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Review Article

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Electrocardiographic manifestations of hypothermia: pathophysiology, clinical implications, and future directions

Abstract

Hypothermia, defined as a core body temperature below 35°C, profoundly affects cardiovascular physiology and is associated with characteristic electrocardiographic (ECG) abnormalities. These changes, ranging from sinus bradycardia to life-threatening arrhythmias, provide important diagnostic and prognostic information in both accidental and therapeutic hypothermia. Among these, Osborn waves remain the most recognized hallmark, although other findings including PR, QRS, and QT interval alterations, atrial and ventricular arrhythmias, and conduction blocks are equally relevant for clinical management.

This review synthesizes the current evidence on the pathophysiological mechanisms underlying hypothermia-induced ECG changes, their diagnostic and prognostic significance, and their implications for clinical management. In addition, we highlight the challenges in differential diagnosis, particularly the overlap of ECG changes with acute coronary syndromes and other metabolic disorders. Emerging technologies, including artificial intelligence and wearable monitoring, may improve recognition and risk stratification.

Future directions emphasize the need for standardized diagnostic criteria, multicenter studies, and exploration of advanced rewarming and anti-arrhythmic strategies. Understanding the spectrum of ECG alterations in hypothermia remains essential for timely recognition, effective treatment, and improved patient outcomes.

Keywords: hypothermia, electrocardiography, osborn wave, arrhythmia, therapeutic hypothermia

Volume 18 Issue 4 - 2025

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Received: September 23, 2025 | **Published:** October 17, 2025

Introduction

Hypothermia, defined as a core body temperature below 35 °C, remains a clinically significant condition that challenges physicians across emergency medicine, critical care, and cardiology. It can occur as a consequence of accidental environmental exposure, metabolic disorders, or therapeutic interventions. The clinical classification of hypothermia is based on severity: mild (32–35 °C), moderate (28–32 °C), and severe (<28 °C). This staging correlates with the degree of physiological dysfunction and clinical risk, particularly in the cardiovascular system.[1,2]

Historically, the recognition of electrocardiographic (ECG) changes in hypothermia dates back to the early 20th century, with Osborn's seminal description of the "J wave" in 1953 marking a milestone in the electrocardiographic characterization of cold-induced cardiac abnormalities.[3]. Since then, the ECG has remained a cornerstone in the diagnosis and monitoring of hypothermic patients, providing not only supportive evidence of low core temperature but also prognostic information regarding arrhythmia risk and mortality.[4]

Epidemiologically, accidental hypothermia affects populations exposed to harsh environmental conditions, including mountaineers, disaster victims, and the homeless. Additionally, iatrogenic hypothermia occurs during cardiac surgery or after cardiac arrest when therapeutic hypothermia is induced to preserve neurological outcomes.[5,6] Mortality rates vary widely, ranging from 20% to over 50% depending on severity and comorbidities, with cardiac arrhythmias being one of the leading causes of death.[7]

From a pathophysiological standpoint, hypothermia exerts profound effects on virtually every organ system, but its impact on the cardiovascular system is particularly striking. Cooling of myocardial tissue alters ion channel function, conduction velocity, and action potential duration, thereby creating a substrate for rhythm disturbances. In parallel, systemic hemodynamic changes such as bradycardia, decreased cardiac output, and increased systemic vascular resistance further compromise perfusion and amplify the risk of adverse outcomes.[8]

Electrocardiographically, hypothermia manifests as a spectrum of abnormalities. The most frequently encountered features include sinus bradycardia, atrioventricular (AV) conduction blocks, prolonged QT interval, widened QRS complexes, and atrial or ventricular arrhythmias.[9] Among these, the Osborn wave, or J wave, is considered pathognomonic of hypothermia, though it is not entirely specific, as similar findings may appear in other conditions such as hypercalcemia and Brugada syndrome.[10] Nonetheless, in the appropriate clinical context, its presence is highly suggestive of hypothermia.

Beyond diagnosis, ECG monitoring serves as a vital tool in prognostication. The amplitude of the J wave has been shown to correlate with the degree of hypothermia, while progression to ventricular fibrillation or asystole is strongly associated with severe cooling. Importantly, during rewarming, ECG abnormalities may persist or even worsen transiently, highlighting the dynamic nature of cold-induced cardiac electrophysiology.[11]

In modern practice, therapeutic hypothermia, more recently termed targeted temperature management (TTM), has introduced a controlled dimension to hypothermia in post-cardiac arrest care. This practice has renewed interest in the systematic study of hypothermia-induced ECG changes, as clinicians must differentiate between benign

findings of controlled cooling and malignant arrhythmias requiring intervention.[12,13]

Given the increasing clinical relevance, this review aims to synthesize the available evidence regarding the electrocardiographic manifestations of hypothermia. By exploring underlying pathophysiology, characteristic ECG changes, differential diagnosis, clinical implications, and future perspectives, this article provides a comprehensive framework for clinicians and researchers alike.

Pathophysiological mechanisms of hypothermia on the cardiovascular system

Hypothermia produces complex effects on the cardiovascular system through changes in electrophysiology, autonomic regulation, and systemic hemodynamics. The clinical consequences are most evident on the electrocardiogram (ECG), which reflects abnormalities in depolarization, repolarization, and conduction. A mechanistic understanding of these processes helps explain the characteristic ECG features of hypothermia (Table 1).

Table 1 Pathophysiological effects of hypothermia on the cardiovascular system

Mechanism	Effect	ECG manifestation	Reference
Ion channels	Slowed Na+ influx, reduced Ca2+ entry, inhibited K+ currents	QRS widening, AV block, QT prolongation	15–17
Autonomic system	Early sympathetic activation, late vagal predominance	Tachycardia → bradycardia, junctional rhythm, AV block	19–21
Hemodynamics	Decreased contractility, reduced CO, increased SVR	Ischemia-related arrhythmias	22–25
Metabolic/biochemical	Acidosis, electrolyte shifts, impaired O2 delivery	QT prolongation, ST–T changes	26–28
Osborn wave	Epicardial–endocardial heterogeneity	J wave	10,29

Abbreviations: AV, atrioventricular; CO, cardiac output; ECG, electrocardiogram; O2, oxygen; SVR, systemic vascular resistance.

Effects on ion channels and action potential

Cooling alters the kinetics of sodium, calcium, and potassium channels, slowing conduction and prolonging repolarization. Reduced sodium channel activity delays phase 0 depolarization and widens the QRS complex.[15] Suppressed calcium influx diminishes myocardial contractility and slows atrioventricular conduction.[16] Potassium current reduction prolongs action potential duration and contributes to QT prolongation. Enhanced transient outward potassium current in epicardial cells explains the genesis of Osborn waves.[17] These combined effects create a substrate prone to reentrant arrhythmias and ventricular fibrillation.[18]

Modulation of the autonomic nervous system

Hypothermia induces a biphasic autonomic response. In mild stages, sympathetic activation leads to vasoconstriction and tachyarrhythmias. With progressive cooling, vagal tone dominates, resulting in sinus node suppression, junctional rhythms, and advanced atrioventricular block.[19,20] Heart rate variability decreases significantly, reflecting impaired autonomic balance and correlating with arrhythmic risk.[21]

Mechanical and hemodynamic effects

Cooling impairs myocardial contractility through reduced calcium handling and cross-bridge cycling.[22] Cardiac output decreases by approximately 6–7% per °C drop in core temperature, driven by bradycardia and reduced stroke volume.[23] Increased systemic vascular resistance from cold-induced vasoconstriction raises afterload and predisposes to ischemia.[24] Coronary perfusion is reduced by higher blood viscosity and vasoconstriction, further enhancing arrhythmic risk.[25]

Metabolic and biochemical changes

Enzymatic activity and metabolic rate decline with hypothermia. Acidosis, electrolyte imbalances, and oxygen–hemoglobin dissociation shifts reduce oxygen delivery and increase arrhythmogenic potential.[26,27] Elevated plasma viscosity worsens microvascular circulation and ischemic injury.[28] These changes manifest as QT prolongation, ST-T alterations, and electrical instability.

Electrophysiological substrate for the osborn wave

The Osborn (J) wave reflects repolarization heterogeneity between ventricular epicardium and endocardium. Cooling accentuates the epicardial action potential notch due to stronger transient outward potassium current, generating the characteristic deflection at the junction of the QRS complex and ST segment.[10] Prominent J waves are associated with increased susceptibility to ventricular fibrillation.[29]

Integration of mechanisms and clinical manifestations

The above processes converge to produce the hallmark ECG features of hypothermia: sinus bradycardia, PR/QRS/QT prolongation, Osborn waves, and a high burden of atrial and ventricular arrhythmias. These reflect the combined effects of altered ion channel activity, autonomic imbalance, metabolic stress, and ischemia.

Electrocardiographic manifestations of hypothermia

Electrocardiography is the most practical non-invasive method for identifying and monitoring cardiac disturbances in hypothermia. The tracing reflects slowed conduction, delayed repolarization, and altered autonomic tone. Recognition of these findings is essential both for diagnosis and for anticipating malignant arrhythmias (Table 2).

Table 2 Clinical and prognostic significance of ECG findings in hypothermia

ECG Finding	Clinical Relevance	Prognostic Value	Reference
QT prolongation	Delayed repolarization	Increased risk of VF	38,47
QRS widening	Slowed conduction	Higher risk of malignant arrhythmias	34,47
High-grade AV block	Severe conduction system involvement	Poor prognosis, pacing may be required	48
Osborn wave	Diagnostic marker	May predict VF susceptibility	42,49
AF	Common in moderate hypothermia	Usually benign, resolves with rewarming	31,50

Abbreviations: AF, atrial fibrillation; AV, atrioventricular; ECG, electrocardiogram; VF, ventricular fibrillation

Citation: Birgün A, Kalçık M, Çelik MC, et al. Electrocardiographic manifestations of hypothermia: pathophysiology, clinical implications, and future directions. *J Cardiol Curr Res.* 2025;18(4):189–195. DOI: 10.15406/jccr.2025.18.00631

Sinus bradycardia and rhythm disturbances

Sinus bradycardia is the most frequent arrhythmia in hypothermia, caused by direct suppression of sinoatrial node activity.[9] The heart rate falls proportionally with core temperature, and profound bradycardia may mimic sinus arrest. At lower temperatures, junctional escape rhythms appear.[30] Atrial fibrillation is common in moderate to severe cases and usually resolves with rewarming.[31] Less frequently, atrial flutter or multifocal atrial tachycardia may occur.[32] These atrial arrhythmias are often hemodynamically tolerated but can signal progression toward instability.

Conduction abnormalities

Slowed conduction through atria, ventricles, and the atrioventricular node results in PR prolongation, QRS widening, and sometimes bundle branch block. High-degree atrioventricular block, including second- and third-degree block, is observed in severe hypothermia.[33–36] These abnormalities are clinically important because they predispose to malignant ventricular arrhythmias.

Repolarization changes: QT interval and ST-T abnormalities

Hypothermia prolongs ventricular repolarization through reduced potassium currents and heterogeneous action potential duration.[37] This manifests as marked QTc prolongation, predisposing to torsades de pointes and ventricular fibrillation.[38] ST-segment elevation or depression may mimic acute myocardial infarction, complicating diagnosis.[39] T-wave inversions or biphasic T waves, particularly in precordial leads, are also frequent.[40].

The osborn (J) wave

The Osborn wave is the most characteristic manifestation of hypothermia (Figure 1). It appears as a positive deflection at the junction of the QRS complex and ST segment, best seen in inferior and lateral leads.[3] Its amplitude increases as core temperature falls, becoming prominent below 32 °C.[41] While not entirely specific, in the correct clinical setting it is strongly suggestive of hypothermia.[42] Prominent J waves have been linked to increased risk of ventricular fibrillation.[43]

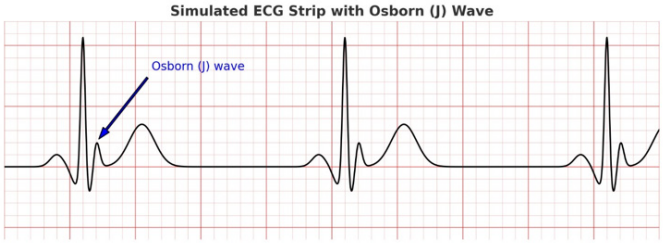

Figure 1 Simulated ECG strip demonstrating a subtle Osborn (J) wave. A small positive deflection following the S wave (arrow) represents the Osborn (J) wave, a characteristic electrocardiographic finding of hypothermia. This figure was generated using artificial intelligence (ChatGPT) for illustrative purposes and does not contain real patient data.

Ventricular arrhythmias

As temperature decreases, vulnerability to malignant arrhythmias increases. Ventricular premature beats are common and usually benign but can trigger more dangerous rhythms. Ventricular tachycardia occurs less frequently than fibrillation, while ventricular fibrillation is the most feared complication, especially below 28 °C.[44]. At extreme temperatures (<20 °C), asystole may occur due to complete suppression of automaticity.[1,2] Even minor stimuli, such as rough handling, may precipitate ventricular fibrillation, underscoring the need for caution.

Temperature-dependent ECG progression

ECG changes follow a predictable pattern with falling temperature. In mild hypothermia (32–35 °C), sinus bradycardia and mild interval prolongation dominate. In moderate hypothermia (28–32 °C), Osborn waves, atrial fibrillation, and conduction block become evident. Severe hypothermia (<28 °C) is marked by profound bradycardia, wide QRS complexes, marked QT prolongation, and malignant ventricular arrhythmias.[45]

Diagnostic and prognostic implications

ECG abnormalities in hypothermia are both diagnostic and prognostic. The presence of Osborn waves and QT prolongation indicates significant myocardial involvement, while atrial or ventricular arrhythmias suggest progression toward electrical instability. Many of these findings resolve with rewarming, emphasizing the dynamic and reversible nature of hypothermia-induced changes.[7]

Clinical and prognostic significance of ECG changes in hypothermia

Electrocardiographic changes in hypothermia are more than diagnostic markers; they also carry strong clinical and prognostic value. Correct interpretation guides acute management, anticipates complications, and influences outcome predictions (Table 3).

Diagnostic utility in emergency settings

In emergency situations, characteristic ECG findings, particularly Osborn waves and marked bradycardia, may provide the first clue to hypothermia, especially when body temperature cannot be measured immediately.[34] In patients with altered consciousness or outdoor exposure, ECG serves as a surrogate marker of core temperature.[46]. Correct differentiation from acute coronary syndromes or primary arrhythmia syndromes prevents unnecessary delays in rewarming.

Predictors of arrhythmic risk and mortality

Several ECG parameters correlate with adverse prognosis. QT prolongation and QRS widening are linked to ventricular fibrillation risk.[47] High-degree atrioventricular block reflects severe conduction system involvement and often requires pacing if not reversed by rewarming.[48] Although debated, J-wave amplitude may indicate electrical instability.[49] Atrial fibrillation, although common in moderate hypothermia, is usually benign but can herald worsening conduction abnormalities.[50]

Reversibility and prognostic implications of rewarming

Most ECG changes improve with rewarming. Sinus rhythm often reappears, conduction intervals normalize, and atrial fibrillation resolves spontaneously.[7,32] Even malignant arrhythmias such as ventricular fibrillation do not necessarily predict poor outcome if timely resuscitation and extracorporeal rewarming are applied.[51] Prognosis therefore depends not only on the severity of ECG findings but also on the rapidity and adequacy of treatment.

Clinical management considerations

Knowledge of ECG abnormalities influences treatment choices. Hypothermic patients must be handled gently, as mechanical stimulation can precipitate ventricular fibrillation in the presence of prolonged QT or J waves.[1] Defibrillation is less effective below 30 °C but should not be abandoned, as success improves with rewarming.[2]

Citation: Birgün A, Kalçık M, Çelik MC, et al. Electrocardiographic manifestations of hypothermia: pathophysiology, clinical implications, and future directions. *J Cardiol Curr Res.* 2025;18(4):189–195. DOI: 10.15406/jccr.2025.18.00631

Antiarrhythmic drugs are generally avoided because of unpredictable pharmacokinetics.[52] Temporary pacing may be needed for persistent high-degree block despite rewarming.[53]

Prognostic scoring and outcome prediction

ECG findings are incorporated into prognostic systems. Models that combine age, core temperature, and conduction abnormalities stratify mortality risk.[54] The Swiss staging system, integrating temperature with ECG and clinical findings, is widely used for triage.[55] Nonetheless, outcomes remain variable, and recovery with good neurological function is possible even in severe cases with malignant ECG features, provided that aggressive rewarming is undertaken.[56]

Table 3 Characteristics of arrhythmias in hypothermia

Arrhythmia	Occurrence	Clinical Relevance	Prognosis	Reference
Sinus bradycardia	Most common, increases with cooling	Usually well tolerated	Resolves with rewarming	9
Junctional rhythm	<32 °C when SA node suppressed	Often transient	Disappears after rewarming	30
AF	Frequent in moderate–severe hypothermia	Generally tolerated	Self-limited with rewarming	31,32
Atrial flutter / MAT	Rare	Limited significance	Only isolated cases reported	32
VT	Reported in moderate hypothermia	May progress to VF	Requires close monitoring	44
VF	Most common cause of death <28 °C	Life-threatening	High recurrence risk even after conversion	2,44
Asystole	<20 °C	Terminal finding	Rarely reversible	1

Abbreviations: AF, atrial fibrillation; MAT, multifocal atrial tachycardia; SA, sinoatrial; VF, ventricular fibrillation; VT, ventricular tachycardia

Implications for differential diagnosis

Hypothermia-related ECG changes may mimic other critical conditions. ST elevation can resemble acute myocardial infarction,[57] Brugada-like patterns may appear,[58] and Osborn waves may be mistaken for benign early repolarization.[59] Accurate interpretation in the clinical context is crucial to avoid misdiagnosis and unnecessary invasive procedures (Table 4).

Table 4 Differential diagnosis of hypothermia-related ECG changes

Condition	ECG Feature	Differentiating Clue	Reference
Acute MI	ST-segment elevation	Clinical symptoms, biomarkers, coronary anatomy	39,58
Brugada syndrome	J-like wave, ST elevation in V1–V3	Persistent pattern, genetic association	59
Hypercalcemia	Short QT, possible J wave	Elevated serum calcium	10
Early repolarization	J-point elevation, concave ST	Common in young healthy subjects	60

Abbreviations: ECG, electrocardiogram; MI, myocardial infarction; ST, ST segment

Overall prognostic perspective

ECG abnormalities in hypothermia should be viewed as dynamic and potentially reversible rather than terminal findings. They provide early diagnostic clues, guide preventive measures against arrhythmias, and reflect response to rewarming. Outcomes depend on both the extent of ECG abnormalities and timely clinical intervention, as well as comorbid conditions.[60]

Treatment and management strategies in hypothermia with ECG abnormalities

Management of hypothermic patients requires a structured and multidisciplinary approach, with electrocardiographic monitoring as a central element. Since conduction disturbances and arrhythmias are common and potentially fatal, continuous ECG assessment is mandatory during both resuscitation and rewarming.

General principles of management

The cornerstone of therapy is gradual rewarming combined with supportive measures such as airway protection, oxygenation, hemodynamic stabilization, and prevention of further heat loss.[2] Patients with moderate or severe hypothermia should be continuously monitored, as arrhythmias may arise abruptly. Handling must be gentle, because physical stimulation can precipitate ventricular fibrillation, especially in patients with Osborn waves or marked QT prolongation.[61] (Table 5).

Table 5 Management Approaches in Hypothermia with ECG Abnormalities

Strategy	Indication	Characteristics	ECG Effect	Reference
Passive external rewarming (PER)	Mild hypothermia	Insulation, blankets, removal from cold	ECG abnormalities resolve rapidly	63
Active external rewarming (AER)	Moderate hypothermia	Forced-air warming, warm water immersion	Risk of arrhythmias; requires ECG monitoring	64
Active internal rewarming (AIR)	Severe hypothermia	Warm IV fluids, humidified oxygen, lavage	Conduction disturbances resolve earlier	65
Extracorporeal rewarming (ECR)	Arrest/refractory arrhythmias	ECMO, CPB support	ECG normalization parallels myocardial recovery	66,67
Arrhythmia management	All stages	Bradycardia usually resolves; VF requires defibrillation + rewarming	ECG monitoring essential	2,32,68,69

Abbreviations: AIR, active internal rewarming; AER, active external rewarming; CPB, cardiopulmonary bypass; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; ECR, extracorporeal rewarming; PER, passive external rewarming; VF, ventricular fibrillation

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Rewarming techniques

- Passive External Rewarming (PER): Suitable for mild hypothermia (32–35 °C). Removal from the cold environment, insulation, and warm blankets are sufficient. ECG abnormalities are usually mild and resolve spontaneously.[62]
- Active External Rewarming (AER): Indicated for moderate hypothermia (28–32 °C). Forced-air systems or warm water immersion accelerate warming but may cause afterdrop from peripheral vasodilation; ECG monitoring is needed to detect induced arrhythmias.[63]
- Active Internal Rewarming (AIR): Required for severe hypothermia (<28 °C) or unstable patients. Options include warmed intravenous fluids, humidified oxygen, and peritoneal or thoracic lavage. These techniques achieve faster correction and earlier reversal of conduction abnormalities.[64]
- Extracorporeal Rewarming (ECR): Reserved for profound hypothermia with cardiac arrest or refractory arrhythmias. ECMO or cardiopulmonary bypass provides rapid rewarming with full circulatory and respiratory support, and survival is possible even after prolonged arrest.[65,66]

Management of arrhythmias

Bradycardia is usually physiologic in hypothermia and resolves with rewarming; pacing and atropine are rarely effective.[2] Atrial fibrillation is frequent but typically transient, requiring no pharmacological intervention.[32] Ventricular arrhythmias, particularly fibrillation, are life-threatening. Defibrillation should always be attempted, but success is limited below 30 °C. If initial shocks fail, further attempts may be delayed until after rewarming.[67] Antiarrhythmic drugs are avoided at low temperatures because of unpredictable metabolism (Table 6).[68]

Table 6 Pharmacological and defibrillation strategies in hypothermia

Intervention	Issue in Hypothermia	Recommendation	Reference
Atropine	Ineffective for bradycardia	Rewarming is mainstay; pacing rarely needed	2
Antiarrhythmic drugs (amiodarone, lidocaine)	Altered pharmacokinetics, unpredictable response	Avoid below 30 °C	53,69
Defibrillation	Reduced efficacy <30 °C	Attempt shocks; success improves with rewarming	52,68
Pacing	Indicated in high-grade AV block	Use if rewarming fails to restore conduction	54

Abbreviations: AV, atrioventricular; ECG, electrocardiogram

Advanced life support considerations

Resuscitation guidelines differ from normothermia. Chest compressions must continue until the patient is rewarmed to at least 32 °C, as survival has been documented after prolonged arrest ("no one is dead until warm and dead").[69] Drug administration intervals are doubled or withheld below 30 °C due to reduced metabolism.[70] Asystole or pulseless electrical activity should not prompt termination of efforts until adequate rewarming is achieved.[71].

Role of ECG in guiding treatment

ECG monitoring informs treatment at several levels. It detects early malignant arrhythmias, evaluates resolution of conduction disturbances as rewarming progresses, and differentiates hypothermia-induced changes from myocardial ischemia to avoid unnecessary interventions.[45] It also helps prioritize patients for extracorporeal rewarming when resources are scarce.[72]

Special clinical situations

In trauma-associated hypothermia, arrhythmias may be compounded by hypovolemia and metabolic acidosis, requiring integrated management.[73] In therapeutic hypothermia after cardiac arrest, ECG abnormalities such as Osborn waves and QT prolongation are common but usually benign; nonetheless, continuous monitoring is essential.[6] Pediatric and geriatric patients are particularly vulnerable due to limited physiological reserves, making ECG surveillance even more critical.[74]

Effective management of hypothermia with ECG abnormalities requires balancing rewarming techniques with arrhythmia prevention and resuscitation. Continuous ECG monitoring is indispensable for early detection of deterioration and for confirming recovery. Prognosis depends on rapid recognition, appropriate rewarming, and avoidance of harmful interventions.

Conclusion and future perspectives

Electrocardiographic changes in hypothermia provide unique insights into the pathophysiological processes of cold-induced cardiac dysfunction. From the presence of Osborn waves to QT prolongation and malignant arrhythmias, these manifestations serve as both diagnostic hallmarks and prognostic indicators. Early recognition of such abnormalities not only facilitates timely diagnosis but also guides appropriate therapeutic interventions, particularly regarding the need for advanced rewarming strategies and arrhythmia management.[75]

Despite decades of clinical observation and experimental studies, many aspects of hypothermia-related ECG alterations remain incompletely understood. Variability in the incidence of Osborn waves, differences in individual susceptibility to arrhythmias, and the prognostic significance of specific ECG patterns all represent areas of ongoing debate. Large-scale multicenter studies are needed to standardize the interpretation of ECG abnormalities in hypothermic patients and to clarify their independent prognostic value.[7]

Technological advances also hold promise. Artificial intelligence and machine learning algorithms have recently been applied to ECG interpretation in other settings, such as myocardial infarction and heart failure, with encouraging results. Their application to hypothermia could enhance early recognition of subtle ECG changes and improve triage decisions in pre-hospital and emergency settings. In addition, integration of continuous ECG monitoring with wearable devices and remote telemetry may allow earlier detection of hypothermia in high-risk populations, including elderly individuals, outdoor workers, and military personnel.[76]

Another frontier lies in the intersection of therapeutic hypothermia (targeted temperature management) and accidental hypothermia. Although both states share similar ECG alterations, their prognostic and therapeutic implications differ substantially. Comparative studies

exploring how induced hypothermia affects cardiac electrophysiology versus accidental hypothermia could provide new insights into protective versus deleterious mechanisms of cold exposure.[22]

In conclusion, hypothermia-induced ECG abnormalities remain a critical clinical tool, bridging diagnosis, prognosis, and treatment. Continued refinement in recognition, supported by advances in rewarming techniques and emerging digital health technologies, offers the potential to reduce mortality and improve neurological outcomes in this vulnerable patient population. Future research should focus on standardizing ECG criteria, evaluating novel therapeutic strategies, and leveraging artificial intelligence to enhance predictive accuracy and clinical decision-making.[77]

Contributorship

All of the authors contributed planning, conduct, and reporting of the work. All authors had full access to all data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Funding

No financial funding was received for this study.

Competing interests

All of the authors have no conflict of interest.

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