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Causes of pain in the muscles of the lower extremities in children with cerebral palsy

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A high level of muscle spasticity in children with cerebral palsy is one of the causes of degenerative and dystrophic changes in tissues. Against the background of high spasticity, there is a violation of blood flow in the vessels of the lower extremities, which leads to changes in subfascial pressure and the formation of primary pain syndromes. The analysis of the literature shows the need to study the issues related to the mechanism of formation of changes in the muscles and determine the primary causes of pain. Objective. To study the mechanism of changes in the muscles of the lower extremities, as well as the primary causes of pain syndrome. Methods. The analysed indicators were obtained during the treatment of 40 patients with cerebral palsy, who were divided into 3 groups depending on the muscle tone disorder. Clinical and instrumental methods were used to examine patients. The data obtained were statistically processed with the determination of M ± m, the coefficient of reliability according to Student's criteria. Results. According to the results of the study, high spasticity leads to impaired blood flow and increased subfascial pressure in the musculofascial sheaths. There is an inverse correlation between the increase in subfascial pressure, the diameter of the vessel lumen and blood flow velocity. This is one of the causes of degenerative and denervation changes in muscles and fascia. It was proved that the occurrence of primary pain in the lower extremities is caused by various etiological causes: tissue ischemia; increased subfascial pressure; narrowing of pathologically significant anatomical areas in which nerves lie; degenerative and dystrophic changes in the joints. Conclusions. The study found that the cause of changes in the muscles and fascia is a violation of blood flow, a change in subfascial pressure in the musculofascial cases of the lower extremities. The occurrence of pain in the lower extremities in children with cerebral palsy is multifactorial in nature and consists of myofascial, articular, and tunnel syndromes.

Високий рівень спастичності м'язів у дітей, хворих на ДЦП, є однією з причин виникнення дегенеративно-дистрофічних змін у тканинах. На фоні значної спастики спостерігається порушення кровотоку судин нижніх кінцівок, що призводить до зміни субфасціального тиску та формування первинних больових синдромів. Аналіз літератури свідчить про необхідність дослідження питань, які стосуються механізму формування змін у м'язах і визначення первинних причин болю. Мета. Вивчити механізм виникнення змін у м'язах і фасціях нижніх кінцівок, а також первинні причини больового синдрому. Методи. Проаналізовані показники отримані під час лікування 40 пацієнтів, хворих на ДЦП, які були розподілені на 3 групи залежно від порушення м'язового тонусу. Для обстеження використовували клінічні й інструментальні методики. Отримані дані оброблені статистично з визначенням M ± m, коефіцієнта достовірності за критеріями Стьюдента. Результати. Виявлено, що висока спастичність призводить до порушення кровотоку та зростання субфасціального тиску в м'язово-фасціальних футлярах. Відзначено обернену кореляцію між підвищенням субфасціального тиску, діаметром просвіту судин і показниками швидкості кровотоку. Це є однією з причин виникнення дегенеративних і денерваційних змін у м'язах та фасціях. Доведено, що виникнення первинного болю в нижніх кінцівках викликане різними етіологічними причинами: ішемією тканин; підвищенням субфасціального тиску; звуженням патологічно значимих анатомічних ділянок, у яких залягають нерви; дегенеративно-дистрофічними змінами в суглобах. Висновки. Встановлено, причини змін у м'язах і фасціях — порушення кровотоку, коливання субфасціального тиску в м'язово-фасціальних футлярах нижніх кінцівок. Виникнення болю в дітей, хворих на ДЦП, має мультифакторний характер і складається з міофасціального, суглобового, тунельного синдромів. Ключові слова. Діти, церебральний параліч, субфасціальний тиск, больовий синдром.

Keywords. Children, cerebral palsy, subfascial pressure, pain syndrome

Introduction

One of the causes of motor impairment in children with cerebral palsy is joint contractures caused by spasticity and the subsequent degeneration of muscles [15, 17]. Excessive activity of the lower parts of the brain is the starting mechanism of motility disorders in children with cerebral palsy [9, 18]. Pathological locomotion is characterized by a loss of selective muscle control, an imbalance between agonists and antagonists, abnormal muscle tone, and development of pain syndrome [2, 3].

The muscles that provide movements in the hip, knee joints and feet are most affected [6, 8, 12]. N. M. Nenko and S. V. Vlasenko showed that complex degenerative and denervation processes occur in muscles under conditions of constant spasticity [19]. The morphological composition of the tissues indicates signs of muscle degeneration: fibrillation, the presence of fibrous tissue. There is also a decrease in the number of vessels around which connective tissue elements are formed in a ring [16]. At the same time, the authors do not analyze the systemic blood flow of the lower extremities, which affects changes in muscle and connective tissues.

Changes in subfascial pressure are one of the negative factors affecting muscle trophicity [5, 23]. However, this indicator in the case of muscle spasticity has not been studied to date.

Research in recent years has shown that patients with cerebral palsy suffer from chronic pain, which is a consequence of muscle overstrain, the formation of pathological synergies, and an increase in contact stress in the patellofemoral and hip joints [7, 8, 10, 14]. Poirot et al. showed that in patients with cerebral palsy, pain is associated with joint contractures and foot deformities [20]. According to some authors, the cause of pain is myofascial syndrome [4, 26]. However, the definition of the primary causes of pain is not well understood.

Development of pain in the lower extremities is associated with tunnel neuropathies [11, 21, 25]. But the issues of etiopathogenesis, presentation and diagnosis of compressive lesions of the peripheral nerves of the lower limbs in children with cerebral palsy remain open.

Thus, the analysis of literary sources indicates the need for further study of issues related to the mechanism of formation of changes in muscles and fascia, determination of the primary causes of pain in children with cerebral palsy.

Purpose: to study the mechanism of changes in the muscles and fascia of the lower extremities, the primary causes of pain syndrome.

Materials and methods

The study was conducted in accordance with the principles of the Declaration of Helsinki. Research protocol No. 9 dated 06.11.2017 was approved by the local ethics commission of P. L. Shupyk National University of Health Care of Ukraine. Patients' informed consent was obtained for the research.

The analyzed indicators were obtained during the treatment of 40 subjects with cerebral palsy, spastic diplegia and tetraparesis. In all cases, hip and knee joint contractures and foot deformities were recorded.

Patients were divided into three groups depending on the violation of muscle tone (according to the author's classification) [5]. The first group included 14 children with reflex tonic tension, the second group involved 13 patients with spasticity, the third one 13 patients with rigidity.

To determine the reference values, a group of 14 children aged 12–14 years without neurological disorders was recruited.

Patients with cerebral palsy and impaired muscle tone were included in the study, and children with lower limb injuries and neurogenic diseases of other etiology were excluded.

The analysis of the pain syndrome in time course was carried out according to the numeric rating scale (NRS), according to its results, pain is classified (in points):

– 0–3 — absent or weak, which is of little concern;
– 4–6 — moderate or medium intensity;
– 7–10 — strong intensity [5, 13].

The Modified Ashworth Scale was used to assess spasticity in patients. It is aimed at studying the resistance of muscles to passive movement in a joint with variable speed [19].

Motor motility analysis was carried out according to the Gross Motor Function Classification System for Cerebral Palsy (GMFCS) [19].

The degree of changes in the muscles, as well as the cross-sectional area of the muscles of the lower limbs was determined on the ALOCA ultrasound diagnostic device (USD) with a linear sensor with a frequency of 5 MHz. In order to objectify the study, the ratio of the size of the cross-section of the muscle to its length was calculated (*m. biceps femoris, m. gastrocnemius, m. tibialis anterior, m. tibialis posterior, m. peroneus brevis*). The study of blood flow

in the lower extremities was carried out by ultrasound dopplerography.

Measurements of subfascial pressure were carried out according to the classic invasive technique using the serial device Stryker Intra-Compartmenta Pressure Monitor. On the tibia, this procedure was performed taking into account four bony-fascial cases. The needle is inserted into the anterior fascial sheath in the middle third of the lower leg 2 cm lateral to the tibial crest. In the lateral case — in the middle third of the leg, in the projection of the fibula. The needle is inserted into the posterior deep sheath at the same level with a margin of 1 cm from the medial edge of the tibia. In the back surface case — in the middle of the back surface of the lower leg, at the same level [22, 23]. Determination of subfascial pressure was carried out under local anesthesia: 2–3 ml of 2 % lidocaine solution.

Depending on the type of foot deformity, subfascial pressure was measured in the anterior myofascial sheath in heel abnormality; in the lateral one in case of pronation; in the superficial-posterior part in equino-varus impairment.

Results

Ultrasound examination of the Hamstring group and the muscles of the lower leg showed a moderate increase in cross-sectional area indicators in patients with reflex tonic muscle tension. The muscles had a uniform structure, reduced echogenicity, elongated or oblique-longitudinal fiber direction. When the degree of activity of a certain myofascial chain increased, the fascia, as adaptive links of the musculoskeletal system, reacted by compacting their structures. Thus, in the process of ultrasound examination, the average thickness of the shin fascia (anterior, lateral, or superficial posterior), depending on the deformation of the foot, increased to 0.5 mm (± 0.1) for the reference value of 0.3 mm (± 0.1). Moderate synovitis of the knee and supracalcaneal joints was noted (Fig. 1).

In patients with impaired muscle tone manifested in the form of spasticity, the muscles had a heterogeneous structure due to areas of reduced echogenicity, distinct striations, and the direction of the fibers was obliquely transverse. The thickness of muscle fibers was significantly increased in volume. The average thickness of the lower leg fascia increased to 0.7 mm (± 0.2). During the study of the knee joint, increased echogenicity of the posterior horns of the medial and lateral menisci, shortening and thickening of the posterior cruciate ligament, signs of severe synovitis were noted (Fig. 2).

In patients with muscle stiffness as a result of their slow transformation and the occurrence of degenerative-dystrophic changes, the ultrasound parameters reflected a decrease in the cross-sectional area (Table 1). The muscle had a heterogeneous structure due to areas of reduced echogenicity, combined with areas of hyperechogenicity, characteristic of fibrous tissue.

An increase in the acoustic shadow in the muscles indicated the formation of scar tissue, containing multiple calcifications. The average thickness of the fascia increased to 1.3 mm (± 0.2). Pronounced degenerative-dystrophic changes in the joints involved fragmentation of the posterior horns of the medial and lateral menisci, heterogeneity of the structure

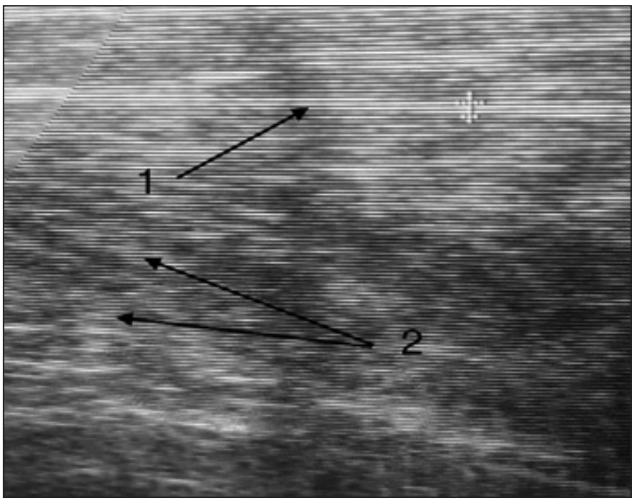

Fig. 1. A 14-year-ol patient K., diagnosis: cerebral palsy with reflex tonic muscle tension. Sonography of the calf muscle: 1 — fascia, diameter — 0.5; 2 — muscle fibers of the oblique-longitudinal direction

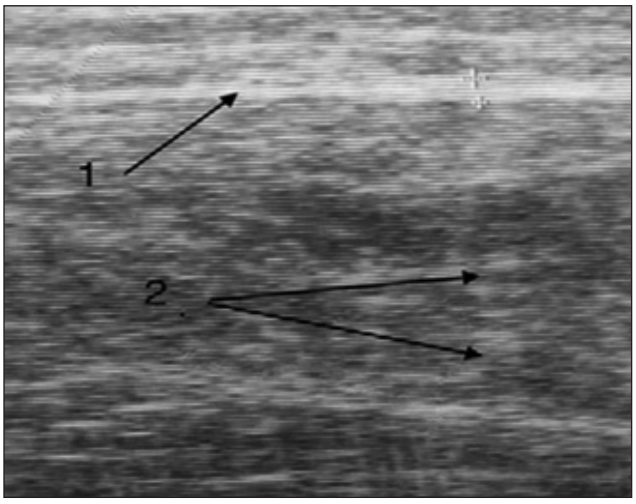

Fig. 2. A 13-year-old patient V., diagnosis: cerebral palsy with impaired muscle tone in the form of spasticity. Sonography of the calf muscle: 1 — fascia, diameter — 0.7; 2 — destructured muscle fibers, increased in volume

of the collateral, cruciate ligaments (Fig. 3). To assess changes in muscle mass in children with cerebral palsy, we have developed its index:

$$MMI = ML: (CSAR + CSAF),$$

where ML is the muscle length; CSAR is the cross-sectional area of the muscle; CSAF is the cross-sectional area of the fascia; MMI is the muscle mass index.

According to the results of the study, this index changed depending on the degree of impaired muscle tone (Table 1).

An increase in the cross-sectional area of the muscles, the formation of fibrosis centers indicated a long-term subacute inflammatory process in the muscle tissue, resulting in not only the deterioration of contractile-restorative properties, thickening of the fascia, but also in a change in subfascial pressure (Table 2).

An increase in subfascial pressure in myofascial sheathes (more than 20 mm Hg) was one of the reasons for the formation of chronic compartment syndrome. The relationship between the increase in subfascial pressure and the muscle mass index was recorded (Fig. 4).

We have developed a subfascial pressure coefficient to determine the compensatory and restorative properties of the muscle and diagnostic criteria for chronic compartment syndrome. The determination of the coefficient consisted in dividing the in-

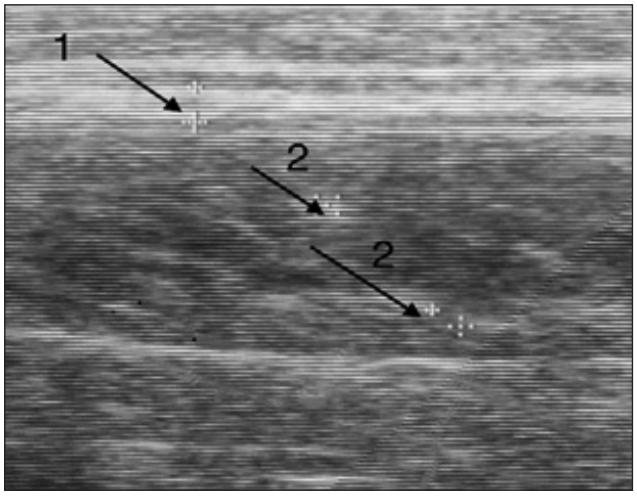

Fig. 3. A 15-year-old patient S., diagnosis: cerebral palsy with impaired muscle tone in the form of rigidity. Sonography of the calf muscle: 1 — fascia, diameter — 1.2; 2 — fibrous tissue in the thickness of muscle fibers

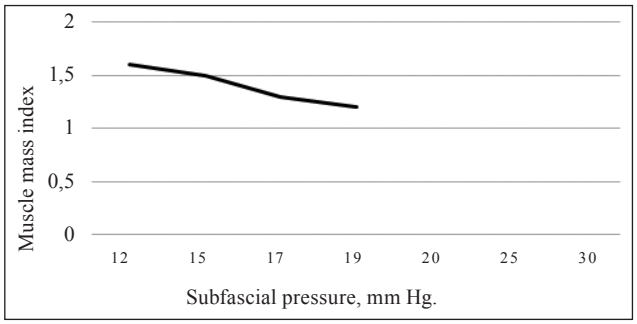

Fig. 4. Dependence of muscle mass index on subfascial pressure

Table 1

Indicators of muscle mass index in children with cerebral palsy

Muscle	Group 1, n = 14 (M ± m)		Group 2, n = 13 (M ± m)		Group 3, n = 13 (M ± m)	
	Muscle mass index					
	reference value	disorder	reference value	disorder	reference value	disorder
m. biceps femoris	1.7 ± 0.2	1.4 ± 0.1	1.7 ± 0.2	1.3 ± 0.5	1.7 ± 0.2	2.4 ± 0.4
m. gastrocnemius	2.9 ± 0.5	2.5 ± 0.4	2.9 ± 0.4	2.2 ± 0.7	2.9 ± 0.7	4.2 ± 0.7
m. tibialis anterior	3.9 ± 0.7	2.7 ± 0.6	3.9 ± 0.6	2.5 ± 0.4	3.9 ± 0.6	7.2 ± 1.1
m. tibialis posterior	3.2 ± 0.4	2.4 ± 0.5	3.2 ± 0.5	2.2 ± 0.3	3.2 ± 0.4	5.4 ± 0.8
m. peroneus brevis	4.2 ± 0.6	3.5 ± 0.3	4.2 ± 0.7	3.0 ± 0.6	4.2 ± 0.5	5.5 ± 0.6

Table 2

Indicators of subfascial pressure

Muscle	Group 1, n = 14 (M ± m)		Group 2, n = 13 (M ± m)		Group 3, n = 13 (M ± m)	
	Muscle mass index					
	reference value	disorder	reference value	disorder	reference value	disorder
Anterior	8.2 ± 1.2	17.3 ± 1.1	8.2 ± 1.2	18.5 ± 1.4	8.2 ± 1.2	6.2 ± 1.1
Lateral	10.4 ± 1.1	19.3 ± 1.3	10.4 ± 1.1	21.5 ± 1.2	10.4 ± 1.1	8.4 ± 1.5
Superficial-posterior	7.3 ± 1.4	16.1 ± 1.2	7.3 ± 1.4	17.4 ± 1.6	7.3 ± 1.4	5.3 ± 1.4
Mean indicator	8.6 ± 1.2	17.6 ± 1.2	8.6 ± 1.2	19.1 ± 1.4	8.6 ± 1.2	6.6 ± 1.3

dex of subfascial pressure, which was fixed before the load, by the index, which was determined 5 minutes after the loading of the triceps muscle of the leg for 30 seconds. Thus, the subfascial pressure in the anterior musculo-fascial sheath of the lower leg before the load was equal to 17.0 mm Hg, 5 minutes after exercise — 28.0 mm Hg, then the subfascial pressure coefficient was equal to 0.6 (for the reference value of 0.8), which indicated a violation of the contractile-restorative properties of the muscle and the formation of a chronic compartment syndrome.

According to the results of duplex scanning of the arteries of the lower extremities in patients with impaired muscle tone (femoral, popliteal, anterior and posterior tibial arteries), the speed and spectral characteristics of blood flow in standard points of the locations of the arteries of the lower extremities remained practically unchanged.

Triplex scanning of the deep veins of the lower extremities (popliteal, posterior tibial) showed a decrease in the diameter of the venous lumen in the deep veins in patients with increased subfascial pressure (Fig. 5).

Indicators of the diameter of the venous lumen of superficial veins (large and small subcutaneous) remained practically unchanged. An inverse correlation was noted between the increase in subfascial pressure and the diameter of the lumen of the deep veins (Fig. 6).

The reference value of pressure in venous capillaries in children was from 15 to 25 mm Hg in a horizontal body position. Subfascial pressure, which increased to 20 mm Hg triggered chronic compartment syndrome: violation of venous blood flow and development of ischemia of soft tissues. Clinically, this is manifested by pain, motor motility disorders, increased muscle tone of the affected segment of the limb, and paresthesias (Table 3). The intensity of pain depends on the primary etiological causes and the degree of muscle tone impairment (Fig. 7).

In reflex tonic muscle tension patients of the first group according to the results of ultrasound examination were found to have signs of synovitis of large joints, an increase in the cross-sectional area of muscles, impaired blood flow, swelling of soft tissues, leading to an increase in subfascial pressure and, as a result, the development of arthralgic and myofascial pain. In patients of the second group, in whom a violation of muscle tone manifested itself in the form of spasticity, in addition to an increase in the intensity of arthralgic and myofascial pain, neurogenic pain developed, caused by the compaction of the fascia structure, the narrowing of pathologically significant anatomical areas, development of tunnel syndrome and the formation of compression-ischemic neuropathy.

In the third group, a decrease in pain intensity due to denervation and degenerative processes in muscles and joints was observed.

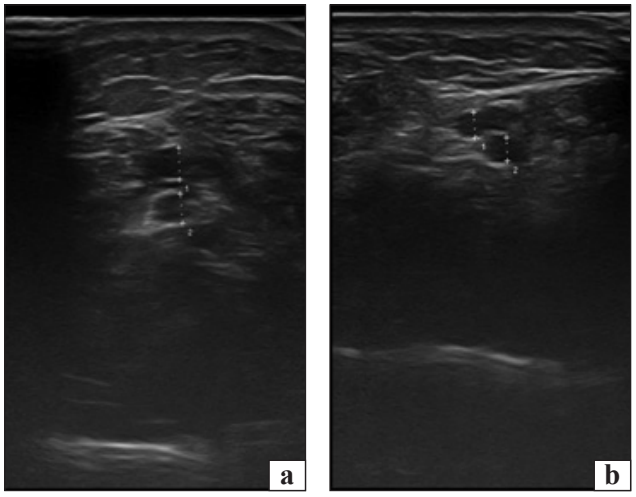

Fig. 5. A 16-year-old patient V., diagnosis: cerebral palsy, hemiparetic form with reflex tonic muscle tension of the right lower limb. Doppler imaging of the posterior tibial vein: a — left lower limb (1D — 0.34; 2D — 0.31); b — right lower limb (1D — 0.26; 2D — 0.24)

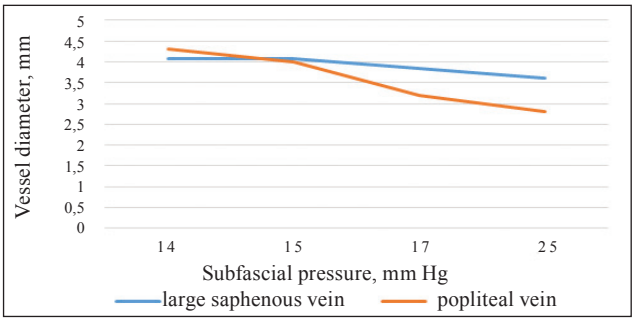

Fig. 6. Dependence of the diameter of venous vessels on subfascial pressure

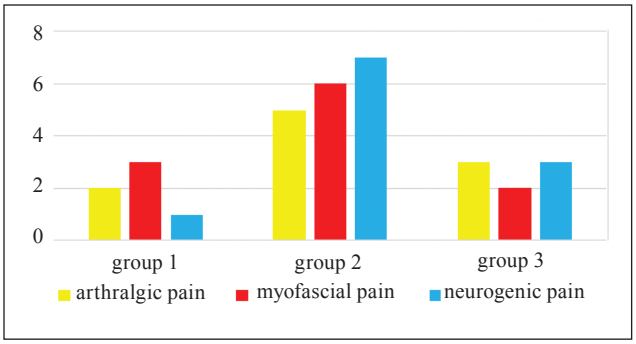

Fig. 7. Intensity of the pain syndrome

Table 3

Indicators of clinical examination

Indicator (in points)	Group 1, n =14 (M ± m)	Group 2, n =13 (M ± m)	Group 3, n =13 (M ± m)
Assessment of spasticity by the Ashworth Scale	2.50 ± 0.20	3.00 ± 0.15	3.80 ± 0.17
Arthralgic pain	2.00 ± 0.10	5.20 ± 0.21	3.10 ± 0.21
Myofascial pain	3.10 ± 0.30	6.10 ± 0.23	2.00 ± 0.10
Neurogenic pain	1.00 ± 0.07	7.10 ± 0.19	3.00 ± 0.20

The results of motor motility assessment, which was carried out in groups according to the gross motor functions classification system (GMFCS), were as follows: Group 1 — 10 patients — grade 1 violations, Group 4 – grade 2; Group 2 — 10 subjects — grade 2, 3 cases — grade 3; Group 3 — 5 patients — grade 3, 8 children — grade 4.

We investigated the causes of the pain syndrome. An increase in subfascial pressure, ischemia of soft tissues, compaction of fascias triggers the narrowing of pathologically significant areas and the formation of compression-ischemic neuropathies of peripheral nerves. The first tunnel develops as a result of compression of the *nervus ischiadicus*, which passes between *m. iliopsoas* and *m. gemellus superior*. Later on, it can result in compression of the *nervus tibialis* in the area of the Hamstring group in the middle third of the thigh, causing tension in the joint capsule and the fascia of the popliteal space. In the upper part of the soleus muscle there are *arcus tendineus musculi solei*, where *arteria et venae poplitea* and *nervus tibialis pass*. Hypertonus *m. soleus* and *m. popliteus* during equinus deformity of the foot leads to compression of the nerve in the popliteal area and in the Gruber tunnel. Collapse of the medial vault during pronation deformation of the foot leads to displacement of the navicular bone in the medial-plantar direction, causing compression of the *nervus plantaris medialis*. In the case of valgus deviation of the calcaneus, the tension of the *retinaculum musculorum flexorum* leads to compression of the tibialis nerve in the tarsal tunnel. During the formation of the heel of the foot, weakening of the *musculus tibialis posterior* causes hypertonicity of the pronators: *musculus fibularis brevis* and *musculus fibularis longus*. Conditions are created for compression of the *nervus fibularis communis* under the head of the fibula. Thus, regardless of the location of the tunnel syndrome, the innervation of the entire tibial group (muscles innervated by the tibial nerve) is disturbed, the pain syndrome increases, and joint contractures increase (Fig. 9).

The pathological chain in children with cerebral palsy is as follows: pathological innervation — excessive spasticity — impaired blood flow — chronic compartment syndrome — pain syndrome — degenerative-dystrophic changes in muscles and joints — hip and knee contractures, foot deformities.

Discussion

According to some authors, the cause of morphological changes in muscles is their spasticity with the formation of pathological synergies [12, 19]. Our own research proves that the main cause of changes in muscles and fascia is microcirculation disturbance and subacute inflammatory process with edema in soft tissues. This leads to an increase in subfascial pressure in the musculo-fascial cases of the leg, structural changes in the tissues. Compaction of the fascia structure leads to the narrowing of pathologically significant anatomical areas, the emergence of tunnel syndrome and the formation of compression-ischemic neuropathy. A similar opinion is held by R. Schleip, who during the study of the thoracolumbar fascia observed the compaction of structural elements against the background of a chronic inflammatory process in the vertebral-motor segment, which caused pain syndrome [24].

I. Poirot et al. believe that in most patients with cerebral palsy, the pain is associated with contractures and deformations of the feet and has a combined character [20]. In our opinion, flexion contractures of hip and knee joints

on the contrary, lead to a decrease in the nerve tension reflex and a reduction in pain syndrome. Also, the research showed that contractures cause the development of reactive synovitis and arthralgia, which are most pronounced in patients with a high degree of spasticity.

According to S. Strafun's research, an increase in subfascial pressure in children with Volkman's contracture leads to ischemic-compressive neuropathy and morpho-functional changes in muscles [23]. Our own observations prove that spasticity is the cause of impaired venous blood flow, edema, increased muscle volume, and changes in subfascial pressure. Thus, the average indicator of this pressure in the myofascial cases of the lower leg in children with ce-

rebral palsy with impaired muscle tone in the form of spasticity reached 19.1 mm Hg, which is twice the physiological indicators.

Y. Bezsmertny claims that the values of subfascial pressure increase with the deterioration of the functional state of the limb [1]. However, according to our research, a decrease in the subfascial pressure coefficient (< 0.8) indicated a decrease in the contractile-restorative properties of muscles and their fibrous degeneration. It can be one of the predictors of irreversible changes in muscles.

In children with cerebral palsy, the total mass of flexor muscles increases in relation to extensor muscles [5]. According to the results of our study, there is an inverse correlation between the indicators of the cross-sectional area of the flexor muscles and subfascial pressure. Thus, the muscle mass index decreases with an increase in subfascial pressure.

Most of the authors found morphological changes in muscles, fascia and blood vessels based on histological analysis of tissues [19]. Our own observations prove the possibility of assessing the presence of degenerative-dystrophic changes in tissues based on the results of ultrasound.

During the study of the histological material of the muscles of the lower limbs in children with cerebral palsy with impaired muscle tone, V. L. Lontkovskyi proved a decrease in the number of vessels around which, as spasticity increased, connective tissue elements formed in a ring [16]. This is confirmed by our own duplex scanning results. Blood flow and vessel diameter indicators were found to be inversely correlated with subfascial pressure values. There was a decrease in blood flow in the popliteal artery in patients of Group 1 by 7.2 % (± 2.4); in patients of Group 2 by 12.6 % (± 1.8); in children of Group 3 by 10.5 % (± 2.1).

Triplex scanning of the popliteal vein showed a decrease in its diameter: in Group 1 by 10.3 % (± 1.4); Group 2 by 29.3 % (± 1.7); Group 3 by 25.9 % (± 2.8).

These processes lead to the obliteration of blood vessels, the appearance of fibrotic areas of muscle tissue and the formation of myofascial pain syndrome.

Conclusions

The study has shown that the changes in the muscles and fascia are caused by a blood flow disorder, a change in the subfascial pressure in the muscle-fascial cases of the lower extremities. The development of pain in the lower extremities in children with cerebral palsy is multifactorial in nature and consists of myofascial, joint, and tunnel syndromes.

Conflict of interest. The authors declare no conflict ofinterest.

References

1. Bezsmertnyi, Y., Shevchuk, V., Jiang, Y., Bezsmertna, H., & Bezsmertnyi, O. (2021). Impact of post- amputation pain syndrome on the results of bone stump formation. *Morphologia, 15*(3), 50–56. doi:10.26641/1997-9665.2021.3.50-56
2. Bordoni, B., & Varacallo, M. (2021) Bony pelvis and lower limb, thigh quadriceps muscle. StatPearls. Anatomy
3. Cheverda, A. I., Huk, Y. M., Zyma, A. M., Kincha-Polishchuk, T. M., Syvak, M. F., & Zotia, A. V. (2019). Diagnosis and treatment of contractures of the hip and knee joints in patients with the consequences of open forms of spinal Dysraphism. *Visnyk ortopedii travmatologii protezuvannia, 103*(4), 61–72. doi:10.37647/0132-2486-2019-103-4-61-72
4. Simons, D. G., Travell, J. G., & Simons, L. S. (1999). Travell & Simons' Myofascial pain and dysfunction: Upper half of body. Lippincott Williams & Wilkins.
5. Danilov O., Balitskaya Y., & Motsya M. (2013) Mechanism of formation and clinical course of flexion contractures of knee joints in children with cerebral palsy. *Surgery of paediatric age, 4*, 8–15.
6. Danylov, O., & Shulha, O. (2023). Optimisation of methods of diagnosis and correction of heel foot in children with cerebral palsy. *Paediatric surgery Ukraine, 4*(81), 49–58. doi:10.15574/ps.2023.81.49
7. McDowell, B. C., Duffy, C., & Lundy, C. (2017). Pain report and musculoskeletal impairment in young people with severe forms of cerebral palsy: A population-based series. *Research in developmental disabilities, 60*, 277–284. doi:10.1016/j. ridd.2016.10.006
8. Fishchenko, V., & Obeidat, K. J. (2022). Lower limb muscles functioning in the condition of flexion contracture of the knee joint. *Trauma, 23*(2), 17–24. doi:10.22141/1608-1706.2.23.2022.886
9. Graham, H. K., Rosenbaum, P., Paneth, N., Dan, B., Lin, J., Damiano, D. L., … Lieber, R. L. (2016). Erratum: Cerebral palsy. *Nature reviews disease primers, 2*(1). doi:10.1038/nrdp.2016.5
10. Hauer, J., Houtrow, A. J., Feudtner, C., Klein, S., Klick, J., & Murphy, N. A. (2017). Pain assessment and treatment in children with significant impairment of the central nervous system. *Pediatrics, 139*(6). doi:10.1542/peds.2017-1002
11. de Bruijn, J. A., Wijns, K. C., van Kuijk, S. M., Hoogeveen, A. R., Teijink, J. A., & Scheltinga, M. R. (2021). Chronic exertional compartment syndrome in the differential diagnosis of peripheral artery disease in older patients with exercise-induced lower limb pain. *Journal of vascular surgery, 73*(6), 2114–2121. doi:10.1016/j.jvs.2020.11.027
12. Klochkova, O. A., Kurenkov, A. L., & Kenis, V. M. (2018). Development of contractures in spastic forms of cerebral palsy: Pathogenesis and prevention. *Pediatric traumatology, orthopaedics and reconstructive surgery, 6*(1), 58–66. doi:10.17816/ptors6158-66
13. Komshina, K. (2020). Pain syndrome and comorbidity in adult patients with cerebral palsy. *Saratov journal of medical scientific research, 16* (1).
14. Komshina, K, & Antipenko, E (2020). Evaluation of the features of pain syndrome in adult patients with infantile cerebral palsy. *MA*, 1(62), 7.
15. Lontkovsky, Y. (2016). Treatment of adults and children with organic localised spasticity of the lower extremity muscles. *International neurological journal, 6*, 51–57. doi:10.22141/2224-0713.6.842016.83.
16. Lontkovskyi, Y., Pichkur, L., Vaslovych, V., & Shmeleva G. (2016). Morphological changes of the adductor muscles

of the thigh in local spasticity in patients with cerebral palsy. *Ukrainian neurosurgical journal, 2*, 28–32
17. Mathewson, M, & Lieber R. (2015). Pathophysiology of muscle contractures in cerebral palsy. *Physical medicine and rehabilitation clinics of North America, 26*(1), 57–67.doi: 10.1016/j.pmr.2014.09.005
18. Martyniuk, V., & Nazar, O. (2016). Unified clinical protocol «Cerebral palsy and other organic brain lesions in children, accompanied by movement disorders». Part I. *Sovremennaya pediatriya, 75*(3), 100–105. doi:10.15574/sp.2016.75.100
19. Nenko A., Vlasenko S. (2009). *Diagnstics and treatment neuroorthopedic syndromes at patients by snfantile cerebal palsy. Algorithms choice and tactic of therapy.* Evpatoria: Managements for doctors., 1–152
20. Poirot, I., Laudy, V., Rabilloud, M., Roche, S., Ginhoux, T., Kassaï, B., & Vuillerot, C. (2017). Prevalence of pain in 240 non-ambulatory children with severe cerebral palsy. *Annals of physical and rehabilitation medicine, 60*(6), 371–375. doi:10.1016/j.rehab. 2017.03.011
21. Tondiy, O., Zavalna, O., & Korenev, S. (2016). Tunnel mononeuropathies: Etiology, pathogenesis, clinical manifestations, diaghosis, treatment. *Shidnoevropejskij zurnal vnutrisnoi ta simejnoi medicini*, 2016(1), 68–74. doi:10.15407/internalmed2016.01.068
22. Semkovych, Y., & Dmytriiev, D. (2022). Visual analogue scale as a tool for assessing quality of life (PEDSQL™) and emotional stress in children after anterior abdominal wall surgery. *Pain, anaesthesia & intensive care, 2*(99), 41–48. doi:10.25284/2519-2078.2(99).2022.265838.
23. Strafun, S., Tkach, A., Strafun, A., & Saliy, A. (2014). Risk of local hypertensive ischaemic syndrome in trauma. *Trauma, 15*(3), 5–10. doi:10.22141/1608-1706.3.15.2014.81418.
24. Willard, F. H., Vleeming, A., Schuenke, M. D., Danneels, L., & Schleip, R. (2012). The thoracolumbar fascia: Anatomy, function and clinical considerations. *Journal of anatomy, 221*(6), 507–536. doi:10.1111/j.1469-7580.2012.01511.x
25. Zharikova, A., Krivoshey, O., & Tsukanova, S. (2020). *Tunnel neuropathies of the pelvic girdle and lower extremities.* Gomel: GU «RNPC RMEH», 1–50.
26. Zozulya, I., & Bredikhin, A. (2011). Myofascial pain syndrome: Diagnosis, treatment. *Ukrainian medical magazine, 3*(83)

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CAUSES OF PAIN IN THE MUSCLES OF THE LOWER EXTREMITIES IN CHILDREN WITH CEREBRAL PALSY

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