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Fatal and nonfatal cardiac arrhythmias in electrical injury

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Abstract. ***Background.*** *Electrical injuries are among the most common public health problems and are frequently associated with high morbidity and mortality.* ***Evidence acquisition.*** *Literature sources were included in the study if they: 1) were published in Ukrainian, Spanish, or English; 2) reported on cardiac arrhythmias associated with electrical injury; 3) provided data on the prevalence of cardiac arrhythmias in electrical injury; 4) used an observational study design (cohort or cross-sectional). A retrospective literature search was conducted using a spatial-vector descriptive model, supplemented by a manual review of the referenced articles.* ***Evidence synthesis.*** *The heart is affected more frequently than other internal organs because electrical current usually follows the path of least resistance in the body — along blood vessels and nerves, toward the heart. Extensive surface burns and a vertical current pathway through the torso indicate an increased risk of cardiac damage. The most common manifestation of cardiac injury due to electrical exposure is arrhythmia, which typically occurs immediately after the electric shock. Conduction disturbances may result from alterations in myocytes surrounding the sinoatrial and atrioventricular nodes and are not necessarily related to myocardial ischemia or arterial injury. The most frequent ECG findings upon hospital admission were sinus bradycardia and sinus tachycardia. Other documented arrhythmias included newly diagnosed atrial fibrillation, frequent multifocal atrial complexes, sinus node arrest with atrial escape rhythm, prehospital-resolved ventricular fibrillation, ventricular bigeminy, and recurrent nonsustained ventricular tachycardia. Arrhythmias caused by electrical injury are mostly functional in nature, generally benign, and tend to resolve within the first few hours after hospitalization. Ventricular fibrillation is the most common fatal arrhythmia, occurring in 60 % of patients in whom the electrical current passes transthoracically. Alternating current has a greater propensity to induce ventricular fibrillation, whereas direct current more often causes asystole.* ***Conclusions.*** *Cardiac arrhythmias are the leading and most dangerous complications of electrical injury. Their incidence is directly dependent on the characteristics of the electrical current, its pathway through the body, the duration of exposure, body mass, and the extent of myocardial structural damage. In some cases, arrhythmias resulting from electrical injury may resolve spontaneously. Cardiac arrhythmias following electrical trauma can also occur in a delayed manner. Timely resuscitation measures can restore normal cardiac rhythm.*

Keywords: *electrical current; electrical injury; current pathways through the body; arrhythmias; pathophysiology*

Introduction

Electrical injuries (EI) are among the most common public health problems and are frequently associated with high morbidity and mortality. In survivors, particular concern arises from cardiac involvement and the risk of delayed cardiovascular complications. Patients with electrical injuries are considered to be at high risk of developing cardiac arrhythmias. Due to the limited number of studies, there are no universally accepted guidelines for assessing risk or managing arrhythmic complications following electrical trauma [1].

Evidence acquisition

Literature sources were included in the study if they: 1) were published in Ukrainian, Spanish, or English; 2) reported on cardiac arrhythmias associated with EI; 3) provided data on the prevalence of cardiac arrhythmias in electrical injury; and 4) used an observational design (cohort or cross-sectional). A retrospective literature search was conducted using a spatial-vector descriptive model, supplemented by a manual review of referenced articles. A total of 41 sources were selected, 97.6 % were published within the past 10 years.

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Evidence synthesis

Damage to the cardiovascular system correlates with the pathway of the electrical current [2]. Transthoracic current flow can lead to cardiac complications, manifested primarily as arrhythmias, conduction disturbances, and myocardial tissue injury, largely depending on the intensity of the current [1, 3, 4]. The heart is affected more frequently than other internal organs because electrical current typically follows the path of least resistance in the body — along blood vessels and nerves, directing the flow toward the heart [5]. The extensive network of neural fibers throughout the heart, together with the conductivity of the blood passing through its chambers, makes it particularly vulnerable to EI [6]. Its central anatomical location within the thoracic cavity also exposes the heart to currents flowing both horizontally (from hand to hand) and vertically (from head to foot or from hand to foot), increasing the probability of pathological cardiovascular effects [2, 5, 7, 8].

The current typically flows from the point of contact with the electrical source to the exit point through tissues offering the least resistance. Extensive surface burns and a vertical current pathway through the torso indicate an increased risk of cardiac damage [4, 9–11].

Dextrocardia, a heart defect first described in the early 17th century, is characterized by right-sided positioning of the heart with the base-apex axis directed to the right. It is a very rare condition, with an incidence of approximately 1 in 12,000 cases [12]. In such an anomaly, the current pathway "right hand — right foot" poses a greater risk than under normal anatomical conditions [13, 14].

An external electrical current cannot interfere with the generation of cardiac impulses unless it is straightly directed toward the heart (the lower loop of electrical current) [15].

The most common manifestation of cardiac injury resulting from electrical exposure is arrhythmia, which usually occurs immediately after EI [2, 3, 11, 16–18]. The severity of cardiac involvement ranges from harmless transient sinus tachycardia to fatal ventricular arrhythmias and severe conduction disturbances that, in some cases, require permanent pacemaker implantation [2]. Arrhythmias caused by EI are mostly functional [19], generally benign, and tend to resolve within the first few hours after hospitalization [2, 20, 21]. Many individuals affected by EI experience palpitations, which is often the reason for seeking emergency medical care [11]. Electrocardiographic abnormalities of varying severity are detected in 10–20 % of patients admitted after EI [20, 22], while cardiac rhythm disturbances occur in approximately 14 % of cases [17]. Some arrhythmias are incidentally detected in about 3 % of patients under continuous cardiac monitoring following EI [12].

Low-voltage household alternating current is considered quite dysrhythmogenic and may induce ventricular arrhythmias [21]. However, clinically significant arrhythmias after low-voltage exposure are rare and often resolve spontaneously without medical intervention [3, 9, 23].

The pathogenesis of cardiac arrhythmias remains unclear and is likely multifactorial. Myocardial necrosis, altered concentrations of sodium, potassium, and adenosine triphosphate, as well as changes in cardiomyocyte membrane permeability act as trigger factors for both fatal and non-fatal arrhythmias [3]. Potential predisposing factors for fatal arrhythmias include a reduction in Purkinje fibers, the formation of large and focal areas of myocardial necrosis (cardiomyolysis), and necrosis of vascular smooth muscle cells in the coronary arteries [24].

Conduction disturbances may result from alterations in cardiomyocytes surrounding the sinoatrial and atrioventricular nodes and are not necessarily associated with myocardial ischemia or arterial injury [21]. The arrhythmogenic effects of EI are therefore not considered primarily due to myocardial necrosis. However, histopathological findings indicate that the most common microscopic feature in victims of EI is myofibrillar rupture, which leads to heterogeneity within the heart's electrical conduction system [1].

Typical flash or thermal burns may be considered a "distracting injury", which can cause the clinician to overlook or underestimate a potentially dangerous arrhythmia [23].

Among patients presenting to emergency departments after EI, the diagnosed arrhythmias include supraventricular (sinus arrhythmia, tachycardia and bradycardia, atrial extrasystoles, or atrial fibrillation) and ventricular arrhythmias (ventricular extrasystoles, tachycardia, or fibrillation) [5, 8, 9, 11, 12, 19–21, 25–27]. Supraventricular arrhythmias are less common than ventricular arrhythmias following EI [28].

The most frequent ECG abnormalities on hospital admission were sinus bradycardia (< 60 bpm, 10.4 %) and sinus tachycardia (> 100 bpm, 4.4 %). Other documented arrhythmias included newly diagnosed atrial fibrillation, frequent multifocal atrial complexes, sinus node arrest with atrial escape rhythm, ventricular fibrillation resolved at the prehospital stage, ventricular bigeminy, and recurrent nonsustained ventricular tachycardia [3, 29]. Cases of atrial fibrillation have been reported after contact with high-voltage power lines. In most of these cases, the rhythm returned spontaneously or after administration of quinidine or digitalis [30]. Isolated reports have also documented flutter-type arrhythmias [3].

The re-entry mechanism represents an arrhythmogenic process in which an electrical impulse within the heart becomes self-perpetuating, circulating repeatedly in a loop, and thereby causing tachyarrhythmia. This phenomenon occurs when there are two pathways of electrical conduction (a primary and an accessory pathway), allowing the impulse to flow down one pathway and return via the other, resulting in tachycardia or extrasystole [31].

Although most life-threatening events occur immediately after electrical exposure, *delayed* ventricular arrhythmias (up to 12 hours post-incident, in both low- and high-voltage injuries) have been repeatedly reported, which required careful observation [21]. Cardiac complications and arrhythmias are more frequently observed in patients exposed to high-voltage electrical current [12].

Although patients without initial ECG abnormalities may appear safe for discharge, the results of targeted follow-up studies question their safety due to the risk of delayed arrhythmias [23]. While the occurrence of late malignant arrhythmias is relatively rare [12], concerns regarding the potential for delayed arrhythmogenesis are the reason why patients are often hospitalized for monitoring, even in the

absence of risk factors, arrhythmic symptoms, or ECG abnormalities [26].

It is believed that delayed arrhythmias result from changes in the membrane potential of fibrotic cardiac tissue. Transient repolarization abnormalities resembling Brugada syndrome may develop due to imbalances in ionic currents, producing an arrhythmogenic effect [1, 9]. Tissue injury leads to temporary and localized disturbances in sodium and potassium transport, hyperkalemia, and increased activity of the Na^+/K^+-ATPase pump, which together promote the formation of arrhythmogenic foci with enhanced automaticity and triggered activity [2, 7, 11].

Direct cardiomyocyte injury, myocardial necrosis, alterations in adenosine triphosphate concentration, and changes in cell membrane permeability represent the morphological substrate for both fatal and non-fatal cardiac arrhythmias following electrical injury [12, 30]. When the cardiac conduction system is affected, bundle branch blocks and varying degrees of atrioventricular block may occur [3, 7, 11, 12, 19–21, 25, 27]. Complete atrioventricular block is rare, but in some cases, implantation of a permanent pacemaker is indicated [2, 8, 25]. Partial intraventricular conduction blocks persisting for up to 10 days have been described, while nodal rhythms have been observed in certain patients for up to 24 hours [30].

Hypothetically, the mechanism of electrically induced cardiac arrhythmias involves initial myocardial damage followed by scar formation, which leads to abnormal electrical activity within the heart. Significant bradycardia may result from disruption of the normal conduction system, which can manifest months or even years after the initial injury [5].

Isolated premature atrioventricular complexes and ventricular premature contractions typically occur within the first few hours after EI [3, 7, 11, 12, 21, 26, 29].

Nonspecific ECG changes may be the only sign of cardiac injury and include ST-segment abnormalities, which often persist for several weeks after trauma, as well as QT interval prolongation [2, 4, 5, 22, 30]. The latter indicates delayed electrical repolarization, which may lead to ventricular arrhythmias. Standards for corrected QT intervals vary, but values above 460–480 ms are generally considered pathological [5].

Currently, 24-hour Holter monitoring is regarded as the best method for timely detection of arrhythmias [3].

The susceptibility of cardiac tissue to the potentially fatal arrhythmogenic effects of external electrical fields distinguishes it from other organ structures [5, 7]. Electrically induced fatal arrhythmias typically occur immediately after electrical injury and represent the most common cause of death in electrocution cases [3, 9, 19, 27].

Disturbances of cardiac electrical impulse rhythms due to exposure to high-voltage current (> 20 000 V, usually direct current) are most often accompanied by acute electrical myocardial injury, leading to immediate sudden cardiac arrest as a result of asystole or pulseless electrical activity [5, 7, 8, 15, 16, 20, 21, 23, 30, 32, 33].

Both alternating current at 50–60 Hz and direct current above 5 A can induce sudden asystole without preceding fibrillation, as the heart remains in a state of sustained contraction [34]. Spontaneous restoration of sinus rhythm may occasionally occur after asystole in cases of electrical trauma. However, since respiratory paralysis persists longer than this process, the rhythm may deteriorate into ventricular fibrillation due to circulatory hypoxia [20, 21, 34].

A current of 50 mA or more at 50–60 Hz, passing through the body, typically induces ventricular fibrillation within 1–3 seconds [34]. In the worst cases, low-voltage electrical energy traversing the heart can trigger pulseless ventricular tachycardia or ventricular fibrillation, which usually occurs immediately after the electrical exposure. Patients in this cohort often presented with such arrhythmias upon hospital admission, although they were mostly young and without prior medical history [4, 5, 7, 23, 30].

Ventricular fibrillation is the most common fatal arrhythmia, seen in 60 % of patients with transthoracic current flow [8, 20, 21, 32].

This potentially lethal arrhythmia can also develop in a delayed manner, days or even months after the initial injury [7, 23].

During the development of ventricular fibrillation induced by direct current, the direction of the electrical pathway — whether ascending (anode at the caudal part of the body and cathode at the cranial part) or descending — is significantly important. Ascending direct current is considerably more dangerous than descending current in the same orientation, because the cathode increases the excitability of the sinoatrial node, while the anode decreases it. Cardiac impulses are regulated by electrical potentials generated by the cells of the natural pacemaker located in the sinoatrial node on the posterior wall of the right atrium [15]. The sinoatrial and atrioventricular nodes, which are responsible for the generation and conduction of impulses within the heart, may be more vulnerable to electrical injury than other cardiac cells, as autopsy studies demonstrate widespread cell death primarily in these conduction pathways [5]. In an ascending current pathway, the sinoatrial node is under the excitatory influence of the cathode, whereas the apex of the heart is suppressed by the anode. In a descending current pathway, the sinoatrial node is inhibited by the anode, while excitability at the apex is increased by the cathode. The excitation originating from the sinoatrial node in ascending current encounters conduction suppression along its pathway. When conduction falls below a critical threshold, ventricular fibrillation occurs. Conversely, the wave of excitation originating from an anode-inhibited sinoatrial node is accelerated along the pathway under cathodal influence in descending current. Based on these mechanisms, ascending current establishes conditions favorable for the development of ventricular fibrillation throughout the duration of the closed electrical circuit, whereas in descending current, such conditions arise only at the moment of the circuit interruption [31].

From a biological perspective, voltage represents a measure of pressure or electrical energy force passing through a conductor, whereas current primarily reflects the rate of electrical flow [25, 35, 36]. It is the flow of current through the body that compresses the heart or induces its fibrillation, potentially resulting in death. Ventricular fibrillation is the most common arrhythmia caused by electrical injury and constitutes the primary cause of death from electrical trauma, particularly with low-voltage alternating current [3, 5, 15, 25, 26, 34–36].

Both alternating and direct current can disrupt normal cardiac rhythm and induce ventricular fibrillation. However, alternating current is more hazardous than direct one in the generation of multiple excitations [37]. Accordingly, alternating current has a higher propensity to cause cardiac fibrillation, whereas direct current primarily leads to cardiac arrest [38].

The electrical current capable of inducing ventricular fibrillation when passing through the body is called *the fibrillating current*, and its minimal value is termed *the critical fibrillating current* [34].

The threshold fibrillating current is influenced by numerous factors. Its leading triggers are the duration of current exposure and body mass. The threshold for ventricular fibrillation is inversely proportional to the square root of the exposure duration and directly proportional to body mass. When the heart is exposed to gradually increasing electrical current, myocardial susceptibility to fibrillation initially rises and then decreases at higher current intensities.

Based on these thresholds, the touch voltage for alternating current should not exceed 50 V and for direct current — 120 V in healthy adults to avoid life-threatening situations. For children, the permissible touch voltage is set at 60 V for direct current [37].

Low-voltage alternating current with a frequency of 50–60 Hz passing through the thorax for a fraction of a second can induce ventricular fibrillation at current levels of 60–100 mA; for direct current, approximately 300–500 mA is required. If the current has a direct pathway to the heart (e.g., via a cardiac catheter or pacemaker electrodes), less than 1 mA (alternating or direct current) can induce ventricular fibrillation [6, 7, 21, 26, 34–36, 39].

When current passes through the human body for 10 seconds or longer, the thresholds for ventricular fibrillation become more distinct, amounting to 40 mA for alternating current (15–100 Hz) and 140 mA for direct current [37].

Main mechanisms underlying the development of ventricular fibrillation:

— direct effects of electrical current on cardiomyocytes and the cardiac conduction system lead to alterations in cardiac electrical potentials;

— electron flow through the heart disrupts all myocardial functions, including automaticity, excitability, conductivity, and contractility;

— the vascular component manifests as injury to the coronary arteries;

— irritation of cardiomyocytes due to the formation of a persistent reentrant wave (pseudo-Wolff-Parkinson-White syndrome);

— generation of paradoxical excitation potentials with opposite orientations in different regions of the myocardium;

— shock reactions with circulatory centralization during prolonged exposure lead to current irradiation to the heart [40];

— premature contractions can sometimes evolve into ventricular fibrillation and become fatal if treatment is not initiated promptly [11, 25], as cardiomyocytes begin to contract independently, potentially causing circulatory arrest [15];

— circulatory centralization followed by decentralization under electrical exposure induces tissue and systemic hypoxia and hypercapnia, with secondary injury to the vasomotor center of the brainstem and development of coronary spasm [40].

Analysis of fatal electrical injuries demonstrates that the duration of current flow through the body significantly influences the outcome of electrical trauma: the longer the duration of current exposure, the higher the risk of severe or fatal consequences. Cardiac sensitivity to electrical current varies across different phases of the cardiac cycle. The probability of developing ventricular fibrillation depends not only on the physical characteristics of the current but primarily on the phase of the cardiac cycle during which the current passes through the heart's projection. This dependence can be explained by the fact that as the duration of current exposure on living tissue increases, the cumulative pathological effects on the body also increase, along with the probability that the current aligns with the T-wave phase of the cardiac cycle, when the heart is most vulnerable at the end of ventricular contraction during the transition to relaxation. This phase lasts approximately 0.2 seconds. When the duration of current flow equals or exceeds the length of the cardiac cycle (0.75–1 second), the current interacts with all phases of the cardiac cycle, including the highly vulnerable T-wave phase. Conversely, if the current duration is shorter than the cardiac cycle by 0.2 seconds and does not align with the T-wave, the probability of ventricular fibrillation decreases sharply [34].

It should be noted that ventricular fibrillation can sometimes be reversible, when the heart spontaneously returning to normal rhythm shortly after fibrillation [20]. If exposure to low-voltage current is brief (1–2 seconds) and does not cause structural cardiac damage (e.g., burns or heating), the heart usually restores normal sinus rhythm spontaneously after current cessation [20, 32, 34], although cardiac disturbances may occasionally persist [5].

Conclusions

1. Cardiac arrhythmias are primary and dangerous complications of electrical trauma.

2. The incidence of cardiac arrhythmias is directly related to the characteristics of the electrical current, its pathway through the body, the duration of exposure, body mass, and structural myocardial damage.

3. In certain cases, cardiac arrhythmias induced by electrical trauma may resolve spontaneously.

4. Cardiac arrhythmias following electrical injury can occur in a delayed manner.

5. Timely resuscitative interventions can restore normal cardiac rhythm.

References

1. Pilecky D, Vamos M, Bogyi P, et al. Risk of cardiac arrhythmias after electrical accident: a single-center study of 480 patients. Clin Res Cardiol. 2019 Aug;108(8):901-908. doi: 10.1007/s00392-019-01420-2.

2. Koren O, Paz E, Rozner E, Mahamid M, Turgeman Y. Late myocardial sequelae of electrical injury. Clin Case Rep. 2020 Oct 27;8(12):3408-3411. doi: 10.1002/ccr3.3413.

3. Dhital BM, Regmi S, Regmi SR, et al. Cardiac Arrhythmias in Patients with Electrical Injury: Hospital Based Study. Nepalese Heart Journal. 2020;17 (2):29-32. doi: 10.3126/njh.v17i2.32675.

4. Gentges J, Schieche C. Electrical injuries in the emergency department: an evidence-based review. Emerg Med Pract. 2018 Nov;20(11):1-20.

5. Waldmann V, Narayanan K, Combes N, Marijon E. Electrical injury. BMJ. 2017 Apr 12;357:j1418. doi:10.1136/bmj.j1418.

6. Maritz SM, Motsepe TA, Gamizelo S. Electrocution induced cardiac dysrhythmia: an Izinyoka case. Int Surg J. 2025 Mar;12(3):395-398. doi: 10.18203/2349-2902.isj20250575.

7. Zemaitis MR, Lopez RA, Huecker MR. Electrical Injuries. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2025 Jan.

8. Houshmand G, Sadeghpour M, Tabesh F. Delayed cardiac consequences unveiled by magnetic resonance imaging in a high-voltage electric shock survivor. ESC Heart Fail. 2025 Jun;12(3):2377-2381. doi: 10.1002/ehf2.15251.

9. Farzan R, Ziabari SMZ, Jafaryparvar Z, Homaierad E, Ismaili Shoja E, Tolouei M. A Review of Electrocardiography Changes in Electrical Burn Injury: Is It Time To Revise Protocol? Ann Burns Fire Disasters. 2023 Jun 30;36(2):132-138.

10. Jingo K, Kondo Y, Hirano Y, et al. Evaluating the risks of arrhythmia following electrical injury: Two cases of electrical injuries in the upper limbs. SAGE Open Med Case Rep. 2020 May 8;8:2050313X20920421. doi: 10.1177/2050313X20920421.

11. Makharia A, Tiwari V, Barupal KG, Lakhotia M. Atrial fibrillation - A rare arrhythmia after electrical burns. Indian Journal of Burns. 2022 Jan-Dec; 30(1):91-93. doi: 10.4103/ijb.ijb_32_21.

12. Rahman SO, Halder KC, Arif Anam A, Akand S. Prevalence of Cardiac Arrhythmias, Clinical Spectrum and Complications of Electrical Injury. Glob Acad J Med Sci. 2022;4(4):194-199. doi: 10.36348/gajms.2022.v04i04.007.

13. Volkov OO, Dzjak LA, Yekhalov VV, Zozulya OO, Klygunenko OM, authors; Dzjak LA, Klygunenko OM, editors. Mechanical asphyxia. Dnipro: LIRA; 2019. 188 p. Ukrainian.

14. Yekhalov VV, Kravets OV, Krishtafor DA. Electric shock: a clinical lecture. Emergency Medicine (Ukraine). 2022;18(5):18-28. Ukrainian. doi: 10.22141/2224-0586.18.5.2022.1507.

15. Almusawi YK, Al-Sammarraie MRH, Shikara MD. The effect of an electric current on human body (a review). Al-Esraa University College Journal for Medical Sciences. 2023;4(5):2. doi: 10.70080/2790-7937.1016.

16. Guimarães F, Camões J, Mesquita A, Gomes E, Araujo R. A Case Report: Low Voltage Electric Injuries Culminating in Cardiac Arrest and Direct Lung Injury. Cureus. 2020 Oct 30;12(10):e11261. doi: 10.7759/cureus.11261.

17. Xhepa G, Isaraj S, Zikaj G, Kola N. Electrical Burns in Albania and their Treatment: A Review of Cases Treated in 2019—2020. Open Access Maced J Med Sci. 2023 May 4;11(B):620-626. doi: 10.3889/oamjms.2023.11634.

18. Başaran A, Gürbüz K, Özlü Ö, Demir M, Eroğlu O, Daş K. Electrical burns and complications: Data of a tertiary burn center intensive care unit. Ulus Travma Acil Cerrahi Derg. 2020 Mar;26(2):222-226. English. doi: 10.14744/tjtes.2019.65780.

19. Syplivyi VO, Dotsenko VV, Petrenko GD, et al. Frostbite: classification, mechanism of injury, clinical signs, first aid and treatment. Electric shock: mechanism of electric shock, clinical signs, diagnostics, first aid and its features: methodological guidelines for practical classes and independent work of 3rd year students. Kharkiv: KhNMU; 2020. 28 p. Ukrainian.

20. Children's Health Queensland Hospital and Health Service. Electrocution and Electrical Injury - Emergency Management in Children V3.1: Guidelines (CHQ-GDL-00735). Brisbane, Queensland: Children's Health Queensland; 2025. 13 p.

21. O'Keefe KP. Electrical injuries and lightning strikes: Evaluation and management. UpToDate. 2025 Jul 22. Availble from: https://www.uptodate.com/contents/electrical-injuries-and-lightning-strikes-evaluation-and-management.

22. Ghorbel I, Moalla S, Abid A, Karra A, Ennouri K. The Specificities of Electrical Burn Healing. In: Chiriac A, editor. Scars. London, UK: IntechOpen; 2019. doi: 10.5772/intechopen.83582.

23. Corrall S, Laws S, Rice A. Low-voltage electrical injuries and the electrocardiogram: is a 'normal' electrocardiogram sufficient for safe discharge from care? A systematic review. Br Paramed J. 2023 Dec 1;8(3):27-36. doi: 10.29045/14784726.2023.12.8.3.27.

24. Kozlov SV, Bachinskyi VT, Vanchuliak O, Savka IG, Kostenko YeYa. Defeat technical electric current (retrospective analysis of mortal cases). Forensic medical examination. 2019;1:59-61. doi: 10.24061/2707-8728.1.2019.12.

25. Beton O, Efe TH, Kaya H, et al. Electrical Injury-Induced Complete Atrioventricular Block: Is Permanent Pacemaker Required? Case Rep Cardiol. 2015;2015:158948. doi: 10.1155/2015/158948.

26. Seyfrydova M, Rokyta R, Rajdl D, Huml M. Arrhythmias and laboratory abnormalities after an electrical accident: a single-center, retrospective study of 333 cases. Clin Res Cardiol. 2023 Dec;112(12):1835-1847. doi: 10.1007/s00392-023-02274-5.

27. Di Vincenzo M. Pediatric Electrical Burn Injuries. Revista Argentina de Quemaduras. 2023;33(3):1-7. Spanish.

28. Yıldırım BO. A sudden complication: Electrocution-induced atrial fibrillation. Mid Black Sea J Health Sci. 2024;10(1):131-136. doi: 10.19127/mbsjohs.1341920.

29. Fonseca J, Garza J, García M, Aguirre K, Alotaibi H, Mortada H. Electrical burn-induced vocal cord injury: insights from a case report and literature review. Case Reports Plast Surg Hand Surg. 2024 Jul 10;11(1):2374549. doi: 10.1080/23320885.2024.2374549.

30. Bounds EJ, Khan M, Kok SJ. Electrical Burns (Archived). In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2025 Jan.

31. Kolesnyk YuM, Gancheva OV, Abramov AV, et al., authors; Kolesnyk YuM, editor. Pathogenic effect of electric current. Effect on the body of high and low atmospheric pressure. Influence on the body of space flight factors: guidelines for self-training of third-year medical students in the speciality Pediatrics. Zaporizhzhia: ZSMU; 2017. 83 p. Ukrainian.

32. Tapking C, Popp D, Herndon DN, et al. Cardiac Dysfunction in Severely Burned Patients: Current Understanding of Etiology, Pathophysiology, and Treatment. Shock. 2020 Jun;53(6):669-678. doi: 10.1097/SHK.0000000000001465.

33. Noorbakhsh SI, Bonar EM, Polinski R, Amin MS. Educational Case: Burn Injury-Pathophysiology, Classification, and Treatment. Acad Pathol. 2021 Nov 28;8:23742895211057239. doi: 10.1177/23742895211057239.

34. Panchenko SV, Akimov OI, Babajev MM, et al. Electrical Safety: a textbook. Kharkiv: Ukrainian State University of Railway Transport; 2018. 295 p. Ukrainian.

35. Kroll MW, Luceri RM, Efimov IR, Calkins H. The electrophysiology of electrocution. Heart Rhythm O2. 2023 Jun 9;4(7):457-462. doi: 10.1016/j.hroo.2023.06.004.

36. Kroll MW, Luceri RM, Efimov IR, Calkins H. The Mechanism of Death in Electrocution: A Historical Review of the Literature. Am J Forensic Med Pathol. 2025 Mar 1;46(1):3-9. doi: 10.1097/

PAF.0000000000000980.

37. Dechent D, Emonds T, Stunder D, Schmiedchen K, Kraus T, Driessen S. Direct current electrical injuries: A systematic review of case reports and case series. Burns. 2020 Mar;46(2):267-278. doi: 10.1016/j.burns.2018.11.020.

38. Dhaniwala NS, Date S, Dhaniwala MN. Effects of electrical injury on musculoskeletal system: a case report. MOJ Orthop Rheumatol. 2019;11(4):160162. doi: 10.15406/mojor.2019.11.00493.

39. Anggriant V, Roosseno RRN, Pramudyo DP, Chandra Y. Geriatric Patient Outcomes of Major Burn due to High-Voltage Electrical Injury: A Case Report. Intisari Sains Medis. 2022;13(3):551-554. doi: 10.15562/ism.v13i3.1442.

40. Mishalov VD, Khokholeva TV, Bachynskyj VT, Vojchenko VV, Kryvda GF, Kostenko JeJa, authors; Mishalov VD, editor. Forensic medicine: a textbook. Kyiv; 2018, 575 p. Ukrainian.

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Кравець О.В., Клигуненко О.М., Єхалов В.В., Кріштафор Д.А., Пилипенко О.В., Фурсова О.В.
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Летальні та нелетальні серцеві аритмії при електротравмі

Резюме. ***Актуальність.*** Електричні травми є однією з найпоширеніших проблем охорони здоров'я та часто пов'язані із високою захворюваністю і смертністю. ***Збір доказів.*** Літературні джерела включали до дослідження, якщо вони: 1) були опубліковані українською, іспанською або англійською мовами; 2) повідомляли про серцеві аритмії, що пов'язані з електричною травмою; 3) інформували про поширеність серцевих аритмій при електричній травмі; 4) мали обсерваційний дизайн (когортний або перехресний). Проведено ретроспективний пошук інформації з використанням просторово-векторної описової моделі, що була доповнена ручним пошуком задіяних статей. ***Синтез доказів.*** Серце вражається частіше, ніж інші внутрішні органи, оскільки електричний струм зазвичай рухається шляхом найнижчого опору в організмі — поздовж кровоносних судин і нервів до серця. Великі поверхневі опіки й вертикальний шлях проходження струму через тулуб вказують на підвищений ризик пошкодження серця. Найпоширенішим проявом ушкодження серця внаслідок дії електричного струму є аритмії, які зазвичай виникають одразу після електротравми. Порушення провідності можуть залежати від змін міоцитів навколо синусових і атріовентрикулярних вузлів та не обов'язково пов'язані з ішемією міокарда або артеріальним ураженням. Найчастішими порушеннями на ЕКГ при надходженні до стаціонару були синусова брадикардія та синусова тахікардія. Інші задокументовані аритмії включали вперше діагностовану фібриляцію передсердь, часті мультифокальні передсердні комплекси, зупинку синусового вузла з передсердним висхідним ритмом, фібриляцію шлуночків, що була усунена на догоспітальному етапі, шлуночкову бігемінію та рецидивуючу нестійку шлуночкову тахікардію. Спричинені електричною травмою аритмії здебільшого мають функціональний характер, зазвичай є доброякісними та переважно минають протягом перших кількох годин після госпіталізації. Фібриляція шлуночків є найпоширенішою фатальною аритмією, що зустрічається у 60 % пацієнтів, у яких електричний струм проходить трансторакально. Змінний струм частіше спричиняє фібриляцію серця, тоді як постійний здебільшого викликає асистолію. ***Висновки.*** Серцеві аритмії є провідними та небезпечними ускладненнями електричної травми. Частота їх виникнення прямо залежить від характеристик електричного струму, шляху його проходження через організм, тривалості дії, маси тіла та органічних пошкоджень міокарда. У деяких випадках аритмії при електричній травмі минають самостійно, також вони можуть бути відстроченими. Своєчасно проведені реанімаційні заходи допомагають поновити серцевий ритм.

Ключові слова: електричний струм; електрична травма; шляхи проходження струму через організм; аритмії; патофізіологія