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Central neurological injuries in electrical trauma (review)

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Abstract. ***Background.*** *Damage to the central (CNS), peripheral and autonomic nervous systems are the leading complications of electrical trauma (ET).* ***Evidence collection.*** *A retrospective informative search was performed using a spatial-vector descriptive model, which was supplemented by a manual search of relevant articles. Forty-two modern literature sources were selected over the past 10 years, of which 76.2 % — over the past 5 years.* ***Evidence synthesis.*** *The leading mechanisms of neurological ET are electroporation, vascular injury, demyelination and the release of thermal energy, which causes the destruction of macromolecules. In this regard, theories of causality, alternative and neurohumoral hypotheses have been proposed. There are several types of nerve damage in ET, including circulatory disorders, ischemic encephalopathy, hypoxic encephalopathy, various dystrophic changes in brain cellular structures and intracranial hemorrhages. The greatest neuronal damage is observed in cases with minimal skin damage (the most significant amount of current falls on internal structures). Direct or indirect effects of ET on the brain are possible, when craniocerebral trauma is caused by the passage of current or direct damage to the brain due to blunt mechanical trauma (or a combination of both). Electric shock can lead to ischemic stroke due to circulatory disturbances or to hemorrhagic stroke due to direct structural damage with necrosis and axonal degeneration. Acute CNS complications include changes in mental status, coma, amnesia, quadriplegia and localized paresis. Motor impairment occurs more often than sensory deficits. Delayed neurological syndromes, such as cerebellar ataxia or autonomic dysfunction, may occur weeks or months after the initial injury due to the development of subsequent ischemia, a decrease in the number of Purkinje cells or the presence of hemorrhages. In situations where the path of the electric current crosses the spinal cord, which usually occurs at the level of C4-C8 (i.e. from one limb to the other), the patient is at risk of spinal cord injury. This type of trauma leads to direct damage to the spinal cord, which causes significant neurological disorders.* ***Conclusions.*** *The leading mechanisms of neurological electrical trauma are electroporation, vascular injury, demyelination and the release of thermal energy. Brain damage in electrical trauma is possible even when the head is not in a direct electrical circuit. The mechanisms of the development of delayed neurological complications are currently not fully understood.*

Keywords: *electrical trauma; brain; cerebellum; spinal cord; pathophysiology; review*

Introduction

Damage to the central, peripheral and autonomic nervous systems are the leading complications of electrical trauma (ET) [1–8]. They occur with early or late onset and, depending on the severity of the injury, have different durations. Therefore, most neurological complications of ET are diagnosed only after a certain time after the injury [9–11].

In most cases, symptoms appear immediately after ET. However, some neurological complications occur within the first six months after electric shock, or even at a later time (1–5 years) [12–18].

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Due to the peculiarities of their structure, nerves and blood vessels contribute to the passage of current, which determines the neurological and vascular consequences of ET and explains the high frequency (31.0–81.6 %) of electrical injuries of the nervous system [9, 19–22].

Collection of evidence

Selected literature sources were included in the study if they: 1) were published in Ukrainian, English or Spanish; 2) reported acute functional and morphological damage to the central nervous system in electrical trauma; 3) reported on the prevalence of neurological pathology in electrical trauma; 4) used an observational design (cohort or cross-sectional). A retrospective informative search was performed using a spatial-vector descriptive model, which was supplemented by a manual search of relevant articles. 42 modern literature sources were selected over the past 10 years, of which 76.2 % — over the past 5 years.

Evidence synthesis

Mechanisms of neurological electrical injury development

— Electroporation is due to the opening of pores in the cell membrane, which initiates a cascade of events that contribute to cell death (apoptosis).

— Vascular injury occurs as a result of spasm and release of vascular inflammatory mediators, which cause neuronal ischemia, chromatolysis and microglia activation.

— Demyelination occurs due to changes in the structure of proteins and lipids.

— The release of thermal energy causes the destruction of macromolecules.

Electricity promotes the circulation of substances, in particular cortisol, a stress hormone, and the generation of free radicals, which act at a distance and by hyperstimulation of glutamate receptors. The interaction of all these factors may also be responsible for delayed effects in the central and peripheral nervous systems [11, 20, 23, 24].

Causal theories

Central syndromes, including cerebellar syndromes, are often attributed to direct exposure to electrical current. Vascular causes of cerebral infarction with vascular obliteration have been postulated. Focal petechial hemorrhages, especially in the cerebrum and medulla, and in the gray matter of the anterior horns of the spinal cord, are more common after alternating current (AC) injury [23, 24].

Chromatolysis is a state of dissolution or disintegration of the chromatophilic substance (Nissl bodies or rough endoplasmic reticulum) in the cytoplasm of neurons. This phenomenon can cause cell exhaustion and is indicative of cell damage. Chromatolysis is most commonly seen in the pyramidal cells of the medulla oblongata, in the anterior horn cells, and in the Purkinje cells of the cerebellum. These changes are patchy and well-defined. There is expansion of the perivascular spaces, which is due to the formation of gas/vapor and is observed mainly in lethal electric shocks. In severe damage, the entire brain and individual areas of the spinal cord may be swollen, softened, and even leak [24].

Classes of causal processes

1. *General symptomatic endpoint*. It is noted that prolonged incapacity after low-voltage ET resembles the effects of traumatic brain injury.

2. *Direct electrical damage causes damage* along the current path, which includes electrostatic tissue delamination, as well as cytolysis due to electroporation. The least electrical resistance is provided by blood flowing through the subclavian arteries. A potential difference exists between the blood in the carotid arteries, and a (possibly small) part of the current flows from one carotid through the circle of Willis to the opposite carotid artery. Thus, there is an amount of current that enters the brain. Therefore, it would be wrong to assume that a direct current flow from hand to hand cannot affect the brain because it is not in the direct path of the electric current.

3. *Thermal damage* occurs as a result of the contact of the current with the resistance of the tissues and the generation of dissipative (associated with energy losses) heat. Therefore, the statement that the most electrically resistant tissues dissipate more heat is incorrect.

4. *Tissue delamination* under the influence of electricity is due to the formation of gas phases, which, due to an increase in their volume, cause tissue stratification.

5. *Ischemic changes* are due to the passage of current through blood vessels, which leads to vascular spasm, ischemia and inflammatory reaction of the perfused tissues.

6. *The neurohumoral effect of the blood flow* includes the release of neurohormones, which explains both the "action at a distance" and the symptoms of "delayed onset" [20, 23–25].

Delayed development syndromes (alternative hypothesis)

The most dramatic and difficult to explain phenomena are the neurological disorders that occur months or even years after electric shock. These include motor neuron disorders, amyotrophic lateral sclerosis, parkinsonism, and focal dystonia. In some cases, late spinal cord atrophy is observed. Electricity alters protein and/or DNA and this can negatively affect the vascular endothelium, leading to a restriction of blood supply to the central nervous system [23, 24, 26].

Neurohumoral hypothesis

Under the influence of current, circulating substances are released and transported by the bloodstream, acting at a distance. Changes in arterial function have been noted in arteries directly along the line of current flow. However, functional disorders in arteries have also been identified when current does not flow to the vessels on the opposite side of the body [20, 23, 24].

The proposed pathophysiological mechanisms of CNS lesions caused by low-voltage or high-voltage current are the following: direct, thermal or mechanical damage to neurons with their secondary loss, microglial activation with blood-brain barrier dysfunction and neuroinflammation, neuroexcitotoxicity due to glutamatergic hyperstimulation, electroconformational denaturation of proteins and DNA, oxidative stress and ischemic changes in neurons due

to vascular spasm, and endothelial inflammation. This may be due to direct thermal damage to the nervous tissue or to electroporation, which damages nerve cells that depend on existing electrolyte gradients for proper function [15, 21, 28]. Electroporation is considered an important factor in neuronal changes in the plasma membrane after electrical injury, causing immune-mediated demyelination after the formation of electropores in the phospholipid bilayer of cell membranes and structural damage to the myelin antigen with an increase in their permeability [15, 28]. These pores allow ions and certain intracellular molecules to pass through the membrane by simple diffusion and have a limited lifespan. In addition to these cellular effects, electricity disrupts vascular regulation, causing thrombosis and weakening of the wall of small arteries, which can rupture, causing bleeding. This risk exists during the first three days after the initial injury, in contrast to the later formation of vascular aneurysms. Small arteries spasm due to dysregulation of the autonomic nervous system, which also contributes to the development of ischemia and thrombosis [28].

Brain damage from electrical trauma

Any current passing through the head can cause damage to the central nervous system [29]. The skull has a high electrical resistance, which creates a certain protection of the brain from the effects of electricity. CNS injuries are more likely to occur when exposed to high-voltage electricity, given that low electric field strength is rarely sufficient to cause significant neurological injuries [10, 15, 26]. Therefore, until recently, it was believed that CNS damage is possible only when exposed to high-voltage electric current. However, the spread of electric current throughout the body can occur through nerves and blood vessels (which have low electrical resistance), causing electrical brain injury when the head is not necessarily connected to the electrical circuit [23, 24]. However, dysfunction of the central nervous system is still considered a characteristic feature of high-voltage electrical injuries.

The greatest neuronal damage is observed in cases with minimal damage to the skin (the most significant amount of current falls on internal structures) or tissues outside the spinal cord. If the electrical path does not cross the head, ET leads to stunning of the person or short-term amnesia, respiratory arrest or seizures [10, 30].

There are several types of neural damage in ET, including circulatory disorders, ischemic encephalopathy, hypoxic encephalopathy, various dystrophic changes in the cellular structures of the brain and intracranial hemorrhages [19].

Since the genesis of many cerebral pathologies is believed to be vascular in nature, ischemic encephalopathy is expected in ET, and encephalopathic syndromes include confusion and amnesia, seizures, hallucinations, dysphasia and the occurrence of diffuse changes in the white matter of the brain. Long-term complications include glioma, cerebral atrophy, and hydrocephalus requiring surgical shunting [20, 23, 24]. Proposed mechanisms for late sequelae include vasospasm, neuronal ischemia, and chronic inflammation of the vascular endothelium. Dystrophic and necrobiotic processes develop in the cells of the cerebral cortex, subcortical ganglia, and brainstem [15].

It is believed that in ET, hyperstimulation of glutamate receptors in the hippocampus may interfere with long-term potentiation. If such stimulation is of normal intensity, neuronal cell death does not occur in ET; however, if the hyperstimulation is of significant intensity, hippocampal cell death may occur. The relationship between glutamatergic hyperstimulation and cortisol levels is of great importance in the development of insulin-resistant cognitive dysfunction [23, 24]. In the context of ET, glucocorticoids may keep glutamate receptors open for a longer time and more often enhance glutamatergic hyperstimulation. A synergistic effect between cortisol and glutamate has also been suggested. Provided that electricity reaches the brain, massive hyperstimulation occurs, affecting the excitatory neurotransmitter glutamate. In this case, hippocampal dysfunction can be expected [24].

The limited diffusion of white matter in the brain after electric shock can be explained by the physical properties of axons, which create a retrograde flow of energy through extensive neuronal connections, causing symmetrical damage despite the current path outside the brain projection [9]. Basal ganglia ischemia, cerebral hypoxia and anoxia are considered key mechanisms [24].

Direct or indirect effects of ET on the brain are possible, when the traumatic brain injury is caused by the passage of current or direct brain damage due to blunt mechanical injury (or a combination of both). About 18.2 % of patients with electrical burns had signs of brain damage, which included concussion or even brain contusion, which was confirmed by the results of computed tomography. The injury can be closed or penetrating and usually requires immediate surgical intervention. Up to 35 % of high-voltage electric shock victims sustained head injuries, and 9.9 % of patients required neurosurgical treatment, including cranioplasty [8, 13, 20, 31].

There are two types of brain damage: primary thermal effects and late (secondary) degenerative effects that occur in the immediate anatomical proximity of vascular channels or cerebrospinal fluid pools [6, 26]. Primary damage leads to necrosis, subsequent gliosis of neurons (replacement of lost neurons with neuroglial cells). Secondary effects are caused by vascular, toxic (burn disease) and mechanical factors [26]. Intracranial hemorrhages of almost all variations are observed. Many of them are associated with a skull fracture, sometimes a certain chronicity of the process is possible, as well as their conditionality by other concomitant neurological pathology [8, 20, 24]. Electric shock due to contact with a high-voltage electrical cable is considered to be the cause of cerebrovascular hemorrhage, with elements such as exposure to high-voltage current, initial short-term loss of consciousness, and the presence of scalp burns, indicating electrical damage to the area of cerebral hemorrhage [28].

If the current passes through the skull, the generated thermal energy causes blood coagulation in the dural sinuses and coagulation necrosis of the brain parenchyma [26]. High temperature causes coagulation necrosis of the endothelium and muscular coat of brain vessels, arteries dilate, resulting in the formation of aneurysms prone to rupture [20].

In the vascular bed, there is hyperemia with dilation of large vessels and capillaries, stasis phenomena, hemorrhages into the perivascular space, into the substance and ventricles of the brain, and the formation of epidural and subdural hematomas [26].

Subarachnoid and multiple perivascular and diffuse small-focal hemorrhages are mainly located in the subcortical structures, in the walls around the III and IV ventricles of the brain, in the diencephalon and medulla oblongata. Traumatic and non-traumatic bleeding may occur due to vasospasm with venous hyperemia [6]. Thrombosis of the macro- and microcirculatory bed occurs. Vascular thrombosis may be associated with convulsive syndrome [20].

Electric shock can lead to ischemic stroke due to circulatory disturbance or to hemorrhagic stroke due to direct structural damage with necrosis and axonal degeneration [6].

Cerebral infarction in ET is mostly considered vascular in origin [24]. With continuous current exposure for a certain period of time, dyspnea and spasm of vascular smooth muscles cause oxygen transport disorders and can lead to ischemic brain damage [20]. Brain hypoxia (hypoxic-ischemic encephalopathy) after circulatory arrest, severe arrhythmia or mechanical head trauma to some extent increases the risk of acute cerebral circulatory disturbance [9]. High voltage injuries can cause acute stroke as a result of thrombosis of cerebral arteries and veins (to a greater extent), caused by thermal coagulation and activation of the blood clotting process [6].

Edema of the pia mater and brain tissue is secondary to electric shock, affects the brain substance itself, and can also be realized as an element of other complications [6, 8, 20]. In this case, acute intracranial hypertension often occurs (up to 400 mm Hg) [20]. Clinically, this is manifested by headache, meningeal symptoms with rigidity of the occipital muscles, photophobia, positive Kernig's sign, epileptic-like seizures, etc. [32]. Loss of consciousness and amnesia after household electric shock are rare (0.6 and 2.2 %, respectively). An increased risk of unconsciousness and amnesia has been identified in those victims who were exposed to electric shock on both sides of the body compared with those who received a unilateral electric shock, with odds ratios of 2.60 and 2.18, respectively [1, 3, 16, 21, 33–35]. Most victims regain consciousness within 5 to 10 minutes of being removed from the electrical circuit without any special measures. However, unconsciousness can last for several hours or even days, which is usually observed when high-voltage current passes directly through the brain [26].

Acute CNS complications include altered mental status, coma, amnesia, quadriplegia, and localized paresis. Motor deficits are more common than sensory deficits [24]. Less common complications of intracerebral trauma include hemiplegia, aphasia, striatal symptoms (Tourette syndrome), seizures, epilepsy, headache, and impaired memory and attention [20].

Seizures can occur immediately after electrical shock due to cortical stimulation or direct brain damage. This recognized immediate complication is often attributed to electrical disruption of neuronal activity [8, 36]. Patients who experience immediate seizures following electrical trauma are at risk for developing posttraumatic epilepsy (PTE) [15, 24, 36]. Mortality in ET-induced traumatic epilepsy is more than 13 times higher than that in burn injuries, and in refractory status epilepticus, the risk of mortality increases over time [11, 37].

Clinical and experimental histological studies have demonstrated pyramidal cell loss, leptomeningeal lesions, demyelination, and axonal fragmentation, degeneration, and necrosis [6]. Areas of brain tissue thinning, glial network thickening, glial cell proliferation, sclerosis, and hyperchromia of cortical nerve cells are found in the brain and spinal cord [6, 26].

In some cases, it is impossible to clearly identify the electric current stigmata during clinical examination, which makes radiological evaluation a crucial step in the examination of these patients [9].

In encephalopathic syndromes, MRI reveals lesions suggestive of cytotoxic edema. Hyperintensity has been observed predominantly in the white matter, forming finger-like projections [30]. Encephalopathic manifestations are more commonly seen after hypoxic injury due to cardiovascular failure or circulatory arrest [22].

In the central nervous system, changes in brain parenchymal signal intensity may be reversible or permanent, depending on the time of exposure and the voltage of the electrical current. Imaging models can be useful in determining the possible mechanism of neuronal damage, taking into account the distribution pattern, data on diffusion and T2-weighted sequences, as well as subsequent imaging and clinical development [9].

In severe cases, the central nervous system may be affected to the extent that amnesia, seizures, or apnea occur [10].

It is for the treatment of the above-mentioned consequences and complications that the neurosurgeon, together with a multidisciplinary team, plays a key role in the immediate and future care of victims of electrical burns [20].

Cerebellar disorders after electrical trauma

Injuries to the cerebellum, including cerebellar ataxia, can occur acutely or delayed as a result of electrical head injury and general brain damage [25]. Cerebellar ataxia can manifest as an unsteady gait ("drunken gait"), difficulties with coordination, disequilibrium, tremors, and dysarthria [15, 38]. There are reports of cases where previously rapid and confused speech turned into "robotic" speech, probably of cerebellar origin [24].

Delayed neurological syndromes, such as cerebellar ataxia or autonomic dysfunction, may occur weeks or months after the initial injury due to subsequent ischemia, decreased Purkinje cell count, or hemorrhages, as evidenced by computed tomography [9, 15, 19, 36, 39].

Spinal cord injury due to electrical trauma

Spinal cord injuries are most commonly associated with direct electrical current, circulatory and respiratory compromise, and severe psychotraumatic stress. In situations where the electrical current path crosses the spinal cord, which usually occurs at the C4-C8 level (i.e., from one limb to the other), the patient is at risk of spinal cord injury. This type of injury results in direct

damage to the spinal cord, resulting in significant neurological deficits [21, 27, 40]. High-voltage ET has a higher incidence of spinal cord injury. Most often they are transverse injuries with posterior spinal cord injury syndrome [21]. In the case of a not very high voltage injury, typical among organic spinal cord injuries are spinal atrophic diseases, which are caused by electric current damage to the spinal cord in the areas of the anterior horns and gray matter in the central canal circle, which is accompanied by vasomotor and trophic disorders in the innervated areas. Spinal atrophic processes are associated with subarachnoid hemorrhages in the spinal cord, its focal lesions with loss of spinal neurons and disorders of spinal conduction. Damage to anterior horn cells is recognized as a long-term complication of electrical injury [6, 36].

If the electrical current reaches the spinal cord and the vessels that supply it, it can lead to hyperstimulation of glutamatergic neurons, leading to the formation of free radicals that subsequently gradually destroy endothelial cells in the supplying blood vessels, ultimately leading to thrombosis and death of spinal cord neurons [24]. Secondary neurological damage can also develop due to hypoxia and subsequent ischemia after circulatory arrest or due to vasospasm [21].

Electrical burns have several acute and chronic manifestations, such as spinal cord deficits, which usually do not appear immediately, but later — after weeks or even months. In a certain number of cases, they are reversible and temporary [25, 35, 40]. Motor disorders occur more often than sensory loss. Cases of acute and delayed electrospinal injuries distal to the site of electrical contact at voltages from 75 to 75,000 V have been described. However, no neurological deficits have been identified immediately after ET. Signs and symptoms sometimes debut a few days or up to 2 years after the incident, when hemiplegia or complete quadriplegia occurs. The passage of electric current causes a "spinal cord transection" and a Brown-Séquard-like condition with a combination of upper and lower motor neuron symptoms [35].

There is a fairly high probability of developing amyotrophic lateral sclerosis (ALS, Lou Gehrig's disease), which debuts a considerable time after ET (sometimes years) [20, 23, 24]. ALS and transverse myelopathy are clearly described together with the associated demyelination syndromes. Developmental delay is especially emphasized. Other associated syndromes at the spinal level may also occur: corticospinal paresis, anterior horn myelopathy, dystonia, muscle atrophy, sensory disorders with vasomotor and trophic disorders, and pelvic disorders are sometimes observed [24, 26]. Relatively stable paralysis with sensory, motor and trophic disorders occurs quite often. Thermoregulation disorders with asymmetric temperature in different areas, disappearance of physiological and emergence of pathological reflexes, etc. are possible.

Motor neuron disease (MND) has been widely described, mostly as a delayed-onset syndrome. It has a strong resemblance to ALS (some authors have identified them). Delayed onset and slow progression seem to characterize these disorders. The disease is characterized by loss of muscle control, manifested by weakness, muscle atrophy, and difficulty speaking, swallowing, and breathing. The similarity between general MND and the more specific ALS is evident in the involvement of the terminal motor neurons in both cases [23, 24]. The features are diverse and may include ascending paralysis, amyotrophic lateral sclerosis, or transverse myelitis [9]. Lower limb weakness may initially go undiagnosed, especially if it is not suspected and the patient attempts to walk [3, 6, 7]. MRI findings are used to confirm diagnoses [24]. However, with few exceptions, imaging studies in the early post-ET period usually do not reveal any evidence of structural brain damage [23].

These delayed neurological syndromes can complicate the recovery process and create additional challenges for treatment. Complete neurological recovery is rare in patients with electrical spinal cord injuries [36].

Given the magnitude of the neurological sequelae following electrical accidents, the mechanisms underlying these symptoms require further investigation [41].

Conclusions

1. The leading mechanisms of development of neurological electrotrauma are electroporation, vascular trauma, demyelination and release of thermal energy.

2. Brain damage in electrical trauma is possible even when the head is not in a direct electrical circuit.

3. The mechanisms of development of delayed neurological complications are currently not fully understood.

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Центральні неврологічні ушкодження при електротравмі (огляд)

Резюме. ***Актуальність.*** Ушкодження центральної, периферичної та вегетативної нервової системи є провідними ускладненнями електричної травми (ЕТ). ***Збір доказів.*** Зроблено ретроспективний інформативний пошук із використанням просторово-векторної описової моделі, що була доповнена ручним пошуком відповідних статей. Відібрано 42 сучасних літературних джерела за останні 10 років, з яких 76,2 % — за останні 5 років. ***Синтез доказів.*** Провідними механізмами розвитку неврологічної електротравми є електропорація, судинне ушкодження, демієлінізація та вивільнення термічної енергії, яка спричиняє руйнування макромолекул. Тому запропоновані теорії причинно-наслідкового зв'язку, альтернативна й нейрогуморальна гіпотези. Існує кілька типів нервового ушкодження при ЕТ, включно з циркуляторними порушеннями, ішемічною, гіпоксичною енцефалопатією, різними дистрофічними змінами клітинних структур мозку та внутрішньочерепними крововиливами. Найбільше ушкодження нейронів спостерігається при мінімальній травмі шкіри (найвагоміша кількість струму припадає на внутрішні структури). Можливі прямий або непрямий вплив ЕТ на головний мозок, коли черепно-мозкова травма викликана проходженням струму або прямим ушкодженням мозку внаслідок тупої механічної травми (або при поєднанні обох варіантів). Ураження електричним струмом може призвести до ішемічного інсульту через порушення кровообігу або до геморагічного — через пряме структурне ушкодження з некрозом та аксональною дегенерацією. Гострі ускладнення з боку центральної нервової системи включають зміни психічного статусу, кому, амнезію, квадриплегію та локалізований парез. Моторні порушення виникають частіше, ніж сенсорний дефіцит. Відстрочені неврологічні синдроми, як-от мозочкова атаксія або вегетативна дисфункція, можуть спостерігатися через тижні або місяці після первинної травми внаслідок розвитку подальшої ішемії, зменшення кількості клітин Пуркіньє або наявності крововиливів. У ситуаціях, коли шлях електричного струму перетинає спинний мозок, що зазвичай відбувається на рівні С4-С8 (тобто від однієї кінцівки до іншої), пацієнт має ризик ураження спинного мозку. Цей тип травми призводить до його прямого ушкодження, що спричиняє значні неврологічні розлади. ***Висновки.*** Провідними механізмами розвитку неврологічної ЕТ є електропорація, судинна травма, демієлінізація та вивільнення термічної енергії. Ураження головного мозку при ЕТ можливе, навіть коли голова не знаходиться в прямому електричному колі. Механізми розвитку відстрочених неврологічних ускладнень поки що цілком не з'ясовані.

Ключові слова: електрична травма; головний мозок; мозочок; спинний мозок; патофізіологія; огляд