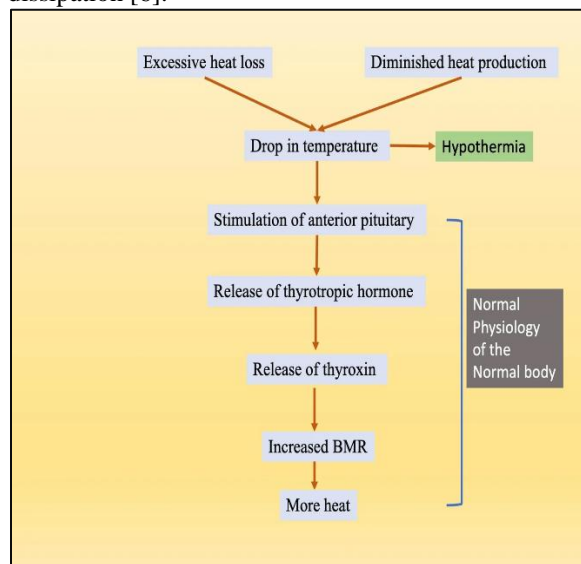


Fig. 2: Etiology of Hypothermia.

Iatrogenic hypothermia is a recognized hazard across perioperative and critical care settings. General and regional anesthetics blunt sympathetic tone and shivering thresholds; commonly used agents such as volatile anesthetics, propofol, and opioids decrease metabolic heat production while promoting vasodilation. Additional contributors include beta-blockers, meperidine, clonidine, neuroleptics, and alcohol, which suppress thermogenic drive, impair

behavioral responses, and exacerbate peripheral heat loss [7]. Fluid and blood product administration at ambient temperatures, exposure during lengthy procedures, and inadequate active warming further compound risk in vulnerable patients [6][7]. Beyond organic etiologies, behavioral and social factors are pivotal determinants. Cognitive impairment from dementia or substance use disorders can delay recognition of cold stress and hinder self-protective actions, while environmental deprivation—lack of adequate clothing, heating, or shelter—places individuals experiencing homelessness at sustained risk of exposure hypothermia, particularly during inclement weather and nocturnal hours [8][9]. Effective prevention therefore requires integrated strategies that address physiologic vulnerability, medication stewardship, perioperative thermal management, and upstream social determinants alongside timely clinical intervention [6][8].

Epidemiology

Hypothermia is an important, yet often underrecognized, contributor to morbidity and mortality across diverse environments. In the United States, surveillance estimates attribute approximately 700 to 1500 deaths annually to hypothermia, a figure that likely underrepresents the true burden because exposure-related deaths are frequently misclassified or obscured by comorbid injuries, intoxication, or cardiac events. The condition most frequently affects adults between 30 and 49 years of age, with occurrence reported to be roughly tenfold higher in men than in women, a pattern plausibly linked to occupational exposures, higher rates of outdoor recreation and risk-taking behaviors, and greater prevalence of alcohol or substance use in some male populations. Nevertheless, hypothermia's true incidence is unknown; many nonfatal events go unreported, and coding practices vary across jurisdictions. Beyond community exposure, clinically significant hypothermia also arises in healthcare settings—particularly during emergency resuscitation and perioperative care—further complicating incidence estimates. Vulnerable groups include individuals experiencing homelessness, people with psychiatric illness or cognitive impairment, those with substance use disorders, and patients with endocrine or neurologic conditions that blunt thermoregulatory responses. Geographic and seasonal factors modulate risk, with higher case counts in colder regions and winter months, yet cases occur year-round in temperate climates through immersion, weather extremes, and inadequate shelter. Even with optimized in-hospital management, the mortality of moderate to severe hypothermia still approaches 50%, underscoring the need for early recognition, prevention strategies, and coordinated prehospital and in-hospital systems to mitigate adverse outcomes.

Pathophysiology

The body's core temperature reflects the dynamic equilibrium between endogenous heat

production and heat dissipation to the environment, with normal homeothermy maintained between 36.5 and 37.5 °C through tightly regulated neuroendocrine and behavioral mechanisms. Heat leaves the body via four principal pathways—radiation, conduction, convection, and evaporation—whose relative contributions shift with environmental conditions, clothing, activity level, and skin moisture. Radiation denotes transfer of infrared electromagnetic energy from the skin to cooler surroundings; conduction refers to direct molecule-to-molecule transfer between the body and any contacting surface; convection is the removal of heat by moving fluids, typically ambient air; and evaporation describes latent heat loss as liquid (sweat or water on the skin) converts to vapor at the surface [10]. Under thermoneutral, dry conditions, radiation predominates. By contrast, clinically significant hypothermia more often develops when convective loss during cold air exposure, conductive loss during immersion in cold water, or accelerated evaporative loss from wet clothing or profuse sweating overwhelms compensatory thermogenesis [10]. In early cold stress, hypothalamic integration of peripheral and central thermoreceptor input triggers autonomic and endocrine responses that elevate metabolic heat production and preserve core temperature. Sympathetically mediated peripheral vasoconstriction reduces cutaneous blood flow, while increased ventilation and cardiac output support oxygen delivery to active thermogenic tissues. Shivering augments heat production by two- to fivefold over basal metabolic rate, but sustained cooling blunts central shivering thresholds and depletes energy substrates, precipitating “shivering exhaustion” without external rewarming [11]. Progressive hypothermia impairs membrane ion channel function and enzymatic kinetics, slows conduction velocity, and depresses central nervous system activity, leading to bradypsychia, ataxia, and coma. Cardiovascular instability emerges as sinus bradycardia gives way to atrial dysrhythmias and decreased myocardial contractility; further cooling destabilizes the sinoatrial and atrioventricular nodes and predisposes to malignant ventricular arrhythmias, including ventricular fibrillation [11]. Concomitantly, cold diuresis and impaired renal concentrating ability promote volume depletion; insulin secretion and peripheral glucose utilization decline, fostering hyperglycemia; and coagulation becomes deranged through platelet dysfunction and enzyme kinetics, amplifying bleeding risk. Rewarming introduces additional hazards: peripheral vasodilation and return of cold, acidic blood from the extremities can produce “afterdrop,” exacerbating hypotension and arrhythmia susceptibility unless managed with controlled, staged techniques [11]. Collectively, these temperature-dependent perturbations across neurologic, metabolic, and cardiac systems explain the rapid progression from compensatory responses to multisystem failure in severe hypothermia and

underscore the necessity of prompt thermal control and cautious rewarming strategies [10][11].

History and Physical

Hypothermia may culminate in cardiorespiratory arrest; therefore, initial evaluation must prioritize life-threatening conditions using a structured primary survey. A rapid assessment of airway, breathing, circulation, disability, and exposure (ABCDE) should immediately identify the need for ventilatory support, circulatory resuscitation, and controlled rewarming. Unconscious, pulseless, and apneic patients require prompt initiation of resuscitative measures irrespective of the underlying cause, with concurrent attention to limiting further heat loss through insulating layers, warmed oxygen, and warmed intravenous fluids. Once the patient is stabilized—or if immediate life-threats are excluded—the clinician transitions to a focused secondary survey to delineate the history of exposure, contributory comorbidities, and the extent of systemic involvement. In many cases, a clear history of significant cold exposure is elicited; however, in older adults, individuals with cognitive impairment, or those with substance intoxication, the exposure timeline may be incomplete, necessitating a high index of suspicion and objective confirmation of the core temperature [12]. Accurate thermometry is central to diagnosis and staging. Because management decisions hinge on precise core temperature thresholds, the measurement method must be selected with regard to clinical context and limitations. When properly applied, epitympanic devices reading near the tympanic membrane can approximate carotid arterial temperature and provide reasonably reliable estimates in cooperative, noncritical patients. Rectal and bladder probes are acceptable in awake individuals with mild to moderate hypothermia, yet both sites may lag during active rewarming and can be impractical or counterproductive in prehospital settings where additional exposure risks worsening heat loss. Esophageal temperature monitoring, with the probe positioned in the distal third of the esophagus, is the most accurate surrogate for core temperature during advanced airway management and ongoing resuscitation but should be reserved for intubated patients to avoid aspiration and malposition complications [12]. Conventional oral devices are useful mainly to exclude hypothermia, as many consumer-grade thermometers do not register below 35 °C; similarly, tympanic membrane infrared devices are unreliable in cold environments or when ear canal conditions are suboptimal. Given these constraints, the imperative in both field and hospital settings is to obtain a defensible core temperature as early as feasible, recognizing that prehospital logistics, environmental constraints, and patient factors may complicate measurement and delay precise staging [12].

Clinical manifestations correlate with the depth of hypothermia and the physiologic limits of thermoregulation. Mild hypothermia, defined by a core temperature of 32 to 35 °C (90–95 °F), often presents with nonspecific symptoms—hunger, nausea, fatigue, and pallor—with objective evidence of compensatory thermogenesis: shivering, increased muscle tone, tachypnea, and elevations in blood pressure and heart rate. Notably, shivering may be attenuated or absent when glycogen stores are depleted, as in malnutrition or endocrine deficiency states. Early neurocognitive effects—slowed mentation, impaired executive function, ataxia, and dysarthria—are common and contribute to risk-taking and poor judgment. Peripheral vasoconstriction promotes “cold diuresis,” leading to natriuresis, volume contraction, and potential prerenal azotemia if fluid losses are uncorrected. With moderate hypothermia (28 to 32 °C; 82–90 °F), central nervous system depression deepens, producing lethargy, hyporeflexia, and pupillary dilation with sluggish reactivity. Systemic physiology shifts from compensation to failure: hypotension, bradycardia, and bradypnea emerge, and shivering typically ceases around 30 to 32 °C, signaling a critical decline in endogenous heat production. Paradoxical undressing—disinhibited removal of clothing despite cold stress—may be observed and is associated with advanced neurocognitive impairment. Cardiac irritability increases significantly at these temperatures, with atrial fibrillation, the most frequently encountered dysrhythmia, often responsive to rewarming alone as myocardial conduction recovers. Severe hypothermia (<28 °C; <82 °F) heralds progressive failure of cerebral and cardiovascular function. Cerebral blood flow falls, culminating in obtundation and coma; blood pressure, heart rate, and cardiac output continue to decline, and atrial or junctional rhythms may precede malignant ventricular dysrhythmias. Pulmonary congestion can develop as left ventricular function deteriorates, while oliguria reflects cold diuresis compounded by reduced renal perfusion. Neurologic examination may reveal areflexia, and without prompt, controlled rewarming, the trajectory often culminates in cardiorespiratory failure and death [13]. Importantly, apparent pulselessness can be misleading in profound hypothermia; prolonged pulse checks and careful ultrasound assessment may be necessary before declaring arrest, and resuscitative efforts should continue with attention to minimizing patient movement to reduce dysrhythmia risk [13].

A comprehensive history and physical examination must also screen for concomitant cold-induced local injuries (frostnip, frostbite, trench foot) and for alternative or contributory diagnoses. Discrepancies between vital signs and the degree of hypothermia should prompt evaluation for conditions such as hypothyroidism, adrenal insufficiency, sepsis,

hypoglycemia, carbon monoxide poisoning, malnutrition, and intentional or unintentional overdose. Medication and toxin histories are essential: beta-blockers may blunt catecholaminergic responses; clonidine, neuroleptics, meperidine, and general anesthetic agents depress thermogenic drive or vasomotor tone; and ethanol and sedative-hypnotics impair judgment and heat conservation behaviors, compounding environmental risk. Throughout the encounter, nursing and interprofessional priorities include meticulous insulation, prevention of further heat loss, gentle handling to avoid afterdrop-related instability, and timely escalation to active internal or external rewarming modalities as dictated by accurate, ongoing core temperature measurement and clinical status [12][13].

Evaluation

For any patient with suspected hypothermia, the evaluation begins with the trauma ABCs, as hypothermia can precipitate cardiorespiratory arrest and confound the clinical picture. A rapid primary survey should identify threats to airway patency, ventilatory adequacy, and perfusion while disability and exposure assessments guide immediate risk mitigation. After completing the primary survey, all wet or restrictive clothing should be removed and the chest fully exposed to permit rapid application of dry, warm blankets; warmed, humidified oxygen and warmed intravenous fluids can be initiated in parallel to limit ongoing heat loss and support hemodynamics [13]. Point-of-care testing is integral to this early phase: obtain a finger-stick glucose without delay, as occult hypoglycemia can mimic or exacerbate neurocognitive depression; send a complete blood count and a basic metabolic panel to assess hemoconcentration, renal function, and electrolyte status. Because hypothermia evolves dynamically during resuscitation, repeat serum electrolytes approximately every four hours is prudent to detect hypokalemia from cold diuresis or post-rewarming shifts, rising creatinine from prerenal azotemia, and acid-base disturbances that may herald clinical deterioration. Cold diuresis, driven by impaired antidiuretic hormone secretion and peripheral vasoconstriction, commonly produces a rise in hemoglobin and hematocrit. Serum glucose lacks a reliable directional pattern in hypothermia *per se*; rather, extreme values usually reflect comorbid conditions such as diabetes mellitus, sepsis, or endocrine insufficiency. When invasive procedures are contemplated—central venous access, thoracostomy, or operative intervention—a coagulation panel is essential. Clinicians should interpret these studies cautiously: standard laboratory analyzers warm samples to 37 °C, potentially masking *in vivo* hypocoagulability at the patient's actual core temperature; nonetheless, obtaining fibrinogen helps screen for disseminated intravascular coagulation and guides blood product selection. Expanded diagnostics tailored to the presentation may include serum lactate

for global perfusion, creatine kinase for rhabdomyolysis risk, troponin for demand ischemia or hypothermia-associated myocardial stress, thyroid-stimulating hormone and cortisol for endocrine precipitants, magnesium for arrhythmia propensity, lipase when pancreatitis is suspected, and a comprehensive toxicology screen to uncover sedatives, ethanol, or other agents that impair thermoregulation.

Imaging is dictated by clinical suspicion. Chest radiography may be unremarkable in the absence of trauma or cardiopulmonary disease but can reveal aspiration, edema, or rib fractures. Bedside ultrasound is a valuable adjunct for real-time assessment of cardiac activity during tenuous pulses, evaluation of volume status via IVC dynamics, and identification of occult pneumothorax or hemoperitoneum. In patients whose mental status depression seems disproportionate to the degree of hypothermia—or when there is concern for trauma or stroke—noncontrast head computed tomography should be obtained to evaluate for intracranial hemorrhage or large-vessel occlusion, recognizing that hypothermia can blunt neurologic examination findings [13]. Electrocardiography is mandatory given the high prevalence and prognostic significance of dysrhythmias in hypothermia. Cooling slows myocardial repolarization by affecting potassium currents, prolonging the QT interval and predisposing to early afterdepolarizations. An elevated J point, producing the classic Osborn (J) wave most evident in precordial leads, may be observed; its amplitude often correlates with the depth of hypothermia, though it is not pathognomonic and can be seen in other conditions. Any arrhythmia can occur, but atrial fibrillation is most common and frequently self-resolves with controlled rewarming. Bradycardia predominates in moderate to severe hypothermia due to sinus node suppression; while often physiologic at low core temperatures, it signals heightened vulnerability to ventricular dysrhythmias, particularly during rough handling or rapid afterdrop. Continuous ECG and core temperature monitoring, serial laboratory reassessment, and judicious use of imaging and point-of-care ultrasound together create a comprehensive evaluative framework that informs staging, risk stratification, and the selection of passive versus active rewarming strategies across prehospital and in-hospital settings [13].

Treatment / Management

Hypothermia management is anchored in two overarching priorities: prompt prevention of further heat loss and controlled rewarming, implemented in parallel with rigorous support of airway, breathing, and circulation. The initial approach mirrors trauma resuscitation sequencing to prevent fixation on temperature while critical threats persist. After the primary survey confirms or addresses immediate life threats, all wet clothing must be removed without delay and replaced with dry insulation, and the torso—

especially the anterior chest—should be covered with dry, warm blankets to reduce radiative and convective losses [14][15]. Early initiation of warmed, humidified oxygen and warmed intravenous fluids integrates thermal support with resuscitative care, while secondary assessment documents exposure history, comorbidities, medications, mental status, a focused physical examination, and accurate core temperature to guide the intensity of rewarming [14][15]. A defining nuance in hypothermia care is the need for deliberate, gentle handling, particularly in patients with moderate or severe hypothermia. Cold myocardium is electrically irritable; abrupt movements, rough transfers, or unnecessary procedures can precipitate malignant dysrhythmias, including ventricular fibrillation. Patients should be moved in a horizontal position when feasible, with coordinated team maneuvers that limit jostling, and invasive procedures should be minimized until core temperature is rising and hemodynamics are stable [16]. Simultaneously, clinicians must investigate and treat precipitating injuries and comorbid illness, because trauma, intoxication, infection, endocrine crises, and malnutrition commonly coexist and can derail rewarming or precipitate recrudescence of instability if unaddressed [17]. Immediate correction of hypoglycemia is essential; if the patient is alert and able to swallow, oral glucose may be administered, whereas obtunded or intubated patients require intravenous dextrose with frequent reassessment of glucose during rewarming because metabolic demands and insulin dynamics shift with rising temperature [17].

Selection of a rewarming strategy is individualized by severity of hypothermia, hemodynamic stability, resource availability, and response to initial measures. Broadly, options include passive external rewarming (PER), active external rewarming (AER), active internal (core) rewarming, and extracorporeal techniques. For mild hypothermia with intact shivering, PER is the preferred therapy and typically raises core temperature at 0.5 to 2 °C per hour [14][15]. PER consists of removing wet garments, applying vapor barriers and insulating layers, covering the head and neck, minimizing exposure during examinations, and transferring heat from the patient's endogenous production to the core by reducing peripheral loss. Close monitoring during PER ensures that shivering remains effective and that energy stores are sustained, as shivering thermogenesis depends on adequate glycogen and glucose availability [14][15]. Shivering is a powerful endogenous rewarming mechanism—capable of increasing metabolic heat production up to five-fold over baseline—but this comes at a significant metabolic cost. In patients with limited cardiopulmonary reserve, sustained shivering can exacerbate hypoxemia, precipitate myocardial ischemia, or worsen respiratory fatigue. Clinicians should therefore assess the trade-offs between

allowing robust shivering and instituting more active, externally driven heat transfer. In some cases, analgesia, gentle anxiolysis, or low-dose magnesium may temper excessive shivering while preserving effective thermogenesis; however, sedatives that blunt protective airway reflexes or depress ventilation should be used sparingly and only with appropriate monitoring, especially before the patient's core temperature has risen to safer thresholds [14][15].

AER becomes necessary for patients with moderate to severe hypothermia, for those with mild hypothermia who fail to rewarm as expected, or when shivering is absent or insufficient. Convective forced-air warming systems deliver heated air across a large surface area, reducing convective loss and augmenting heat gain. Conductive warming with chemical or electric heat packs, radiant warmers, or circulating water mattresses can supplement convective techniques. For maximal efficiency and to preferentially warm central blood volume, heat should be applied to high-flow, low-mass regions—the axillae, anterior chest, and upper back—rather than distal extremities alone [18]. This “truncal-first” strategy reduces the risk of afterdrop, the paradoxical decline in core temperature that can occur when peripheral vasodilation returns cold, acedemic blood from the extremities to the core. Throughout AER, clinicians must continue to limit exposure, rotate warming surfaces to avoid thermal injury, reassess skin integrity, and titrate warming intensity to a safe rewarming rate. Warm-water immersion can, in select contexts, provide rapid heat transfer, but it complicates monitoring, impedes access for resuscitation, and increases the risk of uncontrolled peripheral vasodilation. If used, extremity immersion at 44–45 °C requires vigilant hemodynamic and temperature monitoring to avoid precipitous afterdrop and abrupt increases in cardiac workload [18]. Given these challenges, water immersion is generally reserved for controlled environments with experienced teams and is seldom first-line in unstable presentations.

Active internal (core) rewarming is indicated when external techniques are inadequate, when hypothermia is moderate to severe with hemodynamic instability, or when rapid, controllable heat transfer to the central compartment is required. Foundational measures include humidified oxygen at 40–42 °C to warm the bronchial circulation and warmed crystalloid infusions at 40–42 °C to counteract cold diuresis and support preload; both are safe and widely available [14][15]. Warmed cavity lavage increases heat transfer surface area and can be directed to the stomach, bladder, colon, pleural, or peritoneal spaces. Among these, pleural and peritoneal lavages are preferred for efficiency because the serosal surfaces offer extensive vascular contact. Pleural lavage is performed with two thoracostomy tubes: an anterior tube placed in the second to third intercostal space along the midclavicular line for inflow and a posterior tube in

the fifth to sixth intercostal space at the posterior axillary line for outflow; warmed fluid is infused anteriorly and drained posteriorly to bathe the mediastinum and lungs [18]. Peritoneal lavage uses two or more catheters to circulate warmed dialysate through the abdominal cavity, offering the dual benefits of rewarming and evaluation for occult intra-abdominal injury in trauma scenarios [18]. Gastric or colonic lavage may be considered in resource-limited environments but generally provides less efficient core heat transfer and carries risks of aspiration or perforation in unstable patients.

Extracorporeal rewarming techniques represent the most rapid and physiologically supportive options when available. Intermittent hemodialysis is the most accessible modality in many centers and can raise core temperature by approximately 2 to 3 °C per hour while simultaneously correcting electrolyte and acid-base disturbances [14][15]. Continuous arteriovenous (AV) rewarming—warming femoral arterial blood and returning it to the contralateral femoral vein—achieves rewarming rates near 4.5 °C per hour but requires adequate intrinsic blood pressure and vascular access [14][15]. The most potent methods are cardiopulmonary bypass (CPB) and venoarterial extracorporeal membrane oxygenation (VA-ECMO), which can deliver 7 to 10 °C per hour of rewarming while providing full cardiopulmonary support for patients in cardiac arrest, profound shock, or refractory hypothermia [14][15]. These techniques permit controlled rewarming with meticulous hemodynamic management and oxygenation, yet they are highly resource-intensive, necessitate specialized teams, and may be unavailable in many settings. Importantly, both CPB and VA-ECMO typically require systemic anticoagulation, increasing hemorrhagic risk in trauma or in patients with coagulopathy, and these risks must be balanced against the otherwise dismal prognosis of severe, unstable hypothermia [14][15].

Advanced airway and ventilatory strategies must be tailored to the hypothermic physiology. Endotracheal intubation should proceed with minimal patient movement and with preparedness for dysrhythmia. Sedative and paralytic dosing requires caution because hypothermia alters pharmacokinetics and pharmacodynamics; drug metabolism slows, protein binding changes, and receptor sensitivity shifts, prolonging effect duration and increasing the risk of hypotension and bradycardia. When neuromuscular blockade is necessary, dose reductions and extended monitoring are prudent. Ventilator settings should target normocapnia based on temperature-corrected values when available, as hypothermia affects gas solubility and may lead to misinterpretation of blood gases if uncorrected. Warm, humidified gases mitigate airway heat loss and support mucociliary function [14][15]. Concurrently, gentle fluid resuscitation with warmed isotonic crystalloids counters cold diuresis and restores preload, but

overzealous fluid administration may aggravate pulmonary edema in a myocardium with reduced contractility; dynamic assessments of volume responsiveness and bedside ultrasound can refine resuscitation targets [17]. Cardiac rhythm management in hypothermia emphasizes minimizing provocation and prioritizing rewarming. Atrial fibrillation and other supraventricular dysrhythmias are common and often resolve with rising core temperature; routine antiarrhythmic therapy is generally deferred until after rewarming unless the patient is unstable. Sinus bradycardia at low temperatures is physiologic and typically unresponsive to atropine; pacing is reserved for refractory instability after rewarming has begun. In the event of ventricular fibrillation or pulseless ventricular tachycardia, defibrillation attempts should be made, but repeated shocks and vasoactive drugs may be less effective at very low core temperatures; careful attention to guideline-based modifications and continued active rewarming are crucial to restoring cardiac stability [16]. Throughout, unnecessary movement should be avoided to reduce arrhythmia risk during the most vulnerable temperature ranges [16].

Ongoing care during rewarming focuses on anticipating complications and titrating therapy to physiologic responses. Electrolytes, especially potassium and magnesium, should be reassessed frequently, as intracellular–extracellular shifts occur with cooling and rewarming. Hypokalemia may emerge during rewarming and increase arrhythmia risk, requiring judicious replacement, whereas profound hyperkalemia in cardiac arrest may indicate nonviability in some contexts but must be interpreted alongside exposure duration and rewarming progress. Coagulation parameters are challenging to interpret because laboratory analyzers warm samples to 37 °C; clinical assessment of bleeding and point-of-care viscoelastic testing, where available, can complement decision-making around transfusion and anticoagulation in patients undergoing invasive rewarming [14][15]. Glucose monitoring should be frequent, as counterregulatory hormones, insulin secretion, and tissue utilization shift rapidly with temperature change, predisposing to both hypo- and hyperglycemia if therapy is not adjusted dynamically [17]. Disposition planning hinges on severity, response to therapy, comorbid conditions, and resource availability. Patients with mild hypothermia who rewarm promptly with PER, maintain stable vitals, and lack serious comorbidities may be observed and discharged with education on environmental protection, nutrition, and avoidance of alcohol and sedatives in cold exposure. Those with moderate to severe hypothermia, arrhythmias, altered mental status, trauma, or persistent metabolic derangements require monitored care and often intensive care unit admission for continued core temperature monitoring, hemodynamic support, and staged de-escalation of

rewarming modalities [16][17]. When extracorporeal support is contemplated, early consultation with regional centers helps streamline transfer decisions and minimize delays that can compromise neurological outcome in patients with hypothermic cardiac arrest [14][15].

Prevention counseling is integral to comprehensive management and reduces recurrence risk. Education should emphasize layered clothing strategies that maintain a dry microclimate, windproof and waterproof outerwear, planned nutrition and hydration, avoidance of alcohol and sedatives before cold exposure, and the importance of shelter and heat sources in inclement weather. For patients with occupational or recreational cold exposure, instruction on early recognition of shivering exhaustion, cognitive changes, and paradoxical behaviors can prompt earlier self-rescue or activation of emergency services. In healthcare settings, perioperative thermal management—including prewarming, warmed infusates, forced-air warming, and minimization of exposure time—reduces iatrogenic hypothermia, shortens recovery, and mitigates coagulopathy and wound complications [14][15]. In sum, the successful treatment of hypothermia requires disciplined adherence to resuscitative priorities, meticulous attention to gentle handling, and a staged, physiology-informed selection of rewarming methods. Passive external rewarming suffices for many patients with mild hypothermia who retain effective shivering, while active external and internal techniques address the needs of those with moderate to severe disease or inadequate endogenous thermogenesis. Extracorporeal modalities offer life-saving, rapid rewarming with concurrent cardiopulmonary support for the most critically ill, albeit with resource and anticoagulation constraints that demand judicious use [14][15][16][17][18]. Across all severities, integrating comorbidity management, vigilant monitoring for dysrhythmias and coagulopathy, and careful titration of fluids, oxygen, and medications ensures safer rewarming trajectories and improves the likelihood of neurological and functional recovery.

Differential Diagnosis

The evaluation of an abnormally low core temperature must extend beyond environmental exposure to encompass a wide array of pathologies that disrupt thermoregulation, reduce heat production, or accelerate heat loss. Framing the differential by physiologic loci—central control, metabolic supply, peripheral effector failure, endocrine disorders, impaired energy availability, neuromuscular compromise, dermatologic losses, iatrogenic factors, toxin and drug effects, and systemic illness—helps clinicians target history, examination, and testing efficiently while avoiding premature closure on exposure hypothermia alone [19]. Central failure includes cerebrovascular accident, traumatic brain injury, and hypothalamic dysfunction, any of which

can perturb the afferent–efferent loops governing vasoconstriction, shivering, and behavioral responses. Focal neurologic deficits altered pupillary reactivity, or imaging findings on head CT/MRI support these etiologies; concomitant disorders such as intracranial hemorrhage or diffuse axonal injury may explain disproportionate encephalopathy relative to the measured temperature.

In contrast, metabolic failure and toxin-related hypothermia arise from impaired substrate delivery or cellular energy utilization. Ethanol and sedative–hypnotics blunt protective responses and promote vasodilation, whereas selected pharmacologic agents—beta-blockers, clonidine, neuroleptics, meperidine, and anesthetics—depress thermogenesis and autonomic tone, often in concert with altered mental status or bradycardia disproportionate to temperature decline [19]. A careful medication and substance history, toxicology screening, and correlation with ECG and hemodynamics help distinguish these causes. Peripheral failure encompasses acute spinal cord transection and peripheral neuropathies, where loss of sympathetic outflow and motor shivering capacity undermines heat conservation. Physical findings of flaccid paralysis, sensory level, areflexia, or autonomic dysfunction point to these diagnoses. Endocrinologic failure is another pivotal category: hypothyroidism (including myxedema coma), adrenal insufficiency, and severe diabetic or alcoholic ketoacidosis may each present with hypothermia, hypotension, hyponatremia or hyperkalemia, and altered mentation. Targeted assays—TSH with free T4, morning cortisol or cosyntropin testing, serum ketones and blood gas—provide critical discrimination and should be prioritized when vitals are incongruent with the degree of exposure [19]. Lactic acidosis from sepsis or hypoperfusion also drives systemic heat loss and must be considered when leukocytosis, hypotension, and elevated lactate coexist.

Insufficient energy states are common and often underrecognized. Hypoglycemia, severe malnutrition, and failure to thrive decrease basal metabolic rate and shivering capacity, particularly in the extremes of age where limited reserves and inactivity compound risk. Neuromuscular compromise—including advanced neuromuscular disease or critical illness myopathy—reduces the mechanical substrate for shivering; bedside strength testing, respiratory mechanics, and creatine kinase levels support this line of inquiry. Dermatologic conditions such as extensive burns or exfoliative dermatoses increase radiant and evaporative losses; the degree of cutaneous disruption often correlates with the severity of hypothermia and guides the aggressive use of active external rewarming and fluid resuscitation [19]. Iatrogenic etiologies span emergency childbirth in cold environments, infusion

of unwarmed crystalloids or blood products, and deliberate cooling during heat-stroke treatment. In perioperative settings, anesthetic-induced vasodilation and exposure are frequent precipitants; chart review of intraoperative temperatures, warming measures, and infused fluid temperatures can clarify causality and direct prevention strategies. Finally, a broad miscellaneous category comprises carcinomatosis with cachexia, cardiopulmonary disease with low-output states, major infection and sepsis, multisystem trauma, and shock. Here, hypothermia may be both a marker and a mediator of poor perfusion; recognition of the “lethal triad” (hypothermia, acidosis, coagulopathy) in trauma prompts rapid hemorrhage control, blood product resuscitation, and active rewarming. Chest imaging, point-of-care ultrasound, cultures, inflammatory markers, and lactate trends help differentiate these systemic conditions from primary exposure hypothermia and reveal concurrent organ dysfunction [19]. In practice, distinguishing among these categories requires a structured, hypothesis-driven approach: document exposure history and ambient conditions; obtain accurate core temperature; screen for toxins and medications that blunt thermogenesis; interrogate endocrine axes; assess neurologic integrity; inspect the skin for conditions that amplify heat loss; and evaluate for shock, sepsis, trauma, or malignancy. Anchoring the workup in this taxonomy ensures that reversible causes are rapidly identified and treated, that iatrogenic contributors are mitigated, and that rewarming strategies are matched to the underlying pathophysiology rather than applied reflexively. A comprehensive differential thus not only clarifies etiology but also informs targeted management and prognosis across the spectrum of hypothermic presentations [19].

Prognosis

Severe hypothermia can be lethal, but prognosis varies widely and is mediated by mechanism of cooling, duration of exposure, preexisting comorbidities, the presence or absence of hypoxia and trauma, and the timeliness and quality of resuscitation and rewarming. Hemodynamically stable patients with primary environmental hypothermia—who present without major injuries, intoxication, or sepsis—often do remarkably well when promptly managed with active external or minimally invasive rewarming; survival in this group approaches 100%, with frequent full neurological recovery once normothermia is restored and metabolic derangements are corrected. Conversely, when hypothermia is complicated by cardiac arrest, outcomes hinge on rapid deployment of advanced rewarming and circulatory support. Modern extracorporeal strategies such as venoarterial extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass have shifted the survival curve, with survival rates approaching 50% even among patients requiring extracorporeal resuscitation. Factors consistently

linked to better outcomes in this cohort include the absence of hypoxia and severe polytrauma, a short interval to high-quality cardiopulmonary resuscitation, and immediate access to controlled rewarming and hemodynamic support. Extraordinary recoveries have been documented, including cases of accidental hypothermia with core temperatures as low as 14 °C culminating in full neurological recovery when meticulous rewarming and organ support are provided [19]. Rhythm-specific prognostication is nuanced: ventricular fibrillation during hypothermia, while ominous, may resolve with rewarming and defibrillation, and survivors can achieve good neurological outcomes when circulation is rapidly restored. By contrast, hypothermia-associated asystole is often refractory to standard advanced cardiac life support; many patients require rewarming to approximately 35 °C before organized cardiac activity returns, emphasizing the principle that “no one is dead until warm and dead.” Laboratory data can refine decisions in prolonged arrests. Profound hyperkalemia—particularly serum potassium levels exceeding 12 mEq/L—may indicate irreversible cellular injury from global ischemia and cytolysis and can help guide cessation of futile resuscitative efforts when integrated with clinical context and exposure history. Among survivors, residual morbidity is driven less by the hypothermia itself than by concomitant injuries and ischemia–reperfusion effects; frostbite, rhabdomyolysis, neuropathic pain, and functional limitations can persist. Prognosis is generally poorer at the extremes of age and in severe hypothermia, where frailty, limited metabolic reserve, and a higher burden of comorbid disease magnify risks despite optimal therapy. Still, early recognition, gentle handling to avoid dysrhythmias, and rapid, staged rewarming in an experienced center can transform the trajectory for many patients, underscoring the importance of organized systems of care for hypothermic emergencies [19].

Complications

Complications of hypothermia span direct cold-mediated tissue injury, systemic physiologic derangements during cooling and rewarming, and sequelae of concomitant trauma or illness. Frostbite is a paradigmatic complication and, if unrecognized or undertreated, can progress to dry gangrene with tissue mummification. Secondary infection may convert this to wet gangrene, with anaerobic pathogens such as *Clostridium perfringens* producing gas and crepitus; refractory infections, particularly when compounded by vascular compromise and deep tissue necrosis, can necessitate amputation despite optimal wound care. Beyond frostbite, hypothermia triggers a cascade of systemic complications that reflect impaired perfusion, altered cellular metabolism, and membrane instability. Cold diuresis promotes intravascular depletion and prerenal azotemia, predisposing to acute kidney injury; skeletal muscle ischemia and shivering exhaustion can culminate in rhabdomyolysis with

myoglobinuric renal damage; depressed airway reflexes and altered mentation increase the risk of aspiration; and membrane channel dysfunction fosters a spectrum of cardiac rhythm disturbances, with atrial fibrillation, atrial flutter, ventricular ectopy, ventricular fibrillation, and pulseless electrical activity all reported in more advanced cases. Other recognized complications include hyperkalemia from cellular breakdown, pulmonary edema due to myocardial depression and capillary leak, cerebellar dysfunction with ataxia, pancreatitis likely linked to ischemia–reperfusion injury, and progression to coma and death in untreated or refractory cases [11]. Rewarming itself, while lifesaving, introduces additional hazards. Rapid temperature shifts can provoke afterdrop and hemodynamic instability; systemic inflammation may surge as perfusion returns to cold, academic tissues; and several metabolic perturbations are well described, including hyperkalemia from potassium efflux during reperfusion and hypophosphatemia, hypomagnesemia, and hypocalcemia as electrolytes redistribute with normalization of pH and temperature. Myocardial stunning can manifest as stress-induced (Takotsubo) cardiomyopathy during or after rewarming, compounding arrhythmic risk. Rhabdomyolysis may paradoxically worsen transiently as circulation improves. Arrhythmias—including atrial fibrillation, ventricular tachyarrhythmias, and pulseless electrical activity—can recur during rewarming, especially if electrolyte shifts are uncorrected. Infectious complications, notably pneumonia due to aspiration or prolonged ventilation, are frequent in severely hypothermic patients. Hemostatic abnormalities range from platelet dysfunction and thrombocytopenia to inappropriate platelet aggregation and thrombosis, complicated by the limitations of standard coagulation testing at nonphysiologic temperatures. Finally, glucose homeostasis can swing from reduced utilization and relative hyperglycemia to insulin resistance during the rewarming phase, necessitating close glycemic monitoring and titration of therapy [20][21][22][23][24]. Vigilant anticipation, early detection, and targeted management of these complications—paired with careful pacing of rewarming and meticulous supportive care—are central to improving outcomes.

Patient Education

Hypothermia is highly preventable, and clinician-led education can meaningfully reduce risk across communities and care settings. Prevention begins with behavioral modification and situational awareness. Patients should be counseled to minimize exposure during cold, windy, and wet conditions; when outdoor activity is unavoidable, they should dress in layered, moisture-wicking clothing with insulating and windproof outerwear, cover the head, neck, and extremities, and carry spare dry garments to replace wet layers promptly. Hydration and caloric

intake are essential to sustain thermogenesis; individuals engaging in outdoor work, winter sports, or remote travel should plan nutrition and rest intervals and avoid alcohol and sedative-hypnotics, which blunt judgment, induce vasodilation, and impair shivering. Education should emphasize early warning signs—progressive shivering, clumsiness, slurred speech, confusion, and paradoxical behaviors—and the importance of seeking shelter at the first sign of cognitive impairment. Household-level strategies complement individual behaviors. Vulnerable populations—infants and young children with limited thermoregulatory capacity, older adults with reduced metabolic reserve, individuals with cognitive impairment or severe mental illness, and patients on medications that depress thermogenesis—benefit from proactive caregiver oversight. Families and caregivers should ensure appropriate clothing, routine temperature checks in living spaces, and adherence to medication plans that minimize hypothermia risk where feasible. Medications with hypothermia potential, including sedatives, neuroleptics, clonidine, and beta-blockers, should be secured out of reach of children and cognitively impaired adults. Housing conditions also matter; clinicians should counsel on adequate home heating, insulation, and safe use of warming devices, including carbon monoxide alarms and fire safety plans to mitigate the risks of space heaters and fireplaces. For communities with high exposure risk—such as people experiencing homelessness—linkage to shelters, warming centers, and outreach programs during cold snaps can be lifesaving; practitioners can collaborate with social services to provide warm clothing, blankets, and information about emergency resources. In healthcare environments, prevention includes perioperative thermal management with prewarming, warmed intravenous fluids, forced-air warming, and minimizing exposure time to prevent iatrogenic hypothermia. Occupational medicine programs can integrate cold-stress prevention into workplace safety training for outdoor laborers, including buddy systems, scheduled warm-up breaks, and access to heated shelters. Ultimately, effective deterrence relies on tailored education that addresses individual behaviors, caregiver roles, environmental safety, and systemic supports, thereby reducing the incidence and severity of hypothermia episodes across populations.

Pearls and Other Issues

Several pragmatic insights can guide clinicians in the rapid recognition and safe management of hypothermia across prehospital, emergency, perioperative, and intensive care settings. First, remember the pathophysiologic core: hypothermia arises when heat loss overwhelms heat production due to exposure, impaired thermoregulation, or inadequate metabolic substrate. Clinical manifestations span a spectrum from mild, nonspecific malaise with shivering to profound coma

with malignant dysrhythmias, and timely intervention can arrest progression. Second, presentation may mimic cardiopulmonary arrest; some profoundly hypothermic patients appear unconscious, pulseless, and apneic, yet retain low-flow circulation. Resuscitation should therefore commence immediately while carefully verifying pulses with extended checks and, when available, bedside ultrasound, recognizing that the hypothermic heart is exquisitely sensitive to mechanical irritation. Third, measurement accuracy is foundational. Most consumer-grade oral thermometers bottom out near 34–35 °C; low-reading devices are required, and the esophageal probe—placed in the distal third in an intubated patient—offers the most accurate estimate of core temperature, whereas rectal temperatures may lag by up to an hour during dynamic rewarming. Fourth, laboratory and ECG interpretation must be temperature-aware. Hyperkalemia in hypothermia does not always manifest with classic electrocardiographic features, and conventional coagulation assays performed at 37 °C can obscure *in vivo* coagulopathy until the patient is rewarmed. Osborn (J) waves are suggestive but not specific; their presence, coupled with bradycardia and QT prolongation, should heighten concern for malignant ventricular arrhythmias. Fifth, therapeutic escalation should be deliberate and matched to physiology. Passive and active external rewarming may suffice for mild to moderate cases, but hemodynamically unstable patients benefit from active internal rewarming and, when necessary, extracorporeal support; cardiopulmonary bypass and ECMO are the most invasive yet potent options for rewarming unstable patients. Sixth, failure to rewarm as expected warrants a search for reversible culprits such as hypoglycemia, sepsis, hypothyroidism, and adrenal insufficiency; correcting these drivers often restores thermogenic capacity and hemodynamic stability. Finally, prevention is not ancillary but essential. Patient and caregiver education—particularly in households with infants, frail older adults, or individuals with psychiatric illness—is a powerful tool to reduce recurrence, and clinicians should take every opportunity to reinforce practical strategies for insulation, shelter, nutrition, and medication safety. By coupling physiologic insight with methodical assessment, gentle handling, and staged rewarming, clinicians can navigate the complexities of hypothermia care while minimizing iatrogenic harm.

Enhancing Healthcare Team Outcomes

Optimal hypothermia care is inherently interdisciplinary and benefits from coordinated protocols that span the prehospital, emergency, operative, critical care, and rehabilitative continuum. Emergency medical services (EMS) are often the first clinical touchpoint; field providers identify exposure risk, initiate gentle handling, remove wet clothing, insulate the torso, provide warmed oxygen if available, begin warmed intravenous fluids when protocols

allow, and prioritize rapid yet careful transport. In the emergency department, emergency physicians and nurses lead the primary survey, secure the airway when indicated, establish core temperature monitoring, and select appropriate rewarming strategies while investigating precipitating factors and coexisting trauma or illness. Early activation of institutional hypothermia pathways streamlines access to forced-air warming, warmed fluids, and active internal rewarming modalities, and it delineates roles for laboratory, imaging, and pharmacy services. Critically ill patients frequently require intensive care unit admission, where nurses provide continuous hemodynamic and temperature monitoring, titrate analgesia and sedation mindful of altered pharmacokinetics, administer electrolytes and insulin as needed, and vigilantly watch for arrhythmias, coagulopathy, and infection. Pulmonologists support ventilator management with warm, humidified gases and temperature-corrected blood gas interpretation, while cardiologists guide rhythm management and assess for myocardial ischemia or stress cardiomyopathy. Nephrologists contribute when hemodialysis is selected for extracorporeal rewarming or for management of rhabdomyolysis and acute kidney injury. Surgeons—particularly trauma and plastic/reconstructive teams—address frostbite debridement, fasciotomy for compartment syndrome, and operative control of hemorrhage; cardiothoracic surgeons and perfusionists implement cardiopulmonary bypass or ECMO for refractory instability or hypothermic cardiac arrest, coordinating with transfusion services to balance anticoagulation against bleeding risk [13]. Respiratory therapists are integral to airway clearance, ventilator adjustments, and bronchial hygiene, while pharmacists optimize drug choice and dosing in the context of hypothermia-altered metabolism and potential QT prolongation. Physical and occupational therapists engage early to mitigate deconditioning, address balance and gait instability from neuropathy or cerebellar involvement, and plan safe discharge. Social workers and case managers coordinate access to shelters, warming centers, durable medical equipment, and follow-up care, which is especially critical for patients experiencing homelessness or with cognitive impairment. After discharge, primary care clinicians oversee longitudinal recovery, monitor for late complications such as chronic kidney disease after rhabdomyolysis or neuropathic sequelae of frostbite, and reinforce prevention strategies tailored to local climate and the patient's living circumstances. At a systems level, regular interdisciplinary simulations, retrospective case reviews, and protocol updates improve team readiness, reduce door-to-rewarm times, and standardize evidence-informed care. Establishing regional networks for rapid transfer to extracorporeal-capable centers expands access to life-saving therapies for hypothermic cardiac arrest. By integrating prehospital readiness, emergency efficiency, ICU

vigilance, specialty collaboration, and community reentry planning, healthcare teams can markedly enhance outcomes for patients with hypothermia and reduce the burden of preventable cold-related morbidity and mortality [13].

Conclusion:

In conclusion, the effective management of hypothermia is a complex, high-stakes clinical endeavor that demands a disciplined and physiology-guided approach. The path to positive patient outcomes hinges on several core principles: the immediate prevention of further heat loss, meticulous and gentle handling to prevent lethal arrhythmias, and the judicious selection of rewarming strategies tailored to the severity of the patient's condition and their hemodynamic stability. From simple passive external rewarming to advanced extracorporeal life support, each intervention must be applied with an understanding of the underlying pathophysiology, including the risks of afterdrop and rewarming shock. Ultimately, the nurse's role is central to success. As the first and most consistent clinical observers, nurses are essential for early recognition, accurate core temperature monitoring, and the vigilant execution of rewarming protocols. Furthermore, optimal outcomes are achieved through seamless interprofessional collaboration, integrating the skills of prehospital providers, physicians, surgeons, and rehabilitation specialists. By coupling a deep understanding of hypothermia's systemic effects with methodical assessment and staged rewarming, healthcare teams can significantly improve the chances of neurological and functional recovery, even for profoundly hypothermic patients. This comprehensive, team-based strategy is the cornerstone of reducing the significant morbidity and mortality associated with this life-threatening condition.

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