the relative volumes of damaged endotheliocytes, the density of microvessels per 1 mm² of the tissue of the subject were determined on the micropreparations of the prostate body Quantitative indicators were processed statistically.

Results and Discussion. It was established that the morphometric parameters of the venous vessels of the prostate changed significantly during long-term ethanol intoxication. It was found that with long-term ethanol intoxication in laboratory sexually mature white male rats, a pronounced structural rearrangement of the venous vessels of the prostate occurs, which is characterized by thinning of the vein walls, expansion of their lumen, venous congestion, atrophic processes in the intima, apoptotic, dystrophic and necrobiotic changes of endotheliocytes, endothelial dysfunction, hypoxia, dystrophic-necrotic changes in cells and stromal structures, infiltration and sclerosis. The degree of structural rearrangement of the venous vessels of the prostate dominates in experimental animals of the older age group.

Key words: prostate, venous bed, ethanol intoxication, age.

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DOI 10.29254/2077-4214-2023-2-169-374-380 UDC 591.483+616-001+612.014.46+616.441-008.64+616-08+591.169.1 ¹Demydchuk A. S., ¹Shamalo S. M., ²Kotyk T. L., ¹Raskaliei T. Ya., ¹Raskaliei V. B., ²Popadynets O. H., ²Tokaruk N. S. MORPHOLOGICAL CHANGES IN THE PERIPHERAL NERVES IN TRAUMA, MERCURY INTOXICATION, HYPOTHYROIDISM, AND THEIR CORRECTION ¹Bogomolets National Medical University (Kyiv, Ukraine) ²Ivano-Frankivsk National Medical University (Ivano-Frankivsk, Ukraine)

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Traumatic peripheral nerve injury (PNI) significantly impacts patients' quality of life, and its treatment necessitates a multidisciplinary approach. Additionally, different non-traumatic factors such as environmental toxicants and endocrine disorders can significantly affect the regenerative capacity of damaged nerves.

The aim of this study was to assess the morphological changes in the recovery of the damaged peripheral nerve, considering the impact of environmental heavy metal intoxication (mercury) and hypothyroidism, including their treatment.

The study involved a total of 90 white rats divided into three groups, each group further subdivided into subgroups (a and b). All rats underwent surgical cutting of the sciatic nerve. Subgroup Ia underwent sciatic nerve injury alone, while subgroups IIa and IIIa were additionally subjected to experimental mercurialism and hypothyroidism, respectively. Subgroups Ib, IIb, and IIIb received treatments with Cerebrolysin, Thiotriazoline, and L-Thyroxine+Miacalcic, respectively, in addition to the traumatic nerve injury and experimental influences. The damaged nerves were then examined using both light and electron microscopy assessments.

The findings of the study suggest that there are general patterns of de – and regeneration of the damaged peripheral nerve in the context of nootropic administration, experimental mercury intoxication, hypothyroidism, and their correction. In all groups, degeneration was observed, but a noteworthy delay in degenerative processes was observed specifically in cases involving delayed surgical nerve connection, mercurialism, and hypothyroidism, possibly due to the inhibition of Schwann cells. This led to a delayed regeneration process. However, the regeneration of the damaged nerve was significantly accelerated when the experimental pathological condition was corrected.

Key words: peripheral nerve, trauma, mercurialism, hypothyroidism, correction, regeneration.

Connection of the publication with planned research works.

The study is a fragment of scientific research conducted by the Bogomolets National Medical University "The influence of congenital and acguired hipothyroidism in the central and peripheral nervous status and the possibility of its pharmaceutical correction" (state registration № 0109U001804) and the Ivano-Frankivsk National Medical University "Ontogenetic peculiarities of the morphofunctional state of organs and tissues under conditions of iodine deficiency, hypothyroidism" (state registration № 0119U002847).

Introduction.

Traumatic peripheral nerve injury (PNI) is a prevalent condition among military traumas, often occurring alongside other injuries, which contributes to its high rates [1]. While diagnosing PNI may seem relatively straightforward, its treatment necessitates a multidisciplinary team approach. The complexity of PNI can lead to severe complications that significantly impact the patient's quality of life [2]. At the same time, PNI is commonly observed in civilian population, particularly as a result of sports trauma, injuries during everyday activities [3, 4], or clinical procedures [5]. The treatment of PNI remains challenging, as such injuries often result in the reduction in functional activities, require long-term rehabilitation, and increase socio-economic burden [6].

Rapid industrial development and the increasing impact of human activities on the environment require considering the presence of various occupational factors. It has been found that toxicants present in the environment can accumulate in different tissues and adversely affect their condition [7, 8]. Different toxicants can significantly influence the regeneration of damaged peripheral nerves. Mercury is one of the widely spread environmental heavy metals that has a strong association with neural damage and neurodegenerative diseases [9]. Therefore, intoxication with mercury, with or without treatment, has the potential to impair the regenerative capacity of peripheral nerves and delay functional recovery.

Endocrine disorders are another important factor that has a significant influence on nervous tissue. Among them, the prevalence of hypothyroidism and its strong relations with geographic and socio-economic aspects determine the significance of this pathology as a comorbidity of other diseases [10]. The association between hypothyroidism, cognitive impairment [11], and impaired nerve conduction velocity [12, 13] has been established. Management of patients with PNI on the background of hypothyroidism, with or without treatment of such comorbid endocrine pathology, should be also considered as such condition may potentially affect the treatment course.

The aim of the study.

This study aimed to investigate and compare the morphological changes in peripheral nerve recovery after trauma on the background of experimental mercury intoxication and hypothyroidism, including their treatment. By addressing this research gap, it will enable us to recognize the potential impact of occupational toxicants and endocrine pathology on the regenerative capacity and develop a sustainable background for developing effective treatment strategies and improving outcomes for PNI.

Object and research methods.

The experimental study involved 90 white rats (150-200 g) and was carried out in accordance with the "European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes" (Strasbourg, 2005) and the "General Ethical Principles of Animal Experiments" (Kyiv, 2013). The animals were kept in standard laboratory conditions and received water *ad libitum*.

The rats were divided into 3 groups:

• Group I (30 animals; divided into subgroup Ia (Control) and subgroup Ib (Control + Cerebrolysin), with 15 rats per subgroup). The left sciatic nerve was cut in the middle third, followed by surgical hemostasis and wound suturing. After 10 days, a repeated surgical procedure was performed to connect the ends of the severed sciatic nerve using epineural suturing. Subgroup Ib received intraperitoneal administration of Cerebrolysin (0.02 mg/ kg, for 21 consecutive days; Ebewe, Austria) starting from the third day of surgical nerve reconstruction. Subgroup Ia received no additional treatment.

• Group II (30 animals; divided into subgroup IIa (Hg-Control) and subgroup IIb (Hg + Thiotriazoline), with 15 rats per subgroup). The animals were initially subjected to experimental mercurialism by intraperitoneal administration of HgCl2 (1/100 LD50) for a duration of 2 weeks (Hg-Control). Then, the left sciatic nerve was surgically cut, and its ends were fixed together by sutures, maintaining a distance of 1-2 mm between the ends. Subgroup IIa received no additional treatment, while subgroup IIb received intraperitoneal administration of 100 mg/kg of a 2.5% Thiotriazoline solution for a duration of 14 days.

• Group III (30 animals; divided into subgroup IIIa (Hypothyroidism-Control) and subgroup IIIb (Hypothyroidism + Thyroxine + Miacalcic), with 15 rats per subgroup). In both subgroups, the animals underwent experimental hypothyroidism through thyroidectomy, followed by laboratory control of serum thyroxine levels [14]. Starting from day 3, the rats in subgroup IIIb were administered L-Thyroxine (10 μ g/kg orally; Farmak, Ukraine) and Miacalcic (1.0 MU/kg intramuscularly, every other day; Novartis, Switzerland). Subgroup IIIa received no additional treatment. After 100 days of the experiment, the left sciatic nerve was cut, followed by surgical hemostasis and wound suturing.

Tissue sample collection and microscopy assessment.

Six weeks after experimental nerve damage (surgical cutting of the sciatic nerve), the rats in all groups were euthanized using an overdose of sodium thiopental. The damaged sciatic nerves were collected for further assessment.

For light microscopy analysis, the routine silver impregnation staining procedure was applied.

For electron microscopy analysis, the tissue sample were fixed in 2.5% glutar-aldehyde with post-fixation in 1% osmium tetroxide. Semithin and ultrathin slides were prepared according to routine procedures.

Research results and their discussion.

In subgroup Ib (Control+Cerebrolysin), hypo – and hyperimpregnation of single nerve fibers were observed in the proximal ends of damaged nerves, that suggested the possibility of retrograde regeneration. Axonal fragmentation was preserved and visualized in different fields of view. The axons were identifiable with wavy contours. These characteristics were less pronounced as compared to subgroup Ia (Control). Regenerative neuroma formation with connective tissue cells and blood vessels ar-

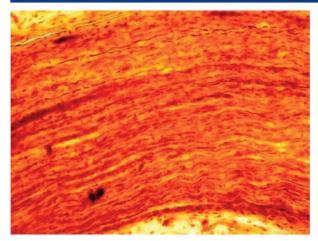


Figure 1 – Distal segment of the sciatic nerve in subgroup Ib (Control+Cerebrolysin). Micrograph. Silver impregnation. Magnification: x400.

ranged in bundles was observed. Small amounts of newly formed nerve fibers with stalled growth cones were found; they showed organized growth along the axis of the nerve, with only a few of them located deviantly, obliquely, transversely, or recurrently. In addition, evenly distributed, thin bundles of collagen fibers were identified, with numerous newly formed blood vessels found between them. In the distal segment of the damaged peripheral nerve, the processes of ascending degeneration were observed. Large numbers of collagen fiber bundles and connective tissue cells were visualized. Single regenerated nerve fibers exhibiting a uniform arrangement were identified. Thus, based on the findings, it could be concluded that a homogeneous and delicate connective tissue scar was formed, allowing newly formed nerve fibers to grow out of the proximal segment of the damaged nerve and extend into the distal one, showing mainly uniform distribution and direct linear growth (fig. 1).

An electron microscopic study of the distal segment of the damaged peripheral nerve showed the regeneration of nerve fibers. Most nerve fibers appeared undamaged, showing round and oval appearance. No splitting or swelling of the myelin lamellae was observed, with the myelin sheath itself appearing continuous and homogeneously osmiophilic. The basement membrane around the fibers was found to be disorganized. The axons displayed a normal structural appearance, with their axolemma coming into tight contact with myelin sheaths. Undamaged mitochondria with cristae and vesicles were clearly visualized in the axoplasm. The mitochondrial matrix appeared transparent, lacking crystals and granules. Microtubules and neurofilaments in most fibers were well-developed and arranged in groups forming the exoskeleton (**fig. 2**).

The regeneration of endoneurium was clearly observed around individual groups of nerve fibers and even some Schwann cells. Several fibroblast processes were observed to be in contact with each other, although occasional gaps between them were also evident. The processes of connective tissue cells, specifically fibroblasts, exhibited intact mitochondria and large numbers of vesicles. Active Schwann cells were found around nerve fibers. The nuclei of Schwann cells contained large amounts of euchromatin, indicating a high synthetic activity of the latter. Heterochromatin was also visualized but beneath the nucleolemma, being dispersed close to and around the nucleus pore complexes.



Figure 2 – Newly formed myelin fibers in subgroup Ib (Control+Cerebrolysin). Electron micrograph. Magnification: x10000.

Thus, subgroup Ib (Control+Cerebrolysin) was characterized by more extensive regenerative processes, specifically the repair of stromal components and myelination. A complete and overall regeneration of endoneurium was observed, which led to the regeneration of newly formed nerve fibers and accelerated the functional recovery of tissues and organs that experienced denervation due to modeling sciatic nerve injury [15]. Such morphological changes indicating positive dynamics of regenerative processed in the peripheral nerves when using nootropics were also observed in other studies [16, 17].

In subgroup IIb (Hg + Thiotriazoline), nerve fibers of the proximal segment of the sciatic nerve exhibited slight signs of irritation. Some nerve fibers were characterized by a mild swelling and wavy contours. However, it should be noted that fragmented axons were rare as compared to subgroup IIa (Hg-Control). The areas with poorly impregnated tissue appeared less pronounced; visually, the connective tissue of the regenerative neuroma showed growth, accompanied by numerous blood vessels and nerve fibers growing through the scar. Fibroblasts were the most numerous connective tissue cells. Individual thick bundles of collagen fibers were visualized. The axial cylinders showed a more organized arrangement as compared to subgroup IIa (Hg-Control); although some of them exhibited transverse motion. A few axons appeared with growth cones at their tips. Thus, regenerative neuroma was characterized by a greater degree of connective tissue differentiation and an increase in the number of nerve fibers growing through it and displaying a higher level of organization and relatively orderly arrangement (fig. 3).

In subgroup IIb (Hg + Thiotriazoline), nerve fibers of the distal segment of the damaged nerve also showed minor signs of irritation, but an increase in their quantity and diameter with preserved wavy contours was visualized. Electron microscopy assessment revealed numerous Schwann cells with no morphological signs of damage. The morphology of the Golgi apparatus, endoplasmic reticulum, and lysosomes showed no significant alterations. However, the cisterns of the endoplasmic reticulum exhibited slight expansion. The number of structurally intact nerve fibers increased due to the activation of macrophages and Schwann cells [18]. Some Schwann cells were involved in the formation of new nerve fibers. Newly formed nerve fibers still exhibiting pathological characteristics were mostly arranged in groups, exhibited various shapes and sizes; however, signs of their regeneration were observed. Most of those fibers appeared

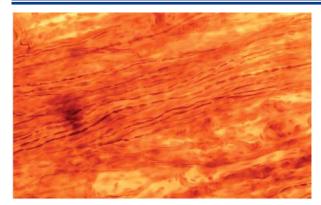


Figure 3 – Regenerative neuroma in subgroup IIb (Hg + Thiotriazoline). Electron micrograph. Silver impregnation. Magnification: x400.

with an axon containing large numbers of microtubules and neurofilaments; additionally, mitochondria exhibiting well-defined cristae were visualized. No splitting of the myelin lamellae was observed in the myelin sheath, although wavy contours of some fibers were preserved (fig. 4). The number of collagen fibers with no signs of depolymerization increased. The rough endoplasmic reticulum showing no signs of irritation was well visualized in fibroblasts indicating their functional activity. The numbers of blood capillaries with electron dense endothelium increased.

It can be assumed that, on the background of mercury intoxication and its correction by Thiotriazoline, the morphological changes in the peripheral nerve were characterized by slight dystrophic damage [19].

Therefore, positive changes were observed in the proximal and distal segments and the area of regenerating neuroma of the sciatic nerve in animals of subgroup IIb (Hg + Thiotriazoline). A significant reduction in nerve fiber swelling was seen, with a reduction in destructive processes. Both newly formed nerve fiber density and myelin sheath thickness increased. At this stage of study, some nerve fibers exhibiting signs of irritation such as wavy contours and uneven impregnation were still present; however, no axonal fragmentation was an indicative of recovery. Thiotriazoline has a positive effect on Schwann cells, thereby accelerating the processes of myelination and promoting the recovery of the damaged sciatic nerve [20].

In subgroup IIIa (Hypothyroidism-Control), significant numbers of degenerative ovoids were observed in the distal segment of the sciatic nerve at the light microscopic

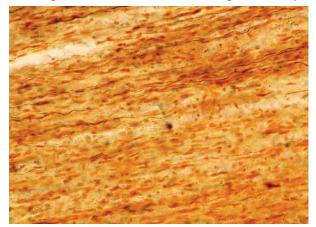


Figure 5 – Site of nerve damage in subgroup IIIa (Hypothyroidism-Control). Micrograph. Silver impregnation. Magnification: x400.



Figure 4 – Newly formed myelin fibers in subgroup IIb (Hg + Thiotriazoline). Electron micrograph. Magnification: x10000.

level. The interstitial space appeared noticeably enlarged due to edema and cell debris. Some nerve fibers were fragmented, with unclear wavy contours. Nerve fiber density in this section of the nerve decreased significantly (fig. 5).

Ultrastructural examination of the distal segment of the sciatic nerve revealed vacuolated remnants of myelin sheaths exhibiting different stages of degradation in the cytoplasm of Schwann cells (fig. 6).

Active degenerative changes in the distal segment of the nerve were accompanied by regenerative changes manifested by the formation of non-myelinated fibers. They were arranged in clusters, displayed variations in their diameter, and contained a significant number of lysosomes, secretory vesicles, electron dense inclusions and a small quantity of neurofibrils and microtubules in the axoplasm.

Ultrastructural examination of the distal segment of the damaged sciatic nerve in subgroup IIIb (Hypothyroi dism+Thyroxine+Miacalcic) revealed an increase in the number of nerve fibers alongside a reduction in the interstitial volume (fig. 7) as compared to subgroup IIIa (Hypothyroidism-Control).

Quality assessment of the sciatic nerve samples under study revealed a higher regenerative activity characterized by the formation of new nerve fibers in rats with hypothyroidism receiving correction as compared to animals with untreated hypothyroidism. Ultrastructural examina-

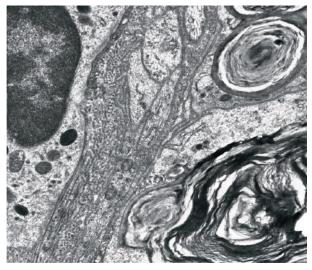


Figure 6 – Fragments of the destroyed myelin sheath in the cytoplasm of Schwann cells in the distal segment of the sciatic nerve in subgroup IIIa (Hypothyroidism-Control). Electron micrograph. Magnification: x12000.

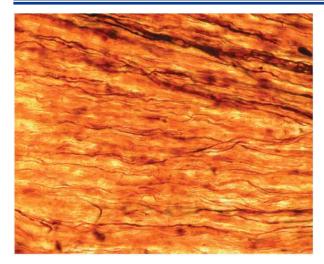


Figure 7 – Site of nerve damage in subgroup IIIb (Hypothyroi dism+Thyroxine+Miacalcic). Micrograph. Silver impregnation. Magnification: x400.

tion revealed a large number of clustered non-myelinated fibers exhibiting uneven contours and containing a significant number of vacuoles and electron dense inclusions in the axoplasm. Newly formed myelin fibers exhibited an incomplete myelin sheath, showing layering in localized areas or being dense, occasionally swollen, with enlarged periaxonal space and local invagination of myelin in axon (fig. 8). Newly formed myelin fibers with an intact myelin sheath and a normal axon, with no signs of pathological alterations, were observed in the field of view.

The regeneration of the damaged sciatic nerve in hypothyroidism (subgroup IIIa) exhibited a wave-shaped pattern similar to that observed in normal thyroid function (subgroup Ia). However, thyroid hormone deficiency significantly hindered that process [21]. The destruction and phagocytosis of primarily damaged nerve fibers altered new fiber formation; larger nerve fibers appeared predominantly aberrant and therefore, underwent simultaneous phagocytosis by Schwann cells, leading to the next stage of nerve regeneration, specifically repeated degeneration of newly formed atypical fibers. The sciatic nerve, on the background of Hypothyroidism + Thyroxine + Miacalcic (subgroup IIIb), underwent the phases of regeneration and degeneration faster, which was associated with no significant delay in the progression of degenerative changes, characteristic of the morphological alterations in the damaged nerve in hypothyroid rats (subgroup IIIa). The onset of regeneration was delayed due to low phagocytic activity and the absence of apoptosis in Schwann cells [22, 23]. Further destruction of



Figure 8 – Newly formed myelin fibers: local layering of the myelin sheath, enlargement of the periaxonal space in the distal segment of the sciatic nerve in subgroup IIIb (Hypothyroidism + Thyroxine + Miacalcic). Electron micrograph. Magnification: x10000.

damaged myelin fibers took place at a slower pace and had a distinct course which differed from the progression of degeneration in rats of subgroup Ia and subgroup IIIa.

Conclusions.

Based on the results, it can be concluded that there are common patterns of de – and regeneration of the damaged peripheral nerve in the context of nootropic administration, experimental mercury intoxication, hypothyroidism, and their correction. Although degeneration was evident in all groups, a noteworthy delay in degenerative processes was observed specifically in cases involving delayed surgical nerve connection, mercurialism, and hypothyroidism, possibly due to the inhibition of Schwann cells. As a result, regeneration was delayed, exhibiting a wave-shaped pattern due to the transition of Schwann cells from their phagocytic role to myelination. However, it should be noted that initiation of regeneration is significantly accelerated when the experimental pathological condition is corrected, leading to the regeneration of the damaged nerve.

Prospects for further research.

The data obtained can serve as a basis for developing the methods to improve the processes of peripheral nerve regeneration in clinical practice. The results of the study on the regeneration of the damaged sciatic nerve on the background of various complicating factors will broaden the understanding of both researchers and practicing physicians regarding the regenerative properties of nerve trunks and ways to influence these processes, thus stimulating further investigations.

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ОСОБЛИВОСТІ МОРФОЛОГІЧНИХ ЗМІН ПЕРИФЕРИЧНИХ НЕРВІВ В УМОВАХ ТРАВМИ, ІНТОКСИКАЦІЇ РТУТ-ТЮ, ГІПОТИРЕОЗІ ТА ЇХ КОРЕКЦІЇ

Демидчук А. С., Шамало С. М., Котик Т. Л., Раскалей Т. Я., Раскалей В. Б., Попадинець О. Г., Токарук Н. С. Резюме. Вступ. Травматичні ушкодження периферичного нерва (ТУПН) займають вагоме місце серед травм як в умовах воєнного часу, так і в побуті, призводять до значних ускладнень і знижують якість життя пацієнтів. Лікування ТУПН зазвичай комплексне та потребує мультидисциплінарного підходу, супроводжується тривалими функціональними обмеженнями та необхідністю реабілітації. Токсиканти навколишнього середовища, такі як ртуть, можуть здійснювати вплив на процеси регенерації периферичних нервів, що зумовлює важливість урахування впливу таких факторів на нервову тканину. Ендокринна патологія, зокрема гіпотиреоз, також має значний вплив на нервову тканину, впливає на когнітивні функції, нейропровідність та є додатковим аргументом для врахування його впливу при лікуванні ТУПН. Розуміння зв'язку між ТУПН, токсикантами та ендокринними розладами може сприяти розробці ефективних стратегій лікування та покращенню результатів відновної терапії периферичних нервів.

Метою даного дослідження є встановлення морфологічних особливостей регенераторних процесів у периферичних нервах після травми на тлі експериментальної інтоксикації ртуттю та гіпотиреозу, а також за умови їх корекції. Встановлення цих особливостей дозволить ідентифікувати потенційний вплив токсикантів і ендокринної патології на регенеративну здатність периферичних нервів та розробити підґрунтя стратегій лікування таких ушкоджень.

Об'єкт і методи дослідження. Дослідження виконано на 90 білих щурах, які були розподілені на три групи, кожна з яких складалася з підгруп «а» і «б». Травматичне ушкодження сідничого нерва моделювали шляхом його перерізання з відтермінованим ушиванням усім щурам. Підгрупам ІІа та ІІІа додатково моделювали експериментальну інтоксикацію ртуттю та гіпотиреоз, відповідно. Підгрупи Іб, ІІб та ІІІб додатково отримували лікування церебролізином, тіотриазоліном та L-тироксином+міакальциком, відповідно. Морфологічне дослідження включало світлооптичний та ультраструктурний аналіз.

Результати. Травматичне ураження супроводжується структурною перебудовою проксимального, дистального відрізків та зони травми нерва. Є виражене розшарування та набряк ламел, дистрофічні зміни у аксональних циліндрах, нейролемі та в сполучнотканинному каркасі нерва. Застосування церебролізину має позитивний вплив на стромальні елементи нерва та мієлінізацію. Меркуріалізм зумовлює нерівномірність імпрегнації при морфологічному дослідженні, виражену хвилястість контурів. При корекції тіотриазоліном помітні позитивні зміни в структурі нейролемоцитів, що, у свою чергу, покращує процеси мієлінізації. Гіпотиреоз супроводжується загальмуванням дегенеративних змін, що сповільнює регенераторні процеси в травмованому нерві. Корекція синтетичними формами тиреоїдних гормонів активує нейролемоцити, у результаті чого чіткіше контурує мієлінова оболонка, нейролема та візуалізуються органели аксоплазми.

Висновки. Встановлено загальні закономірності де – та регенерації ураженого периферичного нерва за умови застосування ноотропів, експериментальної інтоксикації ртуттю, гіпотиреозу та їх корекції. Дегенеративні процеси були значно сповільнені в умовах наявності додаткових пошкоджуючих факторів, ймовірно, через інгібування шванноцитів. У свою чергу, це призводить до відтермінування ініціалізації регенераторних процесів. Однак, корекція патологічних станів (ноотропами, антиоксидантами та цитопротекторами, синтетичними формами тиреоїдних гормонів), які супроводжували регенерацію пошкодженого нерва, безумовно пришвидшували її настання.

Ключові слова: периферичний нерв, травма, меркуріалізм, гіпотиреоз, корекція, регенерація.

MORPHOLOGICAL CHANGES IN THE PERIPHERAL NERVES IN TRAUMA, MERCURY INTOXICATION, HYPOTHYROIDISM, AND THEIR CORRECTION

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Abstract. Introduction. Traumatic peripheral nerve injury (PNI) is a common occurrence in both military and civilian populations, leading to significant complications and reducing patients' quality of life. The treatment of PNI requires a multidisciplinary approach due to its complex nature and the potential for long-term functional limitations and rehabilitation needs. Environmental toxicants, such as mercury, have been implicated in the impairment of peripheral nerve regeneration and delay in functional recovery, highlighting the importance of considering occupational factors and their impact on nerve tissue. Endocrine disorders, particularly hypothyroidism, have significant influence on nervous tissue and can affect cognitive function and nerve conduction, emphasizing the need to address comorbidities in the management of PNI. Understanding the relationship between PNI, environmental toxicants, and endocrine disorders can contribute to the development of effective treatment strategies and improve outcomes for patients with PNI.

The aim of the study. This study aimed to investigate and compare the morphological changes in peripheral nerve recovery after trauma on the background of experimental mercury intoxication and hypothyroidism, including their treatment. By addressing this research gap, it will enable us to recognize the potential impact of occupational toxicants and endocrine pathology on the regenerative capacity and develop a sustainable background for developing effective treatment strategies and improving outcomes for PNI.

Object and research methods. The study involved a total of 90 white rats divided into three groups, each group further subdivided into subgroups (a and b). Traumatic PNI was simulated by surgical cutting of the sciatic nerve and delayed suturing in all rats. Subgroups IIa and IIIa were additionally subjected to experimental mercurialism and hypothyroidism, respectively. Subgroups Ib, IIb, and IIIb were administered additional treatments with Cerebrolysin, Thiotriazoline, and L-Thyroxine+Miacalcic, respectively. The morphological study included light microscopy and ultrastructural analysis.

Results. Traumatic injury was accompanied by structural remodeling of the proximal and distal segments and the area of nerve injury. Significant splitting and swelling of the myelin lamellae, dystrophic changes in axons, neurolemma, and connective tissue framework of the nerve were observed. Cerebrolysin had a positive effect on the stromal components and myelination. Mercurialism caused uneven impregnation during morphological examination, leading to pronounced waviness of the contours. Correction with Thiotriazoline resulted in noticeable positive changes in the structure of Schwann cells, which, in turn, improved the processes of myelination. Hypothyroidism was accompanied by the inhibition of degenerative changes, which slowed down the regenerative processes in the damaged nerve. Correction with synthetic forms of thyroid hormones activated Schwann cells, resulting in clearer delineation of the myelin sheath, neurolemma and visualization of axoplasmic organelles.

Conclusions. General patterns of de – and regeneration of the damaged peripheral nerve in the context of nootropic administration, experimental mercury intoxication, hypothyroidism, and their correction, were identified. Degeneration was significantly postponed in all investigated factors, possibly due to the inhibition of Schwann cells. Thus, it resulted in delayed regeneration. However, it should be noted that initiation of regeneration is significantly accelerated when the experimental pathological condition is corrected (by nootropes, antioxidants and cytoprotectors, synthetic forms of thyroid hormones).

Key words: peripheral nerve, trauma, mercurialism, hypothyroidism, correction, regeneration.

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