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PATHOGENESIS OF TUNNEL NEUROPATHIES IN CHILDREN WITH CEREBRAL PALSY, FOOT DEFORMITIES AND JOINT CONTRACTURES

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Summary.

Tunnel neuropathy is one of the complications of the clinical course in children with cerebral palsy. According to various sources, neuropathies of the lower extremities account for 65% to 80%, and those of the upper extremities for 12% to 20%. However, the aetiopathogenesis of compression-ischaemic neuropathies has not been sufficiently studied. Muscle tone disorders in children with cerebral palsy are often combined with mental and speech disorders, which complicates the diagnosis of neuropathies and requires the development of new examination algorithms.

Objective: *to determine the diagnostic criteria and pathogenesis of tunnel neuropathies in children with cerebral palsy.*

Materials and methods. *The materials obtained from the study of 40 patients with cerebral palsy, spastic diplegia and tetraparesis were analysed. Depending on the muscle tone disorder, patients were divided into 3 groups. Clinical and instrumental examination methods were used for diagnosis.*

The study was conducted in accordance with the principles of the Helsinki Declaration. This study was approved by the Ethics Committee of the P. L. Shupyk National Medical Academy of Postgraduate Education (protocol No. 9 dated November 6, 2017). Informed consent was obtained from patients for the conduct of the study.

Results. *The study found that the occurrence of tunnel neuropathies depends on deformities of the upper and lower limbs in children with muscle tone disorders. An inverse correlation was noted between the stage of tunnel neuropathy and the speed of excitation propagation, as well as a direct correlation between pain syndrome and subfascial pressure, which gives priority to instrumental examination methods.*

Conclusions. *1. The occurrence of spastic-ischaemic neuropathies in children with cerebral palsy is closely related to foot deformities and joint contractures. 2. Assessment of subfascial pressure indicators in combination with ENMG results makes it possible to determine the severity of the pathological process and identify the optimal treatment method.*

Keywords: *Children; Cerebral Palsy; Orthopaedics; Bone Structure; Muscles; Neuropathy.*

Introduction

Compression–ischaemic neuropathy constitutes one of the complications in the clinical trajectory of cerebral palsy in children, representing 23%–40% of all peripheral nervous system disorders. Tunnel neuropathy is a key contributor to the multifactorial genesis of pain syndrome [3]. As reported in the literature, lower extremity neuropathies account for 65% to 80% of cases, while upper extremity involvement ranges from 12% to 20% [7].

Compression and microcirculatory disturbances of peripheral nerves within anatomically constrained fibrous and fibro-osseous tunnels of the limbs have been extensively investigated and described [3,7,8]. However, tunnel neuropathies associated with muscle spasticity, elevated intracompartmental pressure, joint contractures, and foot deformities in children with cerebral palsy remain understudied and warrant further investigation.

According to O. Danilov's classification, children with cerebral palsy exhibit muscle tone disorders manifesting as reflex tonic activity, spasticity, and rigidity [3]. Nevertheless, the interrelationship between the specific type of muscle tone abnormality, intracompartmental pressure, and the stage of tunnel neuropathy has not been systematically evaluated.

Muscles are interconnected via myofascial chains [3]. Under conditions of heightened spasticity, they become incorporated into pathological synergy patterns, leading to the progressive development of joint contractures and limb deformities. However, the association between these structural alterations and the onset of tunnel neuropathies in children with cerebral palsy remains insufficiently characterised.

Multiple clinical and instrumental methods are available for the diagnosis of tunnel neuropathy [7,8]. To date, however, no validated diagnostic criteria have been established for children with cerebral palsy presenting with compressive neuropathies.

Objective: to determine the diagnostic criteria and pathogenesis of tunnel neuropathies in children with cerebral palsy.

Materials and methods. Data obtained from 40 patients with cerebral palsy, spastic diplegia, and tetraparesis were analysed. Participants were stratified into three groups based on the predominant type of muscle tone disorder: Group 1: 14 patients with reflex tonic tension; Group 2: 13 patients with spasticity; Group 3: 13 patients with rigidity. All patients reported neurogenic pain of varying intensity.

Pain severity was assessed dynamically using the numerical rating scale (NRS):

0-3 points: absent or mild pain, causing minimal discomfort;

4-6 points: moderate pain;

7-10 points: severe pain.

Provocative tests were performed to support the diagnosis of tunnel syndrome:

Tinel's sign: percussion over the nerve course elicited pain or paresthesia in the corresponding dermatome;

Elevation test: with the patient supine, each lower limb was elevated for one minute; nerve traction and reduced hydrostatic arterial pressure led to impaired neural perfusion, manifesting as pain and paresthesia;

Phalen's test: sustained wrist flexion for one minute induced paresthesia in the median nerve distribution.

Spasticity was evaluated using the Modified Ashworth Scale, which quantifies resistance to passive joint movement at varying angular velocities.

Nerve conduction velocity was assessed by electroneuromyography (ENMG).

The stage of spastic–ischaemic neuropathy (a term adopted to reflect the combined aetiology of spasticity and ischaemia, per Berzins and Dumbre's classification) was determined as follows:

Stage I – transient subjective sensations;

Stage II – persistent subjective sensations;

Stage III – objective sensory deficits;

Stage IV – persistent motor deficits.

Ultrasonographic evaluation of peripheral nerves was performed using an ALOKA device (Hitachi, Japan) equipped with a 5 MHz linear transducer. Triplex ultrasonography of the lower extremity vasculature was conducted.

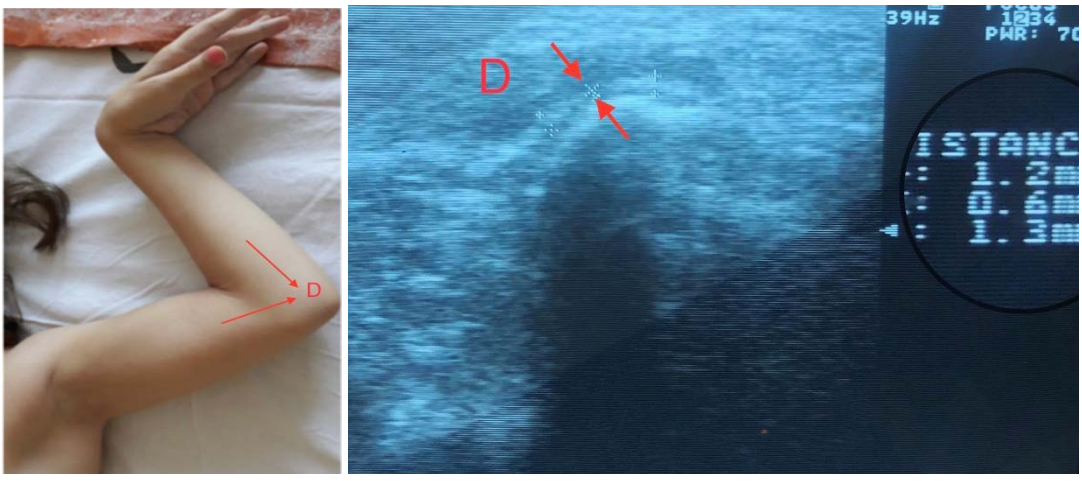
Intracompartmental pressure was measured invasively using the Stryker Intra-Compartmental Pressure Monitor (Stryker Corporation, USA).

Inclusion criteria comprised diagnosis of cerebral palsy with documented muscle tone abnormalities. Exclusion criteria were: absence of neurological pathology; post-traumatic or congenital contractures unrelated to cerebral palsy.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was granted by the Local Ethics Committee of the All-Ukrainian Centre for Motherhood and Childhood, National Academy of Medical Sciences of Ukraine. Written informed consent was obtained from parents or legal guardians.

Research results and discussion

Theoretical justification of the aetiopathogenesis of tunnel neuropathies in the upper and lower extremities in children with cerebral palsy. Ultrasonographic examination of anatomical tunnels revealed that mechanical nerve injury is determined by topographical features of nerve passage, which vary according to limb positional vectors. During high-amplitude, undifferentiated movements, the nerve undergoes extraneural excursion within the tunnel. In children with cerebral palsy, pathological conditions – including muscle tone abnormalities and elevated intracompartmental pressure – induce oedema of the epineurium and endoneurium, alongside loosening and fibrosis of the perineural connective tissue. These changes result in reduced nerve calibre, increased intraneural pressure, venous stasis, and subsequent spastic–ischaemic neuropathy. Oedema triggers an inflammatory response and promotes adhesion formation. Prolonged compression leads to progressive fibrous adhesions, which restrict physiological nerve gliding within the anatomical tunnel. Histopathological alterations at sites of nerve stenosis include demyelination, axonal fragmentation, and axonal loss [6-9 Furthermore, joint contractures and foot deformities markedly alter tunnel dimensions. Figure 1 illustrates the «hourglass» sign of ulnar nerve compression, as demonstrated by sonography.



A B

Figure 1. A – Photograph of the left upper limb of patient B, aged 16. Diagnosis: cerebral palsy, tetraparesis, spasticity-predominant muscle tone disorder; flexion contracture of the elbow and radiocarpal joints; ulnar nerve tunnel neuropathy, stage II. B – Longitudinal sonogram of the left ulnar nerve compression site: D – distal segment, diameter 1.2 mm; compression zone, diameter 0.6 mm; proximal segment, diameter 1.3 mm.

Muscles are anatomically interconnected, forming myofascial meridians, as described in Myers' classification [3,4]. Under conditions of heightened spasticity, these structures are recruited into pathological synergy patterns. For instance, in equinus foot deformity, tensile forces propagate along the superficial posterior and spiral myofascial lines, resulting in nerve compression at anatomically vulnerable sites (Figure 2).

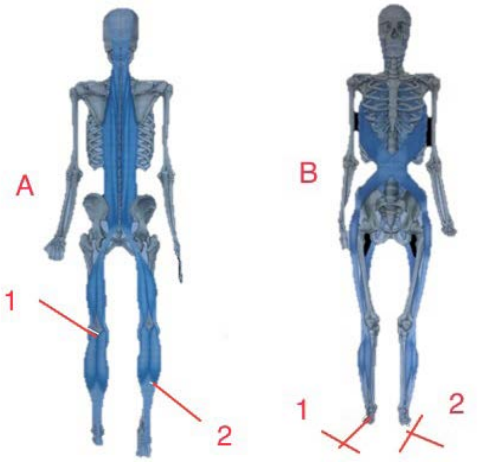


Figure 2. A – Superficial posterior myofascial line: 1 – proximal tibial nerve tunnel neuropathy; 2 – distal tibial nerve tunnel neuropathy; B – Spiral myofascial line: 1 – interdigital nerve tunnel neuropathy; 2 – terminal deep peroneal nerve tunnel neuropathy.

Increased loading on the forefoot induces tensile stress on the interdigital nerves beneath the deep transverse metatarsal ligament, leading to traction-induced spastic–ischaemic neuropathy.

In equinovarus foot deformity, additional tunnel syndrome arises from overstretching of the dorsifoot musculoligamentous apparatus, resulting in traction injury to the deep peroneal nerve.

The tibial nerve, accompanied by the posterior tibial artery and vein, traverses the Gruber canal in the middle third of the leg. Spasticity of the soleus, flexor digitorum longus, flexor hallucis longus, and tibialis posterior muscles contributes to spastic–ischaemic tibial neuropathy.

Similarly, in the popliteal fossa, tibial nerve compression occurs secondary to traction exerted by the popliteus muscle and narrowing of the tendinous arch of the soleus.

According to Danilov's classification, pronated foot deformities are categorised into three types: (1) planovalgus; (2) equinoplanovalgus; and (3) pronated deformity, frequently associated with flexion contractures of the toes at the interphalangeal joints [4,5]. The specific deformity type determines the pattern of tunnel neuropathy development.

For example, in planovalgus deformity, compensatory external tibial torsion and internal femoral rotation develop as a result of pathological synergies within the lateral and deep front myofascial lines, culminating in tunnel syndrome formation (Figure 3).

Medial arch collapse in planovalgus foot deformity results in plantar–medial displacement of the navicular bone, leading to compression of the medial plantar nerve. Concomitant calcaneal valgus deviation induces tension in the flexor retinaculum, causing tibial nerve compression within the tarsal tunnel.

Planovalgus deformity is frequently associated with knee joint contractures. Apposition of the biceps femoris tendon to the fibular head produces compression of the common peroneal nerve.

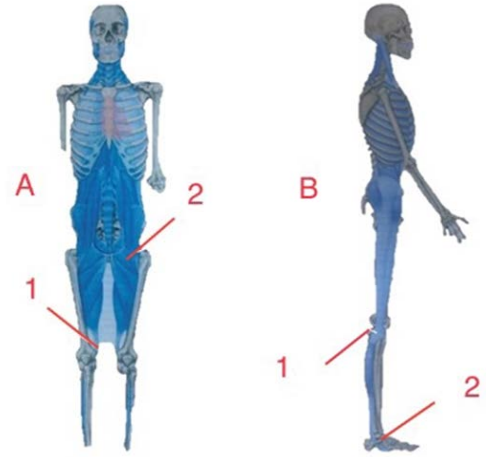


Figure 3. A – Deep front myofascial line: 1 – sural nerve tunnel neuropathy; 2 – sciatic nerve tunnel neuropathy; B – Lateral myofascial line: 1 – common peroneal nerve tunnel neuropathy; 2 – tibial nerve tunnel neuropathy.

Planovalgus deformity with external tibial torsion may induce excessive traction and ischaemia of the sural nerve.

Internal rotational contracture of the femur, in combination with planovalgus foot deformity, leads to piriformis muscle dysfunction and subsequent spastic–traction–ischaemic sciatic neuropathy. Similarly, equinoplanovalgus deformity causes piriformis muscle shortening and the development of spastic–ischaemic sciatic neuropathy.

In patients with tetraparesis and a «spastic hand», tensile forces propagate along the deep and superficial front myofascial lines of the upper limb, increasing the risk of upper extremity tunnel neuropathies (Figure 4).

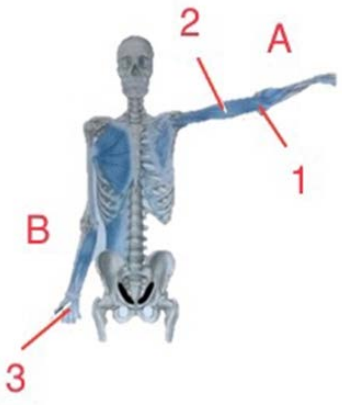


Figure 4. A – Deep front myofascial line of the arm: 1 – ulnar nerve tunnel neuropathy; 2 – musculocutaneous nerve tunnel neuropathy. B – Superficial front myofascial line of the arm: 3 – median nerve tunnel neuropathy.

Pronation contracture of the forearm, flexion contracture of the radiocarpal joint, and adduction–flexion deformity of the first digit collectively contribute to median nerve compression within the carpal tunnel.

In elbow flexion contracture, the medial epicondyle of the humerus functions as a pulley-like structure, stretching the ulnar nerve during maximal forearm flexion and resulting in compression within the cubital tunnel.

The association between upper and lower limb deformities and the development of tunnel neuropathies in children with muscle tone disorders is summarised in Table 1.

Table 1

Association between foot and limb deformities and tunnel neuropathies in children with cerebral palsy

Type of deformation	Tunnel neuropathy	Compressive structures
Equine deformity. Equinoplanovalgus deformity	Spastic–traction–ischaemic neuropathy of the interdigital nerves (Morton's metatarsalgia)	Intermetatarsal space; tension and compression of the nerve beneath the thickened deep transverse metatarsal ligament.
Equinus deformity Equinovarus deformity Equinoplanovalgus deformity Varus deformity	Spastic–traction–ischaemic neuropathy of the terminal deep peroneal nerve (anterior tarsal tunnel syndrome)	Tunnel beneath the extensor hallucis brevis, in the anterior aspect of the ankle joint and dorsum of the foot.
Equinus deformity Equinovarus deformity Equinoplanovalgus deformity Flat foot	Spastic–ischaemic neuropathy of the tibial nerve Spastic–traction–ischaemic neuropathy of the tibial nerve	Gruber canal, formed by the soleus, flexor digitorum longus, flexor hallucis longus, and tibialis posterior muscles
Equinus deformity Equinovarus deformity Equinoplanovalgus deformity Planovalgus deformity Flat foot	Spastic–ischaemic neuropathy of the tibial nerve Spastic–traction–ischaemic neuropathy of the tibial nerve	Popliteus muscle and tendinous arch of the soleus
Equine deformity Equinoplanovalgus deformity Planovalgus deformity	Spastic–ischaemic neuropathy of the sciatic nerve Spastic–traction–ischaemic neuropathy of the tibial nerve	Pelvic compartment: compression between the piriformis, superior gemellus, and sacrospinous ligament
Planovalgus foot deformity	Spastic–ischaemic neuropathy of the common peroneal nerve (Guillain-Barré syndrome, de Sèze syndrome, Blondin-Walter syndrome, occupational paralysis of tulip bulb diggers)	Proximal lateral leg at the fibular neck (junction of biceps femoris tendon and fibular head) .
Planovalgus foot deformity Pronated foot	Spastic–traction–ischaemic neuropathy of the sural nerve	Compression in the area passing through the fascial plane 5-6 cm proximal to the medial femoral epicondyle, beneath the sartorius muscle.

Planovalgus foot deformity Pronated foot	Spastic–traction–ischaemic neuropathy of the tibial nerve	Compression of the shaft in the tarsal canal (space between medial malleolus and flexor retinaculum).
Planovalgus foot deformity Pronated foot	Spastic–ischaemic neuropathy of the medial plantar nerve	Compression between the tendon of the flexor digitorum longus, tibialis posterior muscle, and navicular bone.
Spastic hand	Spastic–traction–ischaemic neuropathy of the ulnar nerve	Cubital tunnel: medial humeral epicondyle, medial intermuscular septum, and flexor carpi ulnaris aponeurosis.
Spastic hand	Spastic–ischaemic neuropathy of the median nerve	Carpal tunnel: transverse carpal ligament, flexor pollicis longus tendon, and flexor digitorum superficialis tendons.
Spastic hand	Spastic–ischaemic neuropathy of the musculocutaneous nerve	Nerve perforation of the coracobrachialis muscle, coursing beneath the short head of the biceps brachii.

Examination results.

All patients exhibited positive or weakly positive responses to provocative testing across multiple anatomical sites, confirming the presence of tunnel syndromes. Pain intensity scores, spasticity grades, nerve conduction velocities, and mean intracompartmental pressures within the myofascial compartments of the upper and lower limbs are presented in Table 2.

Table 2

Clinical and instrumental parameters across study groups

Indicators (in points)	Group I, n=14 M ± m	Group II, n=13 M ± m	Group III, n=13 M ± m
Spasticity (Modified Ashworth Scale)	2,5 ± 0,2	3,0 ± 0,15	3,8 ± 0,17
Nerve conduction velocity (m/s)	34,5 ± 0,4	26,3 ± 0,5	30,8 ± 0,2
Neurogenic pain (NRS)	4,7 ± 1,2	7,1 ± 1,3	3,0 ± 1,1
Intracompartmental pressure (lower limbs, mm Hg)	17,5 ± 1,3	21,5 ± 1,4	16,4 ± 1,5
Intracompartmental pressure (upper limbs, mm Hg)	18,5 ± 1,3	22,51 ± 1,2	17,1 ± 1,4

Ultrasonographic evaluation assessed anatomical integrity of the nerve trunk, internal echotexture, demarcation of nerve margins, and perineural tissue characteristics. Reduced intratunnel nerve mobility during dynamic manoeuvres and perineural inflammatory exudate were observed in all groups. Group II exhibited fibrotic adhesions, whereas Group III demonstrated significant calibre reduction and early signs of nerve atrophy. Sonographic findings in the upper and lower extremities revealed the most pronounced venous stasis in patients with spasticity: Doppler ultrasonography of the posterior tibial vein yielded values of 0.26 ± 0.03 in Group I and 0.24 ± 0.06 in Group II, compared with the reference range of 0.35-0.45.

An inverse correlation was observed between the stage of tunnel neuropathy and nerve conduction velocity, and a direct correlation was identified between pain intensity and intracompartmental pressure (Table 3).

Table 3

Relationship between muscle tone disorder, neuropathy stage, intracompartmental pressure, nerve conduction velocity, and pain severity

Type of muscle tone disorder	Stage of neuropathy	Subfascial pressure (mm Hg)	Nerve conduction velocity (m/s)	Neurogenic pain (NRS)
Reflex tonic tension	I-II	18-20	31-35	3-5
Spasticity	II-III	21-25	20-30	6-8
Rigidity	0–I	14-17	30-40	1-3

Thus, alterations in intracompartmental pressure contribute to the development of tunnel neuropathy at varying stages

Discussion.

Limb pain in children with cerebral palsy is multifactorial, encompassing myofascial, articular, and neurogenic components [3]. Nevertheless, the neurogenic factor plays a pivotal role in pain genesis, driven by microcirculatory disturbances, elevated intracompartmental pressure, fascial thickening, narrowing of anatomically vulnerable tunnels, and consequent spastic–ischaemic neuropathy.

Several pathogenetic theories of compression–ischaemic neuropathy have been proposed: dysmetabolic, inflammatory, vascular, and mechanical [1, 10]. In our view, the neuropathy observed in children with cerebral palsy is best characterised as spastic–ischaemic; in cases of sustained nerve overstretching, a spastic–traction–ischaemic variant may develop.

Muscles of the upper and lower limbs, pelvic girdle, and trunk are integrated into continuous myofascial meridians [3, 11]. In spasticity, tensile forces propagate along these chains, generating pathological motor synergies, foot deformities, joint contractures, and predisposing conditions for tunnel neuropathy.

Recent studies have demonstrated impaired lower limb perfusion during reflex tonic activity, spasticity, and rigidity in children with cerebral palsy [3, 9]. This is exacerbated by orthopaedic interventions (e.g., serial plaster casting), which increase mechanical pressure on vulnerable anatomical sites, elevate intraneural pressure, augment resistance in epineurial arterioles, and induce neural and perineural ischaemia – findings corroborated by triplex ultrasonography. These changes impede rehabilitation in children with muscle tone disorders complicated by tunnel neuropathy.

Elevated intracompartmental pressure within myofascial compartments of the extremities represents a key aetiological factor in tunnel neuropathy and chronic compartment syndrome [1,11]. However, pressure values vary with the type of tone abnormality: in reflex tonic tension, pressures exceeding 20 mm Hg confer risk of tunnel neuropathy; spasticity is associated with the highest pressures (up to 25 mm Hg) and pronounced clinical manifestations; Rigidity induces degenerative–dystrophic muscle changes, partial restoration of venous return, reduced soft-tissue oedema, and consequent pressure reduction (14-17 mm Hg), promoting regression of neuropathic symptoms.

In neurologically intact individuals, tunnel neuropathy diagnosis relies on symptom assessment, history, physical examination, provocative testing, and, in severe cases, instrumental studies [2, 8, 10]. In children with cerebral palsy, however, diagnostic accuracy is compromised by coexisting cognitive and speech impairments. Consequently, electroneuromyography (ENMG) constitutes the most reliable diagnostic modality, with parameters strongly correlating with neuropathy stage.

Future research should focus on developing pathogenesis-guided conservative and surgical strategies, alongside novel diagnostic algorithms for tunnel neuropathy in this population.

Conclusions

1. The development of spastic–ischaemic neuropathies in children with cerebral palsy is closely associated with the type of muscle tone disorder, foot deformities, and joint contractures.

2. Integrated assessment of sonographic parameters, intracompartmental pressure, and electroneuromyography (ENMG) findings enables accurate staging of the pathological process and selection of optimal therapeutic interventions.

Conflict of interest. The authors declare no conflict of interest.

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ПАТОГЕНЕЗ ТУНЕЛЬНИХ НЕВРОПАТІЙ У ДІТЕЙ, ХВОРИХ НА ДЦП, З ДЕФОРМАЦІЯМИ СТОПИ ТА КОНТРАКТУРАМИ СУГЛОБІВ

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Резюме.

Тунельна невропатія є одним із ускладнень клінічного перебігу в дітей з дитячим церебральним параличем (ДЦП). За даними різних джерел, на долю невропатій нижніх кінцівок припадає від 65% до 80%, а верхніх кінцівок – від 12% до 20%. Але етіопатогенез компресійно-ішемічних невропатій є недостатньо вивчений. Порушення м'язового тонусу в дітей із ДЦП часто поєднується з психічними та мовними розладами, що ускладнює діагностику невропатій і потребує розробки нових алгоритмів обстеження.

Мета: визначити діагностичні критерії та патогенез виникнення тунельних невропатій у дітей, хворих на дитячий церебральний параліч.

Матеріали і методи дослідження. Проаналізовані матеріали, отримані при дослідженні 40 пацієнтів, хворих на ДЦП, із спастичною диплегією та тетрапарезом. У залежності від порушення м'язового тонусу, пацієнти були розділені на 3 групи. Для діагностики застосовували клінічні та інструментальні методи обстеження.

Дослідження проводилося відповідно до принципів Гельсінської декларації. Дане дослідження схвалене комісією з питань етики НМАПО імені П. Л. Шупика (протокол № 9 від 06.11. 2017). На проведення досліджень була отримана інформована згода пацієнтів.

Результати. За результатами дослідження встановлена залежність виникнення тунельних невропатій від деформацій верхніх та нижніх кінцівок у дітей з порушеннями м'язового тонусу. Відзначали обернену кореляцію між стадією тунельної невропатії та швидкістю розповсюдження збудження, а також, прямий кореляційний зв'язок між больовим синдромом і субфасціальним тиском, що створює пріоритетність інструментальних методів обстеження.

Висновки.

1. Виникнення спастично-ішемічних невропатій у дітей, хворих на ДЦП, тісно пов'язане з деформаціями стопи і контрактурами суглобів

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2. Оцінка показників субфасціального тиску в поєднанні з результатами електронейроміографії дає можливість визначити ступінь важкості патологічного процесу та визначити оптимальний метод лікування.

Ключові слова: діти; дитячий церебральний параліч; ортопедія; структура кістки, м’язів; невропатія.

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