

STELLATE GANGLION BLOCK AS A BRIDGE TO DEFINITIVE GANGLIONECTOMY IN THE MANAGEMENT OF REFRACTORY VENTRICULAR TACHYCARDIA

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Summary

Electrical storm (ES) is a critical cardiac emergency characterized by recurrent, life-threatening ventricular arrhythmias occurring within a short period, most commonly in patients with structural heart disease, particularly those who have an implantable cardioverter-defibrillator (ICD) for secondary prevention, as the device records recurrent ventricular arrhythmias that characterize electrical storm. Despite advances in antiarrhythmic therapy and catheter ablation, some patients remain refractory to conventional management, resulting in significant morbidity and mortality. Recent evidence highlights the potential role of autonomic modulation, particularly stellate ganglion block (SGB), as an adjunctive or rescue therapy in these cases. This article presents the clinical case of a 75-year-old patient with ischemic cardiomyopathy and recurrent episodes of sustained ventricular tachycardia refractory to standard pharmacologic therapy (amiodarone, lidocaine, beta-blockers) and ICD interventions. This case emphasizes the importance of recognizing SGB as a viable, minimally invasive, and potentially life-saving intervention for patients with ES who do not respond to conventional treatment. Furthermore, as its effects are transient, SGB may serve as a diagnostic and bridging step toward definitive sympathetic modulation through surgical cardiac sympathetic denervation in recurrent or refractory cases.

Introduction

Electrical storm (ES) is a life-threatening arrhythmic emergency that often determines immediate survival in patients with structural heart disease. It is conventionally defined as ≥ 3 episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) within 24 hours requiring termination by antitachycardia pacing or defibrillation. ES occurs in up to 10–20% of patients with implantable cardioverter-defibrillators (ICDs) and carries an in-hospital mortality of 20–40% [1]. Beyond its high lethality, ES is also associated with frequent hospitalizations, impaired quality of life, and accelerated progression of heart failure [1, 2].

The burden of ES extends beyond arrhythmia recurrence itself. Repeated ICD shocks contribute to myocardial injury, heightened sympathetic activation, and psychological distress, perpetuating a vicious cycle of electrical instability. For these reasons, ES is both a marker of advanced structural heart disease and a direct cause of adverse outcomes [2]. Contemporary management requires a rapid, stepwise, multidisciplinary approach. Initial therapy typically involves intravenous antiarrhythmic agents such as amiodarone, lidocaine, or procainamide, combined with non-selective β -blockade (typically propranolol) to suppress adrenergic drive and blunt sympathetic surges. Mild to moderate sedation is recommended for patients experiencing psychological distress. In refractory cases where pharmacological management of electrical storms is ineffective, escalation to deep sedation with endotracheal intubation should be considered. If arrhythmias persist, ICD reprogramming with optimization of antitachycardia pacing strategies is used to reduce the shock burden. In hemodynamically unstable patients, mechanical circulatory support – such as extracorporeal membrane oxygenation (ECMO) or intra-aortic balloon counterpulsation

– may provide temporary stabilization. When feasible, urgent catheter ablation is pursued as a definitive therapy aimed at modifying or eliminating the arrhythmogenic substrate. In cases where pharmacological and device-based management fails to control electrical instability, growing attention has been directed towards autonomic modulation strategies targeting sympathetic overactivity [2]. Nevertheless, in a substantial subset of patients, arrhythmias remain refractory despite exhaustive pharmacological, device-based, and interventional measures [1, 2].

Sympathetic hyperactivation plays a central role in ES pathophysiology. Excess catecholamines increase intracellular calcium loading, shorten action potential duration heterogeneously, thereby increasing dispersion of ventricular repolarization and promoting reentrant circuit formation. The stellate ganglion – formed by the fusion of the inferior cervical and first thoracic sympathetic ganglia – provides dominant sympathetic input to the ventricles, particularly on the left side. Hyperactivity of this structure lowers the arrhythmic threshold and facilitates malignant electrical instability [3]. Stellate ganglion block (SGB) has emerged as a readily accessible bedside intervention for acute sympathetic suppression, while cardiac sympathetic denervation (CSD) offers a more definitive and durable solution in recurrent or refractory cases [3, 4]. This close mechanistic link has created a strong rationale for neuromodulatory strategies that interrupt cardiac sympathetic outflow. While thoracic epidural anesthesia (TEA) has shown clinical benefit, it is technically demanding and often contraindicated. In contrast, stellate ganglion block (SGB), performed via ultrasound-guided local anesthetic injection or radiofrequency neuromodulation, represents a simpler, bedside-accessible, practical and widely applicable option for acute sympathetic suppression. However, as the effect of stellate ganglion block is transient, patients who experience recurrence may benefit from definitive sympathetic modulation through surgical cardiac sympathetic denervation (CSD) [3, 4].

According to the 2022 European Society of Cardiology (ESC) and 2023 AHA/ACC/HRS guidelines, sympathetic blockade, including SGB, is recommended as a therapeutic option in patients with drug- and shock-refractory ES [1, 2]. Recent data, including the 2024 STAR multicenter study and a 2024 systematic review with meta-analysis, have demonstrated consistent efficacy and safety of SGB in this setting [3, 4]. However, evidence remains limited by the lack of randomized controlled trials and uncertainty regarding long-term outcomes, leaving its precise role within treatment algorithms unresolved [3, 4].

Thus, a stepwise approach from temporary sympathetic blockade to definitive denervation represents an evol-

ving paradigm in the management of refractory ventricular arrhythmias [3, 4].

Objective: To present a clinical case of refractory ventricular arrhythmias initially managed with stellate ganglion block (SGB) as rescue therapy, followed by surgical cardiac sympathetic denervation (CSD) for recurrence, and to review and discuss contemporary evidence on the stepwise application of autonomic modulation strategies in the management of refractory ventricular arrhythmias.

Materials and Methods

A clinical case analysis was performed using data collected from the patient's medical records and documentation during hospitalization from September 21 to September 30, 2025 at the Department of Cardiology, Kaunas Clinics, Lithuanian University of Health Sciences. A literature search was conducted using PubMed and ClinicalKey databases. In total, 26 English-language publications were selected and reviewed, all published within the past ten years.

Clinical Case

A 75-year-old male with a history of ischemic heart disease and recurrent ventricular arrhythmias was admitted because of drug-refractory recurrent ventricular tachycardia (VT) presenting with chest discomfort and palpitations. The patient reported pressure-like chest pain and generalized weakness that began several hours prior to admission. At home, no ICD discharges were recorded. On presentation, electrocardiography revealed sustained monomorphic VT with a rate of approximately 140–150 bpm, which was terminated by synchronized cardioversion.

The patient had a significant cardiovascular history, including myocardial infarctions in 2018 and 2022, percutaneous coronary intervention (PCI) with stenting of segments S7 and S12 in July 2021, and dual-chamber ICD (ICD-DR) implantation in 2019. He had chronic ischemic cardiomyopathy, heart failure (NYHA class II–III), arterial hypertension, paroxysmal atrial fibrillation, type 2 diabetes mellitus, and dyslipidemia. Long-term therapy consisted of amiodarone 200 mg daily, metoprolol succinate 47.5 mg twice daily and guideline-directed therapy for atrial fibrillation, heart failure, diabetes, and dyslipidemia. The patient denied any allergies and had no history of recent infection or surgery.

Upon admission in 2025-09-21, telemetry documented recurrent VT episodes despite an intravenous amiodarone bolus (300 mg) followed by continuous infusion, and correction of electrolyte disturbances (K^+ 3.5 mmol/L, Mg^{2+} 0.63 mmol/L). Laboratory findings showed BNP 429 pg/mL and high-sensitivity troponin 28.2 ng/L. Despite optimized pharmacological ma-

nagement, short-lasting VT episodes persisted, indicating a drug-refractory ES. A multidisciplinary team, including cardiologists and anesthesiologists, decided to perform SGB for acute sympathetic modulation and control of ventricular tachycardia. The procedure was performed in the cardiac intensive care unit under continuous monitoring. Following local skin infiltration with 2% lidocaine (5 mL), a left stellate ganglion block at the C6 level was performed under ultrasound guidance (out-of-plane technique). A total of 10 mL of 0.5% bupivacaine was injected slowly after confirming negative aspiration. The patient remained hemodynamically stable throughout the procedure.

After approximately 15 minutes, the patient developed transient Horner's syndrome (ptosis, miosis, facial flushing), confirming effective sympathetic blockade. No procedural complications were observed. The heart rate decreased from 70 bpm to 60 bpm, and no further VT episodes were recorded on continuous telemetry. The temporal relationship between SGB administration and immediate arrhythmia suppression suggested a causal autonomic modulation effect. During the subsequent one-week hospitalization, the patient's clinical condition remained stable. Continuous telemetry monitoring after the stellate ganglion block (SGB) revealed no recurrent ventricular tachycardia or ICD discharges. Electrolyte balance was maintained ($K^+ > 4.0$ mmol/L, $Mg^{2+} > 1.0$ mmol/L), and antiarrhythmic therapy was continued. The patient was monitored for one week after the blockade, since no recurrent arrhythmias occurred, surgical stellate ganglion denervation (destruction) was not

indicated. He was discharged with stable hemodynamics and rhythm control, with a plan that, should ventricular arrhythmias recur, surgical denervation would be considered during follow-up.

After less than one month, the patient was readmitted due to recurrent electrical storm despite optimized antiarrhythmic therapy, ECG demonstrated ventricular tachycardia (figure 1). Given the prior transient response to stellate ganglion block (SGB), a multidisciplinary team decided to proceed with left thoracoscopic cardiac sympathetic denervation (CSD).

The procedure included resection of the lower half of the left stellate ganglion and thoracic sympathetic chain (T2–T4) under general anesthesia. No intraoperative complications were observed. Postoperative two day telemetry and ECG demonstrated complete suppression of ventricular tachyarrhythmias (figure 2). The patient remained hemodynamically stable and was discharged in sinus rhythm on maintenance β -blocker therapy, metoprolol, and antiarrhythmic medication, amiodarone.

This case demonstrates a stepwise autonomic modulation strategy in the management of drug-refractory ventricular arrhythmias. Initial stellate ganglion block (SGB) provided transient arrhythmic suppression, while subsequent cardiac sympathetic denervation (CSD) resulted in sustained rhythm stability.

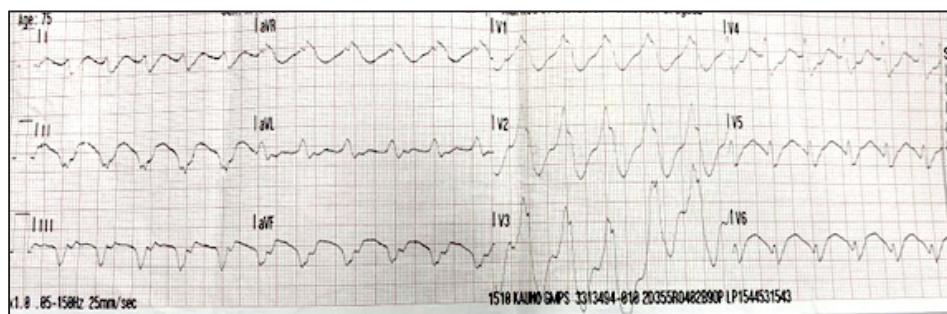


Figure 1. ECG demonstrating VT episode

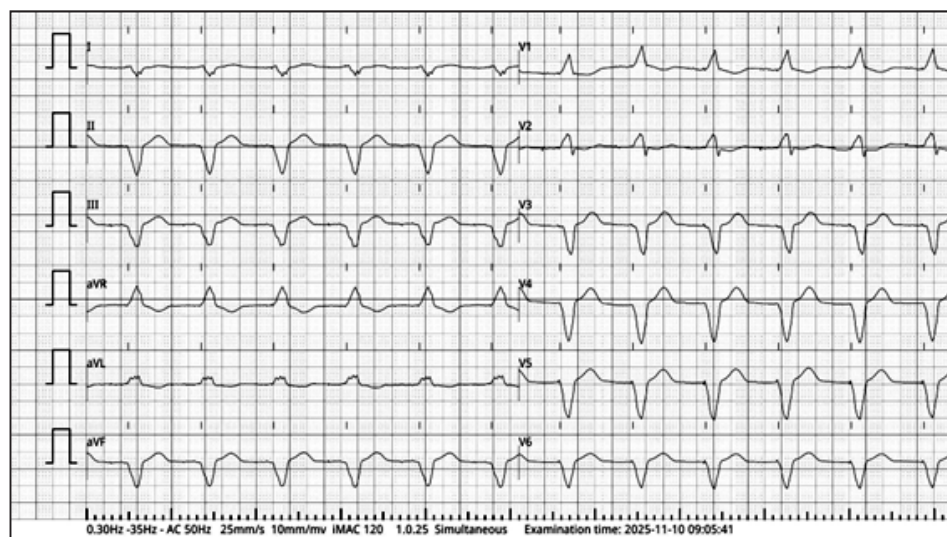


Figure 2. Sinus rhythm and effective stimulation

Together, these interventions illustrate the evolving therapeutic continuum from temporary sympathetic blockade to definitive denervation.

Literature review

Epidemiology. Ventricular tachycardia (VT) is defined as three or more consecutive ventricular beats at a rate exceeding 100 beats per minute and it typically presents as a wide-complex tachycardia [1]. A subset of VT manifests as torsade de pointes (TdP), a polymorphic ventricular tachycardia often associated with long QT syndrome (LQTS) – a group of inherited or acquired disorders characterized by delayed cardiac repolarization [5]. VT can also arise from structural heart disease, including ischemic cardiomyopathy or Purkinje fiber-mediated mechanisms, genetic channelopathies such as Brugada and short QT syndrome [6]. Electrical storm (ES) is defined as three or more episodes of sustained ventricular arrhythmia within 24 hours requiring termination through antitachycardia pacing, cardioversion, or defibrillation, most commonly in patients with an implantable cardioverter-defibrillator (ICD). While monomorphic VT is the most frequent form of ES, polymorphic VT and ventricular fibrillation can also precipitate ES [7]. Clinical presentations of VT range from self-terminating episodes causing syncope to malignant arrhythmias that degenerate into ventricular fibrillation (VF) and sudden cardiac arrest.

Diagnostics. The diagnosis of ES is established when a patient experiences three or more episodes of sustained VT or VF, or appropriate ICD interventions within a 24-hour period, with each episode separated by at least five minutes [1]. Continuous ECG monitoring or ICD review is essential to confirm arrhythmia type and frequency. The diagnostic evaluation should include identification of reversible triggers, such as acute myocardial ischemia, electrolyte imbalances (e.g., hypokalemia, hypomagnesemia), drug toxicity, and increased sympathetic activity [8]. Baseline investigations should comprise a 12-lead ECG, transthoracic echocardiography to assess left ventricular function and identify structural abnormalities, and, when indicated, coronary angiography or cardiac magnetic resonance imaging to define ischemic, structural and fibrotic arrhythmogenic substrates [9, 10]. Early and accurate diagnosis is essential for stabilizing the patient and guiding appropriate therapy.

Initial treatment. Initial management of VT includes intravenous antiarrhythmic drugs combined with sympathetic inhibition. According to the 2022 European Society of Cardiology (ESC) Guidelines on ventricular arrhythmias, intravenous amiodarone and non-selective beta-blockers (e.g., propranolol, esmolol) are the primary drugs used in patients with structural heart disease and ES. Intravenous

amiodarone should be initiated with a loading dose of 5 mg/kg infused over 20 minutes to 2 hours, which may be repeated two to three times within 24 hours if required. This is followed by a continuous infusion of 600–1200 mg per 24 hours for 8–10 days, after which treatment is transitioned to an oral maintenance dose of 200–400 mg daily once rhythm control is achieved and oral administration is feasible. This gradual loading approach allows adequate myocardial tissue saturation and promotes sustained antiarrhythmic efficacy [1]. Beta-blockers reduce adrenergic drive and lower arrhythmia recurrence. Non-selective agents, such as propranolol, may be more effective than β 1-selective agents in suppressing ES due to broader sympathetic inhibition [5, 11]. Mild to moderate sedation carries a Class IIb recommendation, Level of Evidence C, and is used to blunt sympathetic surge, particularly in patients receiving multiple ICD shocks or exhibiting severe agitation and tachycardia [5].

In patients with an ICD, reprogramming and optimization are essential. The ESC Guidelines recommend reducing unnecessary shocks through anti-tachycardia pacing (ATP) and simultaneous pharmacologic or autonomic therapy [1]. Excessive ICD shocks increase sympathetic activation and myocardial injury, creating a vicious cycle of recurrent arrhythmias. Catheter ablation is the definitive therapy for drug-refractory electrical storms whenever feasible. A 2023 meta-analysis demonstrated that early ablation significantly reduces long-term mortality and arrhythmia recurrence compared to conservative management [11]. Similarly, a 2024 analysis confirmed that patients undergoing ablation during ES experience improved one- and three-year survival and reduced recurrence rates, especially in patients with reduced ejection fraction [12]. Early referral to an electrophysiology center is therefore strongly encouraged once the patient is hemodynamically stabilized.

In patients with refractory shock or severe ventricular dysfunction, mechanical circulatory support – such as intra-aortic balloon pump, Impella, or veno-arterial extracorporeal membrane oxygenation (VA-ECMO) – may be necessary to maintain end-organ perfusion while definitive therapy (e.g., ablation) is performed [13]. Autonomic modulation techniques, including thoracic epidural anesthesia and SGB, are increasingly used as measures when conventional therapy fails or the desired results are not reached. A 2024 prospective, multicentre study provided evidence in favour of the effectiveness and safety of percutaneous SGB for the treatment of refractory ES [4]. These interventions offer a promising therapeutic option for patients who remain refractory to conventional medical and interventional treatments. In cases where pharmacologic, device-based, and catheter ablation strategies fail to suppress electrical instability, au-

tonomic modulation targeting sympathetic overactivity has emerged as an adjunctive therapeutic approach [1, 2, 19–22].

Indications for Stellate Ganglion Block: Rescue and Bridge Therapy. SGB is typically reserved for patients with electrical storms in whom conventional measures have been exhausted. Standard management includes intravenous antiarrhythmic drugs (e.g., amiodarone, lidocaine, procainamide), combined with β -adrenergic blockade and deep sedation, while ICD optimization and urgent catheter ablation represent the cornerstone of interventional strategies [14–15]. Nevertheless, in a significant proportion of patients, arrhythmias remain uncontrolled, either because drug therapy fails to suppress the arrhythmogenic substrate or because ablation is not technically feasible due to hemodynamic instability, extensive scarring, or limited access to electrophysiology expertise [16].

In such scenarios, SGB can provide rapid stabilization as a rescue therapy in the acute setting. By attenuating sympathetic outflow to the ventricles, it may immediately reduce the arrhythmia burden and interrupt the cycle of recurrent ICD shocks and hemodynamic deterioration [17, 18]. Unlike pharmacological therapy, its effect is immediate and independent of systemic pharmacokinetics, making it particularly valuable in critically ill patients where recurrent ventricular tachyarrhythmias can otherwise prove fatal within hours [17].

Several methods are available to achieve stellate ganglion modulation. The most common approach is local anesthetic injection, usually performed under ultrasound guidance. This technique provides rapid sympathetic blockade and is often used as an acute test or temporizing measure, but its effects are transient, lasting hours to days. To prolong benefit, neuromodulation techniques have been explored. Pulsed radiofrequency (PRF) alters ganglionic excitability without neurodestruction, offering intermediate efficacy with lower risk of nerve injury, while continuous radiofrequency (CRF) induces thermocoagulation and results in longer-lasting denervation, albeit with greater procedural risk [15]. However, current evidence for PRF and CRF in electrical storms remains limited, and larger studies are needed to establish their comparative efficacy and durability [13].

SGB is most often performed at the C6 or C7 level using ultrasound guidance, which improves accuracy and reduces complications compared with landmark-based techniques [14]. Clinical confirmation is typically provided by ipsilateral Horner's syndrome. Complications are rare and generally limited to transient hoarseness, dysphagia, hematoma, pneumothorax, or inadvertent intravascular injection. In contemporary practice, particularly with ultrasound guidance, SGB is considered a relatively safe intervention that can be performed even in critically ill patients [4, 18].

Beyond its role as an emergency intervention, SGB may also function as a bridge strategy. In patients who are poor candidates for immediate ablation or surgical cardiac sympathetic denervation, temporary neuromodulation through local anesthetic or continuous blockade can stabilize the rhythm and improve hemodynamic status until definitive therapies can be safely performed [4, 14–17]. In this respect, SGB expands the therapeutic window, buying critical time to transfer patients to specialized centers or prepare for invasive interventions.

Thus, SGB currently occupies a unique position in the treatment algorithm – as a salvage therapy in acute decompensation and as a bridge to more definitive interventions such as surgical cardiac sympathetic denervation (CSD) [14, 15, 19–22].

Cardiac Sympathetic Denervation (CSD): Definitive Therapy and Long-term Outcomes. Cardiac sympathetic denervation (CSD) represents a definitive autonomic modulation strategy for patients with drug- and ablation-refractory ventricular arrhythmias. By resecting or interrupting the lower half of the left stellate ganglion and thoracic sympathetic chain (T2–T4), CSD reduces sympathetic efferent input to the heart, thereby decreasing catecholamine-mediated arrhythmogenic triggers [19]. Initially developed for the management of long QT syndrome, the procedure has since gained increasing use in structural heart disease and electrical storm, especially in cases refractory to pharmacologic and catheter-based therapies [19, 20].

The clinical efficacy of CSD has been consistently demonstrated across observational and prospective studies. In a large multicenter cohort, Vaseghi et al. reported that surgical CSD significantly reduced the frequency of ICD shocks and ventricular tachyarrhythmia recurrences in patients with refractory electrical storm [19]. Similarly, Schwartz et al. emphasized its role as a life-saving therapy for malignant ventricular arrhythmias, showing sustained benefits over long-term follow-up [20]. A 2022 meta-analysis by Meng et al. confirmed that left-sided CSD resulted in approximately 70% reduction in ICD shocks and 60% reduction in VT/VF recurrence, with a favorable safety profile and low perioperative complication rate [21]. More recently, Ajijola et al. demonstrated that CSD provides durable suppression of electrical storms and improved survival in patients with advanced structural heart disease [22].

Current evidence supports CSD as a definitive treatment option in the stepwise management of refractory ventricular arrhythmias. Compared with temporary sympathetic blockade, CSD offers a permanent reduction of sympathetic drive and a corresponding decrease in arrhythmic burden. While most data support left-sided procedures, bilateral CSD may

be considered in selected cases of persistent arrhythmia after unilateral intervention [19–22]. Despite growing evidence and procedural refinement, CSD remains primarily indicated in highly selected patients treated in specialized centers. Future studies are needed to optimize patient selection, assess the long-term prognostic impact, and define the role of CSD in earlier stages of arrhythmia management. Collectively, these studies establish CSD as an effective and safe intervention for the suppression of refractory ventricular arrhythmias. Thus, within the evolving framework of autonomic modulation, CSD complements temporary sympathetic blockade (SGB) as the definitive step toward durable arrhythmic control and long-term rhythm stabilization [19–21].

Stepwise Autonomic Modulation: From SGB to CSD.

International guidelines currently recognize autonomic modulation — including both stellate ganglion block (SGB) and cardiac sympathetic denervation (CSD) — as adjunctive options for patients with drug- and shock-refractory electrical storms when conventional pharmacologic and catheter-based interventions have failed. The 2022 ESC Guidelines [1] and the 2023 AHA/ACC/HRS consensus [2] position these techniques within a stepwise therapeutic framework, where temporary sympathetic blockade (SGB) serves as an acute stabilizing measure, whereas surgical denervation (CSD) offers a more definitive and durable suppression of arrhythmic activity [19–22]. This evolving paradigm underscores the growing clinical recognition of excessive sympathetic activity as a key driver of electrical instability and highlights the therapeutic potential of its modulation in carefully selected patients. Although clinical experience with autonomic modulation (particularly SGB) is expanding and recent multicenter data such as the STAR study [3] and a 2024 meta-analysis [15] have reported encouraging results, the overall evidence base remains limited. Most available data come from case reports, small single-center series, or retrospective analyses, and robust long-term outcome data are lacking. As a result, current guidelines stop short of assigning these interventions a more definitive role in the treatment algorithm, restricting their use to rescue therapy or as a bridge to catheter ablation or surgical sympathetic denervation [14,15,19–22].

Future perspectives depend on the accumulation of higher-quality evidence and on integration of autonomic modulation into standardized treatment pathways. Ongoing investigations, including the STAR trial [3] and recent multicenter studies of surgical CSD [19, 22], aim to clarify optimal timing, durability, and patient selection. Ultimately, a stepwise approach — from temporary sympathetic blockade to definitive denervation — may evolve from a rescue concept into a structured component of guideline-directed

therapy for refractory ventricular arrhythmias [1, 2, 19–22].

Complications of Ventricular Tachycardia. In patients with nonischemic cardiomyopathy (NICM), VT is associated with increased risk of both nonsudden cardiovascular death (NSCVD) and overall mortality, suggesting that VT may serve as a marker of advanced myocardial disease [23]. NICM patients are also at heightened risk for progression to end-stage heart failure and sudden cardiac death (SCD) secondary to ventricular arrhythmias [23]. Evidence suggests a positive correlation between VT burden and mortality, though causality remains unproven. ICD shocks have been implicated in worsening clinical outcomes, but it remains uncertain whether VT burden itself is a modifiable risk factor for death [24]. Patients experiencing ES demonstrate markedly increased risk of cardiac mortality, as highlighted in the meta-analysis, that included 13 studies [25]. Smaller studies corroborate that ES independently predicts VT recurrence and rehospitalization [26]. VT, particularly when presenting as ES, is a strong prognostic indicator of adverse outcomes in patients with structural heart disease and channelopathies. In NICM, VT identifies patients at elevated risk for SCD, NSCVD, and progression to end-stage heart failure. These findings underscore the importance of timely recognition, risk stratification, and tailored therapeutic interventions, including antiarrhythmic therapy and ICD optimization, to reduce arrhythmic recurrence and improve long-term survival.

Conclusions

1. Stellate ganglion block (SGB) provides immediate but transient attenuation of sympathetic activity and is an effective acute intervention for refractory ventricular arrhythmias and electrical storm unresponsive to guideline-directed therapies.

2. As a minimally invasive autonomic modulation strategy, SGB reliably decreases arrhythmic burden and frequently functions as a hemodynamic stabilizing bridge to definitive interventions.

3. In patients with recurrent or persistent arrhythmias despite SGB, cardiac sympathetic denervation offers a more durable autonomic interruption, and early escalation to this therapy may enhance arrhythmic control and improve long-term clinical outcomes.

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**ŽVAIGŽDINIO MAZGO BLOKADA KAIP
TARPINĖ PRIEMONĖ PRIEŠ GALUTINĘ ŠIRDIES
SIMPATINĖS NERVŲ SISTEMOS DENERVACIJĄ
GYDANT REFRAKTERINĘ VENTRIKULINĘ
TACHIKARDIJĄ**

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Raktažodžiai: skilvelinė tachikardija, elektrinė audra, žvaigždinio mazgo blokada, širdies simpatinės nervų sistemos denervacija, autonominės nervų sistemos moduliacija, refrakterinė aritmija.

Santrauka

Elektrinė audra (EA) – gyvybei pavojinga būklė, kuriai būdingos pasikartojančios skilvelių aritmijos, pasireiškiančios per trumpą laikotarpį. Dažniausiai ji pasitaiko pacientams, sergantiems struktūrinėmis širdies ligomis, ypač tiems, kuriems antrinei prevencijai implantuojamas kardioverteris-defibriliatorius. Nepaisant pažangaus antiaritminio gydymo ir kateterinės abliacijos galimybių, kai kurie pacientai lieka atsparūs įprastiniam gydymui, o tai lemia didelį sergamumą ir mirtingumą. Naujausiais duomenimis, autonominės nervų sistemos moduliacijos, ypač žvaigždinio mazgo blokados, gali būti ne tik gydymo metodas, bet ir tarpinė priemonė prieš galutinę širdies simpatinės sistemos denervaciją. Šiame straipsnyje pateikiamas 75 metų amžiaus paciento, sergančio išemine kardiomiopatija ir patiriančio pasikartojančius ilgalaikės skilvelinės tachikardijos epizodus, nepasiduodančius standartiniam farmakologiniam gydymui ir intervencijoms, klinikinis atvejis; pacientui buvo atlikta žvaigždinio mazgo blokada kaip pereinamoji terapija prieš galutinę autonominės sistemos denervaciją. Be to, pateikiama naujausios mokslinės literatūros apžvalga, kurioje pabrėžiama žvaigždinio mazgo blokados taikymo svarba ir terapinis požiūris, naudojant šią procedūrą ne tik kaip gydymo pasirinkimą, bet ir kaip pereinamąją terapiją.

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