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MEDICINE AND PHARMACY

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No 275

MEDICINE AND PHARMACY

Physiological mechanisms of Wallerian degeneration in brachial plexopathy: the role of axonal transport and energy metabolism

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Abstract. The goal is to investigate the pathophysiological mechanisms of Wallerian degeneration of nervous structures caused by long-term compression when using turnstiles in the conditions of military operations, in particular, during explosive wounds in Ukraine. An analysis of morphological, biochemical, and functional changes accompanying the Wallerian degeneration process after traumatic injury of the brachial plexus was carried out. The study is based on the classical experiments of August Waller (1850) with the intersection of the hyoid and vagus nerves in frogs, adapted to the modern understanding of ischemic damage to peripheral nerves. Special attention is paid to the time sequence of degenerative processes in the distal segment of the axon after the loss of communication with the neuron's body, including the destruction of the axonal cytoskeleton and myelin sheath. Conclusion. Long-term use of turnstiles leads to ischemic nerve damage and the development of Wallerian degeneration, which determines the degree of neurological deficit in plexopathies. The value lies in the fact that the results of the study form the physiological basis for the development of effective methods of therapy and rehabilitation of patients with compression neurodegenerative injuries in conditions of combat injuries.

Keywords: *Wallerian degeneration (WD), Schwann cells, fiber, neuroprotective therapy.*

In the modern world, especially in conditions of war and the use of tourniquets in cases of explosive wounds, the problem of neurodegeneration due to long-term compression of nerve structures becomes especially relevant. Prolonged use of tourniquets can lead to ischemic nerve damage, which causes their degradation, particularly Wallerian degeneration.

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Research into the physiological basis of this condition is key to advancing effective treatment and recovery methods. Wallerian degeneration (WD) is an active process of gradual destruction of the distal segment of the axon that occurs after loss of its connection with the neuron's cell body due to trauma or compression. This process is accompanied by specific morphological, biochemical, and functional changes that lead to the destruction of the axonal cytoskeleton and myelin sheath. Our research is based on the experiment of Augustus Waller, who in 1850, while studying the WD process, transected the hypoglossal (n. hypoglossus) and vagus (n. vagus) nerves, observing subsequent changes in frog behavior. The scientist noted that the distal part of the nerve underwent progressive degeneration, while the proximal part (connected to the neuron) remained morphologically intact; therefore, the process had a characteristic temporal sequence. Brachial plexus injury, or plexopathy, is often accompanied by Wallerian degeneration, which is the leading pathophysiological mechanism determining the degree of neurological deficit in cases of traumatic, compressive, and ischemic damage.

Schwann cells are key to maintaining nerve fiber viability, regulating myelin sheath formation, contributing to axonal degradation and restoration, and also demonstrating phenotypic plasticity, which opens prospects for therapeutic interventions. In the context of plexopathy, Schwann cells acquire special importance due to the specific physiological features of this structure. Their activity determines the quality and speed of recovery. Also critical for nerve fiber functioning is axonal transport, which ensures the movement of mitochondria (the primary source of energy in the form of ATP) and other molecules between the neuronal cell body and the periphery. These organelles represent an essential element, being the unique providers of ATP in distal axons with restricted protein synthesis. With Wallerian degeneration, mitochondrial transport is disrupted, making the functioning of Schwann cells, energy substrates, and their maintenance of the myelin sheath impossible. A decrease in ATP concentration leads to dysfunction of sodium-potassium ATPase, which causes membrane depolarization and opening of voltage-gated calcium channels. An increase in intracellular calcium concentration activates calpains, which begin to degrade cytoskeletal proteins, further disrupting axonal transport and creating a vicious cycle of degeneration. Energy

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deficiency in brachial plexus plexopathy causes dysfunction of various types of nerve fibers; progressive involvement of smaller-diameter fibers occurs. Aβ class fibers that provide tactile sensitivity begin to suffer, which is clinically manifested by decreased sensation, muscle atrophy, and trophic disorders. In Erb-Duchenne upper plexopathy, innervation by the axillary nerve is disrupted, manifested by inability to abduct the shoulder, which is clinically described as a "hanging hand" sign. Lower Klumpke-Dejerine plexopathy, affecting the C8-T1 roots, shows a different pattern with predominant involvement of the small muscles of the hand and forearm. Lesion of the occipital nerve causes a characteristic position of fingers - clawed hand. Total plexopathy is characterized by combination of all described symptoms with development of complete upper limb immobilization. The energy deficit in this case is so pronounced that almost all types of nerve fibers are affected. Early neuroprotective therapy aimed at maintaining energy metabolism with mitochondrial support agents can slow disease progression and improve functional prognosis in patients. Therefore, among recommended supplements, the following should be distinguished: coenzyme Q10, L-carnitine, and α-lipoic acid. These agents have antioxidant properties, reducing oxidative stress and decreasing free radical formation in nervous tissue. Typically, the treatment course for patients is 1-3 months, depending on the condition. For additional support, B vitamin complexes, NMN, and hydrogen antioxidants can be included, which combine well with basic therapy for Wallerian degeneration. In Ukraine, knowledge of various treatment regimens is not just an academic theory but a practical necessity. It makes certain that patients are supported even in adverse conditions, and doctors can adjust their response accordingly.

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