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Electrical Injury (a Literature Review)

E.A. Zhirkova*, T.G. Spiridonova, A.V. Sachkov, K.V. Svetlov

Department of Acute Thermal Lesions
N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Health Department
3 Bolshaya Sukharevskaya Square, Moscow 129090, Russian Federation

*** Contacts:** Elena A. Zhirkova, Candidate of Medical Sciences, Leading Researcher of the Department of Acute Thermal Lesions, N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Health Department. E-mail: ZhirkovaEA@sklif.mos.ru

ABSTRACT The analysis of domestic and foreign literature sources showed that the problem of diagnosing and treating electrical injuries remained relevant as in the early 20th century. Over the past century, the mechanisms of the effects of electric current on organs and tissues have been well studied. However, the search for methods for diagnosing the volume of tissue damage has not been completed, and such methods are necessary, since they are designed to help determine the volume of surgical intervention. Many patients still require repeated surgical interventions to completely excise necrotic tissue. In most patients with severe electrical trauma, reconstructive surgery takes place in several stages. Today, most clinical data and practical recommendations are based on the opinions of individual experts and limited clinical studies.

Keywords: electric trauma, electric burn, bio-exposure of electric current, electrolysis, electroporation, electro-conformational denaturation of membrane proteins, diagnosis of electrical injury, treatment of electrical injury

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Affiliations

Elena A. Zhirkova	Candidate of Medical Sciences, Leading Researcher of the Department of Acute Thermal Lesions,, N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Health Department, Cand. Med. Sci., ORCID https://orchid.org/0000-0002-9862-0229
Tamara G.Spiridonova	Doctor of Medical Sciences, Scientific Consultant of the Department of Acute Thermal Lesions, N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Health Department, https://orchid.org/000-0001-7070-8512
Aleksey V. Sachkov	Candidate of Medical Sciences, Head of the Scientific Department of Acute Thermal Lesions, N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Health Department, https://orchid.org/0000-0003-3742-6374
Kirill V. Svetlov	Candidate of Medical Sciences, Researcher of the Department of Acute Thermal Lesions, N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Healthcare Department, https://orchid.org/0000-0002-1538-0515

CNV — central nervous system
CPK — creatine phosphokinase
CPK-MB— creatine phosphokinase MB
DIC — disseminated intravascular coagulation
ECG — electrocardiography

RELEVANCE

In 1600, the work of W. Gilbert "On a magnet, magnetic bodies and a large magnet — the Earth" was published, which described experiments with electrified bodies and introduced the term "electric charge" [1]. The first recorded death caused by electric current from an artificial source was reported in 1879 — a carpenter in Lyon (France) accidentally leaned against a 250-volt alternator [2]. The electrification of everyday life and industry, on the one hand, is an integral factor in progress, and on the other, the cause of severe, including fatal, injuries.

Victims of electricity today account for about 5% of those admitted to burn centers around the world [3]. Patients with electrical trauma differ from patients who received thermal injury from other etiological factors in following: younger age, smaller area and greater depth of skin lesions [4], severe course and high mortality [3], high incidence of disability [5]. In addition, cases of iatrogenic electric trauma have been described [6, 7].

The aim of the work was to study modern data on the etiology, pathogenesis, clinical presentation, diagnosis and treatment of electrical trauma.

Medical literature was searched using the *eLibrary* and *Pubmed* medical databases for the period 1929–2018. Resources with outdated information were excluded, and some were found from article link lists . The current state of the problem of diagnosis and treatment is reflected in articles over the past decade, however, the data of fundamental experimental and pathomorphological studies are in most cases drawn from earlier publications.

ETIOLOGY AND PATHOGENESIS

In the *Pubmed* resource, publications on electrical injury have been available since 1898, and the first experimental information on this type of pathology appeared only in 1929 [8]. At the same time, some authors not so long ago (in 1976) spoke only about the thermal effect of an electric current on tissues [9]. Currently, several mechanisms of the effect of electric current on biological tissues are described: thermal burns, bio-exposure, electrolysis, mechanical damage [10, 11].

The effect of electricity on the body depends on the voltage, current strength, type of current (constant or variable), resistance, current path, duration of contact and the state of the human body. Other things being equal, the electric current is all the more dangerous, the higher its voltage. High-voltage current (> 500–1000 V), as a rule, leads to deep burns, and low-voltage current (110–220 V) usually causes muscle spasm during exposure — tetany. High-voltage currents (thousands of volts and more) in some cases do not lead to death when an arc discharge (volt arc) occurs at the point of contact: carbonization of the tissues occurs, causing a sharp increase in their resistance and a decrease in current strength [12]. An alternating current of voltage up to 500 V is more dangerous than a direct current, at a voltage of about 500 V, alternating and direct currents are equally dangerous, and at voltages above 1000 V, direct current becomes more dangerous [10]. The difference in exposure is due to the fact that irritation of nerve tissues with direct current occurs at the moment of closure and opening of the electric circuit, with alternating current - during the entire passage of the current. Alternating current is widespread in industry and household [13]. Most often, with electrical injuries, they deal with a current frequency of 40-60 Hz. With increasing frequency of the alternating current passing through the human body, the total resistance of the body decreases, and the magnitude of the passing current increases. However, a decrease in resistance is possible only within the frequency range from 0 to 60 Hz, a further increase in frequency is accompanied by a decrease in the risk of damage, which completely disappears at 450-500 kHz. However, these currents retain the danger of burns both in the event of an electric arc, and when they pass directly through the human body. The severity of electric shock depends on the resistance of the skin: burns at contact points are characteristic with high resistance, damage to internal organs is more likely with low resistance . Thus, the absence of skin burns at the points of entry and exit does not exclude the presence of electrical injury , and the severity of external manifestations does not always determine its severity [12]. According to world statistics, the greatest electric shock occurs in installations up to 1000 V, which is associated with their widespread use [14].

V.M. Alekseyev et al. allocated two types of electric shock: electric trauma (local lesion) and electric shock (the effect of current on the nervous system and muscles) [11]. V.A. Sokolov et al. suggested the following types of electric injury: electric trauma — the general effect of electric current on the body, electric burn — local effects on tissues, burns by a flash of a volt arc and damage by atmospheric electricity [13]. *F. Sturmer* believes that electric burns can occur in contact with a current source, in the event of an electric arc and lightning strike [15].

Over 70% of deaths from contact with electricity causes an electric shock [11], which usually occurs at currents of up to 1 A and voltage up to 1000 V [16]. The path of current flow through the human body is determined by the places of its entry and exit and is called a loop. The most dangerous paths go through the heart and respiratory organs [11]. Most often, in practice, the paths are “arm – arm” (up to 40% of all types of lesions) and “arm – leg” (up to 35%). In such situations, 80% of victims die [10], since such an effect almost always affects the heart, leading to its fibrillation [11].

There are two-phase and single-phase touch. With a two-phase touch, a person is simultaneously in contact with two points of the body with bipolar current-carrying parts. At the moment of contact, the voltage of a working network affects a person. In AC networks with voltages above 100 V, the current passage through the body exceeds freezing threshold (16 mA, which causes irresistible convulsive contractions of the muscles of the arm in which the conductor is clamped) and the fibrillation (100 mA, which causes heart fibrillation). Often this leads to death if assistance is not provided to the victim in a timely manner [10]. With a unipolar touch, a person touches the current-carrying part with only one point of the body, and it is affected by almost 2 times less than the operating voltage [16]. With a unipolar connection without grounding, the current does not appear, since it does not pass through the human body [17].

Unlike metals and semiconductors, the body of a person or animal is a conductor of an electric current of the second kind, that is, the conductivity is due to the ions of various electrolytes contained in the body. The skin and, mainly, its outer layer, the epidermis, have the greatest resistance compared to other tissues. The electrochemical effect of the current is manifested in the form of the ionic equilibrium disturbance in tissues in the form of coagulation (at the anode) and colliquation (at the cathode) necrosis; in the formation of vapor and gas, in the impregnation of the skin with a metal conductor [12]. As a result of melting and then solidification of calcium phosphate in bones, “pearl beads” (“bone beads”) can form. They represent the formation of a regular spherical, ovoid shape or polyhedra, hollow inside, with a diameter of 1-2 mm. This phenomenon was described by K. Roentgen in 1911. Electric current influences the potassium-sodium gradient of cells, disrupts membrane potentials and impulse transmission along nerves [12, 17].

Bioprotection includes electroporation and electroconformational denaturation of membrane proteins. Electroporation (the appearance of a local restructuring in the bilayer lipid membrane leading to the appearance of a through water channel) can cause cell necrosis in the absence of high temperature [5, 18, 19]. *D. Bhatt et al*. in experimental work showed that rhabdomyolysis and secondary release of myoglobin can be the result of electroporation [20]. Transmembrane protein molecules contain polar amino acid residues that can change their orientation in response to the passage of electric current. This effect, known as electroconformational denaturation of membrane proteins, is usually irreversible and is a non-thermal damage mechanism [5, 21].

The most often electrical injuries are thermal burns, which are also divided into arc burns and contact burns. An arc burn manifests itself when a person enters the sphere of influence of an electric arc (this usually occurs between live parts of equipment) [11]. The temperature in the arc channel reaches 7000° C; as a result, skin integuments, muscle and bone tissue can burn out [10]. Contact burn is the result of contact of a part of the human body with a heated element of electrical equipment [11].

Having overcome the resistance of the skin and subcutaneous fatty tissue, the electric current passes along the path of least resistance through deep-lying tissues, through tissue fluid, blood and lymph vessels, and the shaths of nerve trunks [7, 15].

The current passing through the vessels damages their intima, which causes thrombosis and bleeding, impaired vascular-platelet hemostasis, blood coagulation and fibrinolysis, leading to disseminated intravascular coagulation (DIC), microcirculatory disorders, endothelial dysfunction [22].

There are two types of brain damage: immediate thermal effects and late degenerative consequences that appear close to the vascular canals or pools of cerebrospinal fluid [15]. The ideas about the primary and secondary damaging effects of electric current on the nervous system are of particular importance for the practice of managing patients with electrical trauma. The primary damaging effect leads to necrosis and subsequent gliosis of neurons (replacing lost neurons with neuroglia cells) of the central nervous system (CNS) and Waller's degeneration of peripheral nerves. The secondary damaging effect of electric current on the nervous system is due to the action of vascular, toxic (burn disease) and mechanical factors [23]. In the brain and spinal cord, hemorrhages, areas of rarefaction of the brain tissue, thickening of the glial network, proliferation of glial elements, sclerosis and hyperchromic nerve cells of the cortex are detected. In the vascular bed, plethora is observed with the expansion of large vessels and capillaries, stasis, hemorrhages in perivascular spaces and in the substance of the brain. Perivascular hemorrhages most often

occur in the diencephalon, medulla oblongata, in the walls of the third and fourth ventricles, in the region of the anterior horns of the spinal cord [12].

The mechanical effect of the current is manifested in the violation of the integrity of the skin and other tissues (with the formation of abrasions, wounds, isolated bones cracks), as well as in the introduction of metal particles of the conductor into the skin at the point of contact (metallization) [17]. The mechanical effect of a large current is manifested in the stratification of tissues up to the detachment of body parts [13].

Lightning is a special type of electrical injury caused by atmospheric electricity. It is a huge discharge of atmospheric electricity in voltage (millions of volts) and current strength (more than ten thousand amperes). Under its action, processes occur that are similar to the action of high-voltage technical electricity, but having a large quantitative severity. The thermal and mechanical energy of lightning when exposed to a person can lead to widespread burns of I, II and III degrees, to singing part or all of the hair, rupture of internal organs and detachment of body parts. Sometimes, peculiar "figures of lightning" are formed on the skin in the form of a red imprint of a tree shape [17]. A person is struck by a lightning during a thunderstorm less often than by technical electricity, and possibly mainly in the open air, near tall metal structures, trees or indoors through wired devices, as well as through open windows and chimneys. There are known cases of lightning in a tram and trolleybus. When lightning strikes due to the spreading of current on the ground, damage to nearby people is not uncommon.

The causes of death in electrical injuries are different and are due to the nature of the current, through its passage, the reaction and condition of the body, as well as other factors. One of three causes of death, or a combination of both, is possible: impaired heart function (fibrillation), respiratory arrest, and shock [24]. They can occur both with the direct action of an electric current on the heart or brain, and reflexively when exposed to other parts of the body. Acute tissue ischemia as a result of a spasm of the smooth muscles of the vessels is of great importance in the mechanism of development of these conditions. In most cases, the cause of instant death is the disturnabce of cardiac activity under the action of a low-voltage current (110–380 V) and a small force. At a higher voltage and magnitude of alternating current, CNS damage and respiratory arrest occur more often [17].

THE CLINICAL PICTURE AND DIAGNOSIS

The clinical picture is very diverse and largely depends on the severity and characteristics of the electric trauma itself. The current passing through various organs and tissues causes a number of disorders. To determine the severity of electric shock S.A. Polishchuk and S.Y. Fistal suggested a classification that takes into account convulsive muscle contraction, loss of consciousness, impaired cardiac and respiratory activity [24].

Cardiac arrest usually occurs at the time of injury. Many complications are similar to those with thermal burns [25]. Neurological complications, such as loss of consciousness, the effects of peripheral nerve damage, and delayed spinal cord injury syndromes may occur [26–28]. The most common gastrointestinal complications are stress ulcers and intestinal obstruction. Cases of damage to the pancreas and liver, hollow internal organs, including the small intestine, colon, bladder, and gall bladder are known [29, 30].

An alternating current of low voltage (220–380 V), passing through a body with low skin resistance (moist, thin skin, dense, large contact area), may leave no traces [7]. The same current acting on the skin with high resistance (dry, thick, calloused), as a rule, forms a "current mark" or "electric mark" at the contact area. Electrical devices with "double-pole contact" are formed at the place of both contacts, with "single-pole" at the entrance, and at the current output point of the indicated voltages, electric circuits are rarely formed or are weakly expressed [12]. Electric marks can have a different look. The most typical are a dense, gray or gray-yellow rounded skin area of several millimeters in size, with raised edges and a slight depression in the center. Unlike thermal burns, the edges of the electric marks are clear, the surrounding skin is not altered, the hair is not singed. If a voltage above 380 V affects the skin, an electric burn of the III degree occurs, which captures the entire thickness of the skin and may be accompanied by charring. The burn site has a dark yellow, brown or black color, clear boundaries; its area depends on the magnitude of the current and the contact area. Extensive burns with charring and lesions of deeply lying soft tissues and bones occur at 1000 V and higher current. They are often combined with burns from the action of an electric arc and flammable clothing, that is, with thermal burns [17]. Sometimes burns are formed along the current, in the elbow, inguinal and other bends, where two layers of skin come into contact through which current flows (the so-called *kissing burn*) [17, 25].

Electrothermal heating is the main cause of muscle damage, and it is observed almost exclusively when exposed to high voltage with a long (second) contact and current flow [11].

Vascular damage can lead to arrosion bleeding [31], thrombosis or vascular occlusion at different times after the injury, since edema and clots form on the damaged inner surface of the vessel within a few days [32]. Damage to the small muscle arteries leads to progressive muscle necrosis, with external initial signs of their viability [25].

With high-voltage injuries, loss of consciousness may occur, but, as a rule, it is temporary, except when there is a significant concomitant head injury [25]. CNS electric trauma can cause seizures, either as a single event, or as part of a first-time and persisting convulsive disorder [26]. When exposed to high voltage, damage to the spinal cord can be the result of fractures or ruptures of the ligamentous apparatus of various parts of the spine. Neurological symptoms can occur from a few days to several years after an injury in the form of ascending paralysis, amyotrophic lateral sclerosis, or transverse myelitis. Movement disorders prevail over sensitivity disorders, and the prognosis of function recovery is usually unfavorable [27].

It is customary to conduct electrocardiography (ECG) and determine the level of creatine phosphokinase (CPK) and its cardiac fraction (CPK-MB) for all victims with cardiorespiratory disorders, regardless of the magnitude of the electrical voltage. Further cardiological monitoring after a high-voltage injury is not carried out if the victim did not lose consciousness at the time of the injury, arrhythmia did not occur, and abnormalities were not detected during ECG [33]. 24-hour cardiomonitoring is necessary in case of confirmed arrhythmia or abnormal ECG during the initial examination; with loss of consciousness [34, 35]. To exclude myocardial infarction under conditions of electric shock, CPK levels should be interpreted with caution. A high level of CPK does not always indicate myocardial damage if a large volume of skeletal muscle is damaged; myocytes can contain up to 20–25% of the KFK-MB fraction and be its possible source [36]. CPK- MB accounts for 37% of the total amount of CPK in the heart muscle and can also increase with skeletal muscle injury, but the level of this fraction should be less than 6% of the total amount of CPK [37]. There is no information regarding the assessment of changes in the level of troponin after electric shock [7].

The relationship between the level of CPK and the prevalence of skeletal muscle ischemia is unproven. Some authors believe that the level of CPK depends on the volume of ischemic tissues [25], while others do not show such a relationship [38, 39]. It is believed that patients with extremely high levels of CPK and lactate dehydrogenase during the first two days after admission to the hospital will most likely require limb amputation, and the highest mortality rate in this group [40, 41].

Computed tomography is performed for those affected by electric current, who had a disturbance of consciousness [7, 25], myoglobinuria is excluded - a frequent complication of high voltage electrical damage. If urine is pigmented or urine tests are positive for occult blood, and erythrocytes are not detected by microscopic analysis, it is suggested that the patient has myoglobinuria [7, 25]. Radiographs of the cervical spine are performed if a spinal injury is suspected, as well as an X-ray examination of any areas in which the patient complains of pain, or their deformation is noted [25].

Pulse oximetry can be used to diagnose limb ischemia: with a decrease in the saturation rate below 90% and its difference between healthy and damaged areas over 6%, tissue decompression — necrotomy, fasciotomy, may be necessary [41].

Scintigraphy is used to clarify the volume and location of muscle damage [42, 43].

The use of magnetic resonance imaging for the diagnosis of muscle necrosis has been described [41].

Damage to internal organs is rare, but may require interventional treatment and are associated with an increase in mortality [44].

The histological changes observed in damaged muscles as a result of direct contact with an electrical source are coagulation necrosis with a shortened sarcomere. Muscle damage can be "mosaic", so the areas of viable and non-viable muscles are often in the same muscle group. Observations of damage to deep layers of muscles are known - with intact surface layers [45].

R. DeBono, conducted a thorough histological examination of the amputated upper limb of a victim of high voltage (100,000 V), and drew attention to the fact that the tissues on the lateral side of the forearm were significantly more damaged than on the medial side. In addition, he showed that the distal parts of the forearm were more damaged than the proximal [5]. These observations correspond to the ideas about the current paths depending on tissue resistance.

TREATMENT

Today, domestic clinical guidelines for the provision of prehospital and inpatient care for electrical trauma have not been developed.

Pre-hospital care consists in interrupting the victim's contact with the current source, the immediate start of cardiopulmonary resuscitation, immobilization (including the cervical spine) if necessary and the start of infusion therapy, while observing the safety precautions at risk of electrical injury [46].

Intensive therapy. Treatment of severe electrical trauma in a hospital requires simultaneous cardiopulmonary resuscitation and emergency care as with multiple injuries. The volume of injected fluid depends on the severity of the injury and damage to specific organs [7]. Electric skin burns do not give a clear idea of how much tissue is actually damaged. Isotonic solutions for intravenous infusion are prescribed, sufficient to maintain urine production at a level of 1.0-1.5 ml/kg/h. It is necessary to carry out infusion therapy until adequate diuresis is achieved, control the level of CPK and myoglobinuria [47], use antiplatelet agents, including thromboxane synthesis inhibitors and thromboxane receptor inhibitors, broad-spectrum antibiotics; transfuse blood components, since lowering hemoglobin levels below 70 g/l even for 2-3 hours is enough to spread and aggravate ischemic zones. In some cases, the effectiveness of hyperbaric oxygenation in victims of electrical trauma was noted [25].

Surgery. Patients who have received high-voltage electric burns to their extremities need surgical treatment that begins as early as possible. Early necrotomy and fasciotomy, repeated surgical treatment of wounds lead to tissue decompression and reduce the frequency of amputations [43].

T. d'Amato et al. conducted an intraoperative study of the viability of the muscles of the flexors and extensors of the hand and forearm, and also excluded carpal canal compression syndrome immediately after the victim arrived. Then the revision of the operated areas was repeated again 24–48 hours later. Patients underwent extensive fasciotomy, including cases of deep muscles, which subsequently eliminated the need for amputation of affected limbs [38]. In another study, decompression necrotomy or fasciotomy was performed immediately in cases of progressive edema and signs of limb ischemia. Early fasciotomy was effective in patients with high-voltage burns and skin burns of less than 40% of the body surface. This underlines the importance of surgical procedures performed within 4–6 hours after injury to prevent secondary ischemic muscle necrosis [40].

J. Gille et al. performed necrectomy and autodermoplasty with a split skin flap within 72 hours after the injury, depending on the patient's condition. Amputation of the extremities and microsurgical autotransplantation of flaps on the vascular pedicle was performed with extensive muscle necrosis in accordance with the protocols adopted in individual foreign burn centers [48].

Free autodermoplasty and plastic surgery with local tissues were used to close small defects that occur, as a rule, when exposed to low-voltage current. When struck by a high voltage current, replacement was mainly performed with local tissues and grafts on the vascular pedicle, but free autodermoplasty was also used for extensive and complex defects with exposure of deep structures [39, 49]. Microsurgical methods for eliminating tissue defects during electric trauma are described in the literature as a limited number of observations [50–54].

CONCLUSION

Modern ideas about the etiology of electrical trauma formed at the beginning of the 20th century, when electric shock began to be divided into electrical trauma by technical electricity and lightning. As we studied the effect of electricity on living tissues and the body as a whole, in an experiment, clinic, and forensic research, we began to distinguish between electrical injury from low and high voltage currents. By the end of the 20th century, electroporation and conformational denaturation of cell membrane proteins were discovered. This supplemented the previously known pathogenetic mechanisms of electrical injury - thermal burns, bio-exposure, electrolysis.

Diagnosis of the damage volume is often based on clinical manifestations (the presence of characteristic current marks, soft tissue edema, impaired sensory and motor function) and visual examination of the affected tissues (during the surgery as well). However, electrical injury is often a process that develops over time. Diagnosis of the amount of damage sometimes takes 2-3 weeks, during which the victim needs several operations with tissue revision. The search for a prognostic factor that allows, within the first hours or days, to determine the volume of affected tissues on the basis of laboratory (the relationship between the levels of creatine phosphokinase, lactate dehydrogenase, troponin) and instrumental studies (pulse oximetry, scintigraphy, magnetic resonance imaging) did not lead to the desired result. In this regard, the described prognostic parameters should be interpreted with caution and taking into account the individual course and the specific case.

The publications on electrical trauma contain relatively little information about surgical treatment: when should it begin, what necessary interventions should be performed, and what will happen if they are not performed. The results of the studies cited often contradictory, which may be associated with the desire to compare the different nature of the injury (e.g., a combination of electrical and mechanical injury), different levels and timing amputations different uses of microvascular operations.

Given that the percentage of such patients in specialized burn centers is very small, multicenter prospective randomized trials are needed to evaluate the effectiveness of existing methods of diagnosis and treatment, as most researchers have recently claimed.

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