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Flexion contractures of the knee joints in children with cerebral palsy: pathogenesis, clinical manifestations, anatomical and morphological changes in bones, muscles and intra-articular structures

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The main causes of knee contractures are the dominance of the hamstring muscles, particularly the biceps femoris, as well as associated hip joint contractures, which are combined with torsional deformities of the lower limb bones. Femoral deformity involves anteversion of its proximal epimetaphysis, which arose during ontogenesis and due to the action of external rotators, as well as internal rotation resulting from spasticity of the hip adductor muscles. Pathological external torsion of the tibia is a consequence of a compensatory mechanism aimed at restoring the axis of the lower limb, overcoming spasticity of the biceps femoris muscle, and retraction of the anterior cruciate ligament. Objective. To investigate the pathogenesis of flexion contractures of the knee joints, as well as associated anatomical and structural changes in the bones and primary and secondary stabilizers in children with cerebral palsy. Methods. The analyzed data were obtained during the treatment of 40 patients with cerebral palsy, spastic diplegia, and tetraparesis. In all cases, hip joint contractures were noted, and in most patients, knee joint contractures and foot deformities were observed. Clinical and instrumental diagnostic methods were used. Results. The causes of flexion contractures of the knee joints were studied, theoretically substantiated, and investigated. A direct correlation was established between the degree of contracture and degenerative-dystrophic changes in the intra-articular tissues of the joint, as well as between the degree of torsional deformity of the lower limb bones. Conclusions. The pathogenesis of knee joint contractures is multifactorial in nature and depends on structural changes in the bones of the lower extremities and in the primary and secondary stabilizers of the knee joint, which influences the clinical course of the disease and the future choice of treatment strategy.

Основними причинами контрактур колінних суглобів є домінування м'язів hamstring-групи, і особливо — двоголового м'яза стегна, а також наявність супутніх контрактур кульшового суглоба, які поєднуються з торсійними деформаціями кісток нижніх кінцівок. Деформація стегна включає антеверсію його проксимального епіметафізу, який виник у процесі онтогенезу та дії зовнішніх ротаторів, а також внутрішнього повороту внаслідок спастики привідних м'язів стегна. Патологічна зовнішня торсія великогомілкової кістки є наслідком компенсаторного механізму, який спрямований на відновлення осі нижньої кінцівки, подолання спастики двоголового м'яза стегна та ретракцію передньої схрещеної зв'язки. Мета. Вивчити патогенез згинальних контрактур колінних суглобів, супутніх анатомічних і структурних змін у кістках, первинних та вторинних стабілізаторах у дітей, хворих на ДЦП. Методи. Проаналізовано показники, отримані під час лікування 40 пацієнтів, хворих на ДЦП, зі спастичною диплегією та тетрапарезом. У всіх випадках відзначались контрактури кульшових суглобів та у більшості дітей — контрактури колінних суглобів і деформації стоп. Застосовувалися клінічні й інструментальні методики діагностики. Результати. Вивчені, теоретично обгрунтовані та досліджені причини виникнення згинальних контрактур колінних суглобів. Встановлено пряму залежність між ступенем контрактури та дегенеративно-дистрофічними змінами в інтраартикулярних структурах суглоба, а також — між ступенем торсійної деформації кісток нижньої кінцівки. Висновки. Механогенез контрактури колінного суглоба має мультифакторну природу виникнення та залежить від структурних змін у кістках нижніх кінцівок, первинних і вторинних стабілізаторах колінного суглоба, що впливає на клінічний перебіг хвороби та майбутній вибір тактики лікування.

Ключові слова. Діти, церебральний параліч, контрактури суглобів, торсійні деформації кісток, дегенеративно-дистрофічні зміни в м'язово-зв'язковій системі, математичне моделювання, больовий синдром.

Key words. Children, cerebral palsy, joint contractures, torsional bone deformities, degenerative-dystrophic changes in the musculoskeletal system, mathematical modeling, pain syndrome

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PREVIEW

Introduction

Flexion contractures of the knee joint are observed in 47–53% of patients with cerebral palsy (CP) and represent an integral component of the pathological process [1–4]. The main cause of this deformity, according to most authors, is muscle imbalance resulting from impaired reciprocal innervation and the pathological effect of co-contraction [5, 6]. Under these conditions, there is a predominance of the hamstring muscles over the quadriceps, as well as of the hip adductors over the abductors [7]. However, the myofascial relationship between these muscle groups in the development of knee joint contracture remains insufficiently studied.

Joint stability depends on the interaction between primary and secondary stabilizers, namely muscles and the ligamentous system [8]. However, joint malposition disrupts gait biomechanics and posture. Load distribution changes across the femoral condyles, tibial plateau, and intra-articular structures. Therefore, the pattern of pathological changes in these regions in children with CP differs from traumatic injuries, which result from the application of a large amount of kinetic energy over a short period of time [9–13]. In flexion contractures caused by muscle imbalance, degenerative and dystrophic changes in muscles and in the anterior and medial knee complexes depend on the degree of spasticity and the duration of the pathological process, which develops gradually.

Hip and knee flexion contractures are well established to be accompanied by hip adduction, valgus deviation, and external tibial rotation [6, 14]. However, the role of associated deformities and degenerative-dystrophic changes in muscles and the ligamentous system in the development of contractures remains controversial. It should be noted that most authors identify muscle imbalance as the primary cause of flexion contracture, but they do not account for changes in the ligamentous system, nor for the comparative mass of agonist and antagonist muscles, the magnitude of their moment arms, and the direction of force vectors.

According to recent studies, pain syndrome is one of the contributing factors in the development of knee flexion contractures [15]. However, its relationship with the degree of contracture and changes in primary and secondary knee stabilizers remains insufficiently investigated.

One component in the development of knee flexion contractures is internal torsion of the distal femur and compensatory external torsion of the tibia, which disrupt the anatomical axis of the limb and the congruence of joint surfaces. This creates a pathological cycle that further limits joint mobility [16, 17]. Therefore, in the treatment of knee flexion contractures, some authors consider correction of torsional deformities of the bones to be necessary [18]. At the same time, the mechanisms underlying torsional deformities remain controversial, leading to differing approaches in defining indications and treatment strategies.

Thus, the mechanisms underlying the development of knee flexion contractures, structural changes in muscles and intra-articular tissues, and torsional deformities of the bones of the lower extremities require further investigation.

Purpose: to investigate the mechanisms of development of knee flexion contractures, associated anatomical and structural changes in bones, and alterations in primary and secondary stabilizers in children with cerebral palsy.

Materials and Methods

The study involved examination during treatment of a total of 40 patients with cerebral palsy, including spastic diplegia and tetraparesis. All patients presented with hip contractures, and the majority also had restricted knee joint mobility and foot deformities.

The study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the local ethics committee of the State Institution “All-Ukrainian Center for Motherhood and Childhood of the National Academy of Medical Sciences of Ukraine” (Protocol No. 12 dated 27.03.2026), and informed consent was obtained from all patients.

The patients were divided into three groups depending on muscle tone impairment: Group 1 — 14 children aged 4–7 years with reflex tonic hypertonia; Group 2 — 13 patients aged 8–12 years with spasticity; Group 3 — 13 patients aged 13–17 years with rigidity.

Pain syndrome assessment was performed considering the etiological causes of pain (myofascial, neurogenic, arthralgic) using the Numerical Rating Scale (NRS).

Depending on the range of motion in the knee joints, four degrees of contracture were defined (Table 1).

The femoral neck anteversion angle was analyzed [14]. The external tibial torsion angle was calculated on radiographs using a developed method [18]. The anatomical axis was assessed on frontal long-leg radiographs of the lower extremities.

PREVIEW

The force distribution acting on intra-articular knee structures, hip adductors, and hamstring muscles was analyzed in the frontal and sagittal planes.

Ultrasound examination (US) was used to evaluate the degree of structural changes in muscles and intra-articular tissues, as well as the cross-sectional size of muscles in the middle third of the thigh.

To assess changes in muscle mass in children with cerebral palsy, we developed a muscle mass index, calculated using the formula:

$$MMI = ML / (MCSA + FCSA),$$

where ML — muscle length, MCSA — muscle cross-sectional area, FCSA — fascia cross-sectional area. A decrease in the muscle mass index indicates an increase in cross-sectional area [11].

The study included patients with cerebral palsy and knee joint flexion contractures. Children without neurological pathology and those with post-traumatic or congenital contractures were excluded.

Results

Mechanism of knee joint contracture formation

We considered the knee joint as a stabilization center located between the ankle and hip joints. Myofascial structures are organized as antagonistic pairs on both sides of the lower limb. When one group is shortened (concentric loading), the opposing group is subjected to tension, resulting in eccentric loading of muscles and fascia in a lengthened state. The development of degenerative-dystrophic changes leads to loss of elasticity of the myofascial structures along the posterior kinetic chain [7]. Increased tension of the sciatic nerve in the proximal thigh and of the common peroneal and tibial nerves in the popliteal region leads to the development of spastic-ischemic traction neuropathies [15]. Clinical manifestations are associated both with neurogenic factors and with degenerative-dystrophic changes occurring in the muscles and ligamentous system of the knee joint.

Another key factor in contracture development is increased physiological motor neuron output to the flexor and adductor muscle groups of the lower limb, which subsequently leads to increased spasticity. A second important factor is the biomechanical distribution of muscle forces acting on the femur and tibia. The *quadriceps femoris* is the largest muscle; however, its mechanical effect is lower than the combined action of the knee flexors. The main reason for this discrepancy is the difference in moment arms acting on the tibia and their number. The moment arm of the *quadriceps femoris muscle* is 21.5 % of tibial length. At the same time, part of the tendon of the *biceps femoris muscle* (force arm, accounting for 43.2 % of tibial length) directly continues into the crural fascia, thereby increasing the moment arm of this muscle. The *quadriceps femoris* has a single force vector, whereas the knee flexors have multiple vectors. In the presence of impaired muscle tone, all these factors lead to a functional predominance of flexors over extensors.

Analysis of the influence of the muscular component on knee flexion contracture formation indicates the need to consider the direction of the force vector of its individual elements. Thus, part of the tendon of the long head of the biceps femoris attaches to the head of the fibula, while another, more distal portion, attaches to the crural fascia. Given the greater force of the biceps femoris compared with the semimembranosus–semitendinosus group, the resulting moment acting on the lateral aspect of the tibia becomes dominant and enhances external tibial rotation (and, in the long term, torsion).

This alters the projected positions of the fixation points of the *semimembranosus–semitendinosus muscles*, increases their kinematic chain length, and creates a pathological cycle in the development of knee flexion contracture.

There is also a direct fascial connection between the long head of the biceps femoris and the fibularis longus muscle, which contributes to a predominance of foot pronators (Fig. 1). Fascial continuity between

Table 1

Degree of knee joint contracture depending on the range of active and passive movements

Grade	Extension position			Number of patients
	supine		standing	
	active	passive	active	
I	158° ± 2.0°	169° ± 3.0°	148° ± 2.6°	13
II	149° ± 1.6°	157° ± 2.5°	127° ± 3.0°	9
III	137° ± 2.4°	149° ± 3.0°	118° ± 2.5°	12
IV	108° ± 2.0°	116° ± 3.5°	100° ± 3.2°	3

PREVIEW

the long head of the biceps femoris and the fibularis longus leads to foot eversion with a tendency toward flattening of the medial longitudinal arch.

The short head of the biceps femoris attaches to the head of the fibula and has fascial continuity with the middle portion of the adductor magnus muscle, which passes beneath the main bulk of the biceps femoris (Fig. 1). This fascial connection increases the overall force of the adductors, which have a large attachment area along the *linea aspera* (adductor brevis, adductor longus, and adductor magnus), thereby enhancing their dominance.

Thus, loss of elasticity in the hamstring muscles and pain syndrome are key elements in the development of flexion posture in the knee joints. Excessive activity of the biceps femoris muscle, which is considered the primary factor in knee flexion contracture formation, also leads to increased torsional deformity of the tibia, alteration of its axis, and valgus positioning of the foot. The short head of this muscle, in addition to its primary function, also contributes to the development of valgus deformity of the knee joint.

Antagonistic myofascial structures of the anterior and posterior muscle groups of the lower limb normally stabilize the knee joint in the sagittal plane, which is confirmed by mathematical modeling (Fig. 2a, b).

The distance KN (segment between the fixation points of the biceps femoris muscle) is calculated using the cosine theorem with F = 10 N, α = 10°, β = 180°–10° = 170°, ON = 50 (tibial length), OB = 60 (femoral length), where ON is the tibial length (50), and OK is the force vector of the biceps femoris muscle.

$$\text{ONK} : KN^2 = F^2 + ON^2 + 2F \times ON \times \cos\beta;$$
$$KN^2 = 10^2 + 50^2 - 2 \times 10 \times 50 \times \cos 170°;$$
$$KN^2 = 100 + 2500 - 1000 \times (-0,9848);$$
$$KN^2 = 2600 + 984,8;$$
$$KN^2 = 3584,8.$$

The results of the study demonstrate that the long moment arm of the biceps femoris muscle is balanced by the force of the quadriceps femoris muscle, which act as antagonistic myofascial structures and allow the knee joint to be maintained in a neutral position.

The flexor and adductor muscles of the lower limb in children with cerebral palsy demonstrate a predominance over their antagonists [18]. Trunk and pelvic inclination leads to eccentric loading of the biceps femoris and gastrocnemius muscles, the tension of which is compensated by knee flexion, which is related to the anatomical characteristics of biarticular muscles. The approximation of the fixation points of the biceps femoris, from the ischial tuberosity to the tibia, reduces tension in the posterior thigh muscle group, thereby decreasing pain syndrome, which is one of the leading factors in the development of pathological movement patterns in the hip and knee joints (Fig. 3a–c).

We calculate the distance KN (distance between the fixation points of the biceps femoris muscle) using the cosine theorem, where ON = 50 (tibial length), OB = 60 (femoral length), and OK is the force vector of the biceps femoris muscle. We demonstrate that when the force of the hamstring muscles increases (F = 20 N, ON = 50, α = 10°), KN^2 = 3584.8, and the angle β decreases.

The angle β of triangle ONK is determined using the cosine theorem.

$$KN^2 = F2 + ON^2 - 2 \times F \times ON \times \cos\beta;$$
$$3584,8 = 20^2 + 50^2 - 2 \times 20 \times 50 \times \cos\beta;$$
$$3584,8 = 400 + 2500 - 1000 \times \cos\beta;$$
$$\cos\beta = -0,6848;$$
$$\cos\beta = \arccos(-0,6848) = 37°.$$

The obtained results indicate that with an increase in force, the angle β decreases and equals: 180° – 10° – 37° = 133°. The mathematical model demonstrates that as the force of the biceps femoris increases, due to synergy with the peroneus longus muscle, knee flexion contracture progresses.

In the upright position, most adductor muscles (adductor longus, adductor brevis, adductor magnus, gracilis, and pectineus) are balanced in their action with the hip abductors. In addition to adduction, in the neutral position of the knee joint the adductors also contribute to external rotation of the femur (Fig. 4a, b).

We calculate the distance CB (distance between the ischial tuberosity and the medial malleolus) using the cosine theorem, where F = 10 N, α = 10°, β = 175°, OB = 50 (tibial length), OA = 60 (femoral length), and OC is the force vector of the adductor magnus muscle.

$$\text{OBC, angle } \Omega = \text{angle BOC} = 360° - 175° - 10° = 175°;$$
$$CB^2 = OC^2 + OB^2 - 2OC \times OB \times \cos\Omega;$$
$$50^2 + 10^2 - 2 \times 50 \times 10 \times \cos 175° = 2600 - 1000 \times$$
$$\times (-0,9962) = 3596,2.$$

The results demonstrate that the long moment arm of the adductor magnus is balanced by the force of the hip abductors, which act as antagonistic myofascial structures and allow the hip joint to be maintained in a neutral position.

PREVIEW

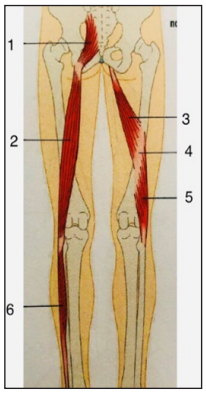


Fig. 1. Myofascial continuity of the heads of the *biceps femoris muscle*: 1 — *sacrotuberous ligament*; 2 — long head of the *biceps femoris muscle*; 3 — middle portion of the *adductor magnus muscle*; 4 — *linea aspera*; 5 — short head of the *biceps femoris muscle*; 6 — *fibularis longus muscle*

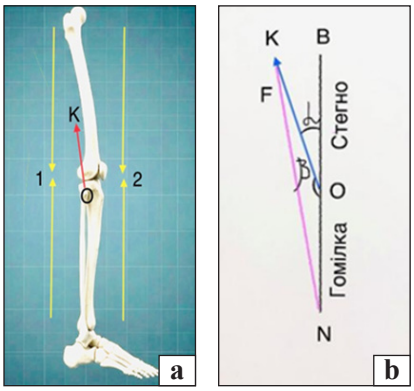


Fig. 2. Model of the lower limb in the sagittal plane under neutral hip and knee joint positioning: (a) schematic model: 1 — stabilizing forces of flexors; 2 — stabilizing forces of extensors; OK — force vector of the biceps femoris muscle; (b) mathematical model

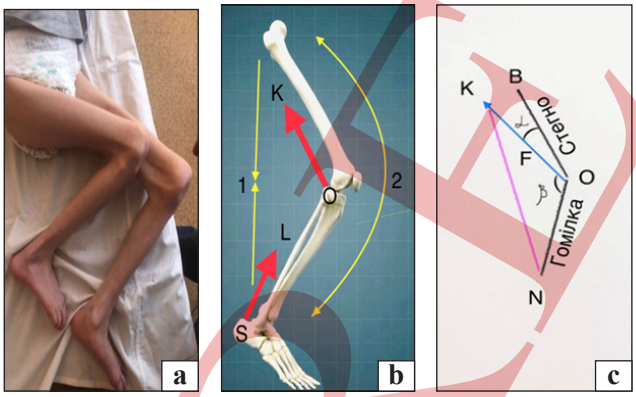


Fig. 3. Lower limb in knee flexion contracture: (a) a 10-year-old patient V., diagnosis: cerebral palsy, hip flexion-adduction contractures grade III, knee flexion contractures grade III; (b) schematic model: 1 — concentric loading of flexors; 2 — eccentric loading of extensors; OK — force vector of the *biceps femoris muscle*; SL — force vector of the *gastrocnemius muscle*; (c) mathematical model

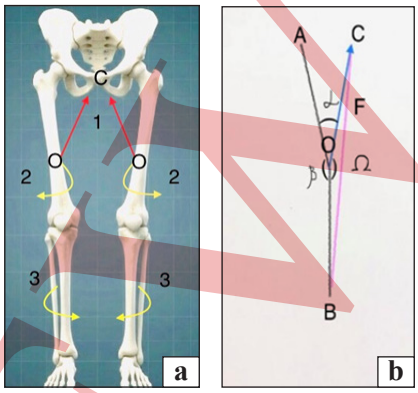


Fig. 4. Model of the lower limb in the frontal plane under neutral hip and knee joint positioning: (a) schematic model: 1 — hip adductors; 2 — external rotation of the femur; 3 — internal rotation of the tibia; OC — force vector of the *adductor magnus muscle*; (b) mathematical model

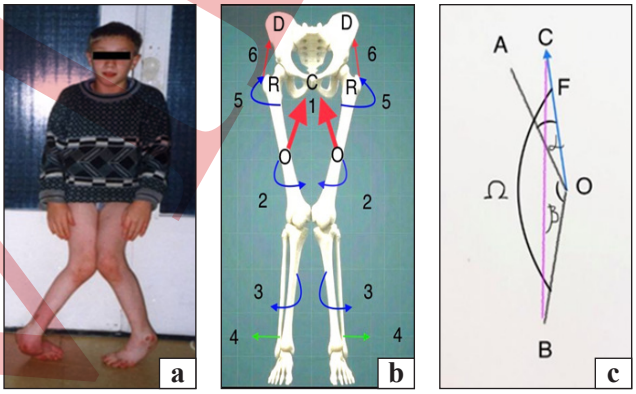


Fig. 5. Valgus deformity of the knee joint: (a) a 12-year-old patient D.; diagnosis: cerebral palsy, hip flexion-adduction contractures grade II, valgus deformity and knee flexion contractures grade II, equinovarus foot deformity; (b) schematic model: 1 — OC — force vector of hip adductors; 2 — internal torsion of the distal femur; 3 — external rotation of the tibia; 4 — lateral deviation of the tibia; 5 — external torsion of the proximal femur; 6 — RD — force vector of hip external rotators; (c) mathematical model

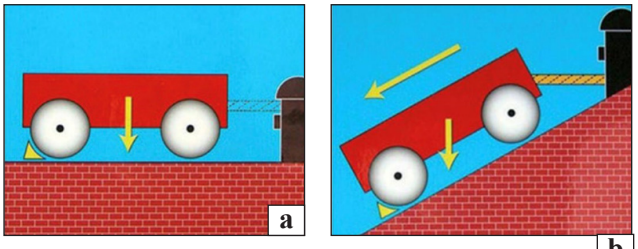


Fig. 6. Loading simulation of the anterior cruciate ligament: (a) unloaded condition; (b) continuous loading simulation due to tibial plateau inclination

In children with cerebral palsy, an increase in the total force of the hip adductor muscles (adductor longus, adductor brevis, adductor magnus, gracilis, and pectineus) is observed due to spasticity and continuous fascial continuity with the short head of the biceps femoris, which leads to increased predominance of these muscles over the hip abductors. In hip flexion, the direction of their tension vector changes. In this condition, the hip adductors, in addition to their primary function, act as internal rotators, which subsequently leads to internal torsion of the distal segment. At the same time, external

PREVIEW

rotators (gluteus maximus, piriformis, obturator muscles, and gemelli) contribute to excessive femoral anteversion. A compensatory mechanism is excessive external tibial torsion (Fig. 5a–c).

We calculate the distance CB (segment between the ischial tuberosity and the lateral malleolus). We demonstrate that with an increase in force (F = 12 N, tibial length OB = 50, femoral length OA = 60, OC as the force vector of the adductor magnus muscle, $\alpha = 10°$), $CB^2 = 3596.2$, and angle β decreases (Fig. 5).

The angle Ω of triangle OBC is determined using the cosine theorem.

$$CB^2 = OC^2 + OB^2 - 2 \times OC \times OB \times \cos \Omega;$$
$$3596{,}2 = 122 + 50^2 - 2 \times 12 \times 50 \times \cos \Omega;$$
$$-1200 \times \cos \Omega = 1088{,}56;$$
$$\cos \Omega = -0{,}9071;$$
$$\Omega = \arccos(-0{,}9071) = 155°;$$
$$\beta = \Omega - \alpha = 155° - 10° = 145°.$$

Thus, with increasing force, angle β decreases and equals $\beta = \Omega - \alpha = 155° - 10° = 145°$. Our findings demonstrate that an increase in the force of the adductor magnus muscle, in synergy with the short head of the biceps femoris, leads to an increase in valgus deformity of the knee joint, as well as internal rotation of the femur (and subsequently torsional deformation of its distal segment relative to the proximal segment). To preserve gait function and prevent scissoring of the limbs, a valgus deviation of the tibia develops.

In hamstring syndrome, patients present with a flexed posture at the hip joints, internal rotation of the femurs, and flexed knee positioning (Fig. 8). The development of pathological patterns in the hip and knee joints results in excessive loading of the primary stabilizers. Changes in the angle between the femur and tibia in both the frontal and sagittal planes create constant tension in the anterior cruciate ligament and the patellar ligament (Fig. 6a, b).

The anterior cruciate ligament plays a key role in stabilizing the knee joint in the sagittal plane [22]. Degenerative-dystrophic changes lead to reduced mechanical strength and reflex overloading of the patellar ligament, medial collateral ligament, lateral meniscus, and medial meniscus (Fig. 7a, b).

At the same time, in the case of injury to the anterior cruciate ligament (*lig. cruciatum anterius*), pathological mobility of the tibia develops in the sagittal plane, with its appearance in the frontal plane. In conditions of high spasticity of the hip adductor muscles, an adductor syndrome develops, in which the child stands and walks with the thighs adducted to the point of full contact or crossing at the level of the middle and distal thirds of the thighs, knee joints, and legs. This results in compensatory valgus deviation of the lower legs at the knee joints (Fig. 5a).

Increased loading on the medial knee complex leads to its damage (Fig. 8a, b). It consists of the superficial medial collateral ligament (*lig. tibiale collaterale superficiale*) and the deep medial collateral ligament (*lig. collaterale profundum*), which are the main stabilizers of the knee joint under forces directed laterally to the tibia (*valgus loading*). The medial complex also includes the oblique popliteal ligament (*lig. popliteum obliquum*), which is responsible for rotational stability of the knee joint.

Progression of the force of the hamstring muscles and hip adductors leads not only to damage of the frontal and medial knee complexes but also to degenerative changes in the menisci. Overloading of the deep collateral ligament (*lig. collaterale profundum*), which is connected to the medial meniscus and consists of meniscofemoral and meniscotibial components, occurs. Considering the anatomical relationship between the meniscus and the ligament, valgus angulation of the knee joint induces structural changes in the deep collateral ligament and the medial meniscus. At the same time, compressive loading of the lateral femoral condyle on the lateral meniscus increases (Fig. 8a).

The superficial medial collateral ligament has proximal and distal portions. Mathematical modeling demonstrates that with increasing force of the hip adductors, valgus deformity of the knee joint increases. Therefore, the greatest load is applied to the distal portion of the ligament, resulting in its injury. This leads to shortening of the obliquely oriented superficial medial collateral ligament (*pars proximalis*) and the oblique popliteal ligament, which slows femoral retrotorsion and enhances compensatory external torsion of the tibia (Fig. 9).

During physiological growth of the lower limb, both the femur and tibia undergo external rotation. The anatomy of the bones and the degree of femoral internal rotation along its longitudinal axis are variable and age-dependent. Gradually, the femoral neck anteversion angle decreases, while external tibial torsion increases with growth. The angle between the femoral neck and femoral condyles at the age of 4–7 years ranges from 31° to 43°; at 8–12 years it decreases to 21°–25°; and at 13–17 years it varies from 10° to 14°. The external tibial torsion angle in patients aged 4–7 years ranges from 0° to 5°; at 8–12 years it increases to 8°–12°; and at 13–17 years it reaches 14°–18° [18, 19].

PREVIEW

In children with cerebral palsy, increased torsional deformity of the femur results from incomplete axial rotation between its proximal and distal ends due to impaired muscle tone [19]. External rotators increase femoral neck anteversion, while hip adductor muscles cause internal torsion of the distal femoral segments, producing twisting of the bone in the vertical plane. External tibial torsion increases both as a compensatory mechanism aimed at restoring limb alignment and due to the pathological influence of primary and secondary stabilizers.

Thus, knee flexion contracture has a multifactorial origin, including impaired interaction between primary and secondary stabilizers, structural changes in muscles and intra-articular tissues, pain syndrome, and torsional deformities of the bones, all of which represent key elements in the pathogenesis of this deformity.

Analysis of clinical observations showed that in Group 1 (14 patients aged 4–7 years), all patients had hip flexion-adduction contractures of grade I. In 11 patients (78.6 %), flexed knee positioning in the upright posture was observed, which may subsequently progress to flexion contractures. The mean body weight in this group was 19.2 kg.

In this group, pain associated with physical activity was localized along the posterior thigh and calf region and was observed in 4 patients (28.6 %).

In all patients of Group 1, ultrasound findings demonstrated signs of long-standing retraction of the biceps femoris muscle, including thickening of muscle fibers and an increase in their volume, in contrast to the stretched and thinned fibers of the quadriceps femoris muscle, in which structural integrity was preserved. A decrease in the muscle mass index indicates an increase in cross-sectional area. Thus, the muscle mass index of the biceps femoris was 1.6 ± 0.1 (reference value 1.7 ± 0.2), and it decreased relative to the quadriceps femoris, which was 1.73 ± 0.3 (reference value 1.60 ± 0.5). In 5 patients (35.7%), isolated degenerative-dystrophic areas of the biceps femoris were also identified. Due to only partial loss of elasticity and function of the muscles, a high gradient between active and passive range of motion in the knee joints was maintained (20.1 ± 0.5). Sonographic evaluation of the knee joints revealed moderate synovitis and loosening of the anterior cruciate ligament structure in 6 patients (42.9 %), while 4 patients (28.6 %) also had injury to the medial complex. Only 1 patient had involvement of the medial meniscus.

There were no signs of malalignment of the lower limb or torsional deformity of the tibia. The femoral neck anteversion angle was within the age norm (35° ± 4.0°).

In Group 2 (13 patients aged 8–12 years), knee flexion contractures of grade I were observed in 2 children (15.4 %), grade II in 7 (53.8 %), and grade III in 4 (30.8 %). The mean body weight in this group was 31.5 kg. Pain syndrome was present in 8 patients (61.5 %).

According to ultrasound findings, the quadriceps femoris showed a marked increase in areas of fiber disorganization, with the appearance of microtears, in contrast to the biceps femoris, in which the fibers remained thickened with focal areas of connective tissue within its structure. The muscle mass index of the biceps femoris was 1.4 ± 0.1, whereas that of the quadriceps increased to 1.80 ± 0.4. Loss of muscle elasticity led to a reduction in the gradient between active and passive knee joint motion (12.5 ± 1.3).

In 10 patients (76.9 %), degenerative-dystrophic changes of the patellar ligament and partial injury of the anterior cruciate ligament were observed. In 7 patients (53.8 %), these injuries were combined with partial rupture of the medial collateral ligament fibers. In 4 patients (30.8 %), injury of the medial meniscus was noted, and in 1 patient, injury of the lateral meniscus.

Moderate signs of lower limb malalignment in the frontal plane were observed. External tibial torsion exceeded physiological values, reaching 11.5° ± 2.5°. The femoral neck anteversion angle was 29° ± 3.0°.

In Group 3 (13 patients aged 13–17 years), muscle tone impairment manifested as rigidity. Knee flexion contractures of grade III–IV were observed in the majority of patients; only 2 patients (15.4 %) had grade II contractures. The mean body weight was 54.5 kg. Pain syndrome was present in only 3 children (23.1 %).

Ultrasound examination of the biceps femoris and quadriceps femoris muscles demonstrated degenerative-dystrophic changes in muscle fibers, with multiple foci of connective tissue. The muscle mass index was similar in both muscles (1.85 ± 0.3 and 1.82 ± 0.4, respectively). A near-complete loss of muscle elasticity was observed, with a significant reduction in the gradient between active and passive knee joint motion (8.5 ± 2.0).

Analysis of primary stabilizers revealed damage to the frontal and medial knee complexes in 12 patients (92.3 %), including microtears and areas of scar tissue formation. In 7 patients (53.8%), degenerative-dystrophic changes of the menisci were observed.

PREVIEW

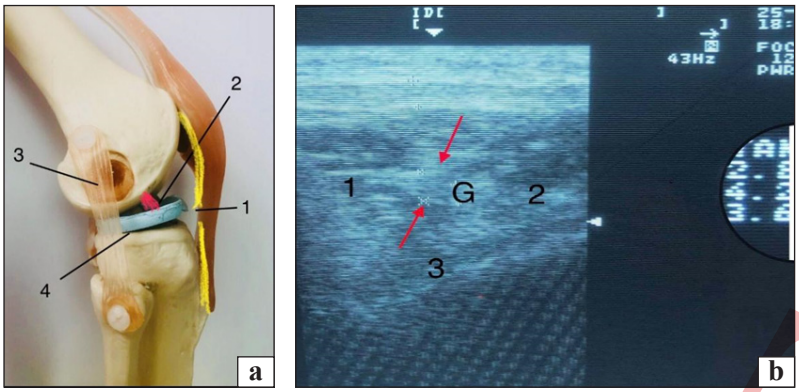


Fig. 7. Model of the right knee joint in the sagittal plane and ultrasonography: (a) model with damage to the anterior knee complex: 1 — *patellar ligament*; 2 — *anterior cruciate ligament*; 3 — *fibular collateral ligament*; 4 — *lateral meniscus*; (b) ultrasonography (patient V., 12 years old; diagnosis: cerebral palsy, knee flexion contracture grade II). 1 — tibial portion of the *anterior cruciate ligament* (Ø — 2.8); G — area of hypoechoic disorganization with internal fiber rupture (Ø — 4.2); 2 — femoral portion of the *anterior cruciate ligament* (Ø — 3.6); 3 — *tibial plateau*

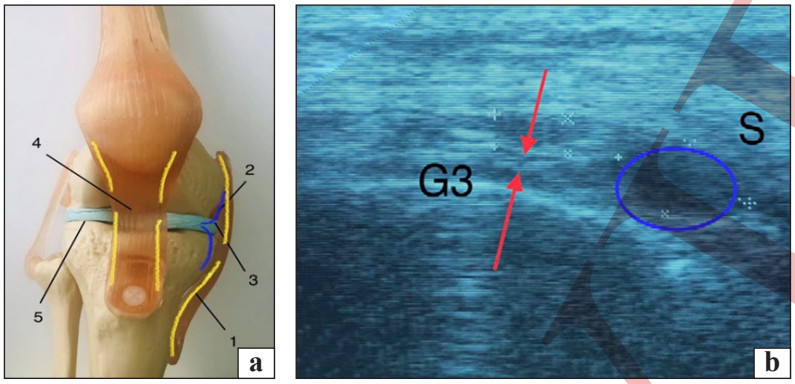


Fig. 8. Model of the right knee joint in the frontal plane and ultrasonography: (a) model with damage to the medial knee complex: 1 — superficial *medial collateral ligament*; 2 — deep *collateral ligament*; 3 — *medial meniscus*; 4 — *patellar ligament*; 5 — lateral meniscus; (b) ultrasonography (patient D., 12 years old; diagnosis: cerebral palsy, valgus deformity and knee flexion contractures grade II); G3 — superficial medial collateral ligament with internal fiber rupture; S — signs of chronic exudative synovitis

Alterations in the anatomical axis of the lower limb in the frontal plane were noted, with lateral deviation of the first interdigital space. External tibial torsion exceeded physiological values, reaching 20.5° ± 1.3°. The femoral neck anteversion angle reached 24° ± 2.0°.

A direct proportional relationship was observed between degenerative-dystrophic changes in primary stabilizers and the degree of knee joint contracture (Fig. 10).

Discussion

A. Mustafaeva et al., noting the high incidence of knee flexion contractures (47–58 %) in patients with cerebral palsy, state that a 15° deformity negatively affects not only the statics and kinematics of gait but also the function of all joints of the lower extremities [20]. According to recent publications, grade I–II deformities are accompanied by pain syndrome associated with impaired microcirculation, altered subfascial pressure, and degenerative-dystrophic changes in myofascial structures [7, 15]. This is confirmed by our findings. Thus, in patients with muscle tone impairment in the form of reflex hypertonicity, pain syndrome was observed in 28.6 % of cases, while in spasticity it was 61.5 %. In contrast, in patients with rigidity, the lowest level of pain was recorded (15.4 %). This is explained by progression of the contracture severity and significant de-

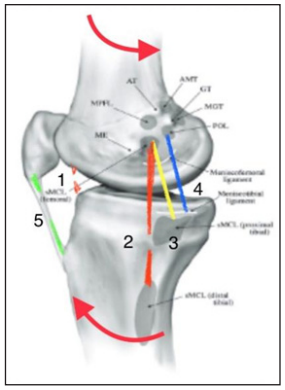


Fig. 9. Development of external tibial torsion in case of damage to the medial and anterior knee complexes: 1 — *anterior cruciate ligament*; 2 — *superficial medial collateral ligament* (distal part); 3 — *superficial medial collateral ligament* (proximal part); 4 — *oblique popliteal ligament*; 5 — *patellar ligament*

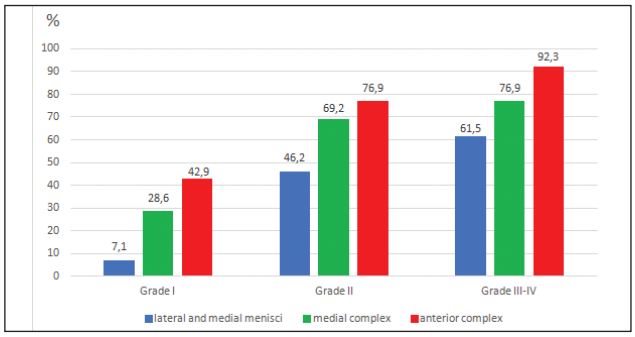


Fig. 10. Relationship between degenerative-dystrophic changes in the ligamentous system of the knee joint and the degree of contracture

PREVIEW

generative-dystrophic changes in both primary and secondary stabilizers of the knee joint. Accordingly, determining the causes of pain syndrome and its management remains one of the main and most complex challenges.

E. Pantzar-Castilla, C. Martinez et al. note that the main cause of contracture formation is muscle imbalance with predominance of knee flexor strength, particularly the hamstring group [21, 22]. I. Levaj et al. and H. Sachn also emphasize the importance of quadriceps femoris weakness, resulting in altered patellar positioning [1, 23]. In our view, pathological muscle synergies also contribute to contracture development. Thus, the myofascial connection between the peroneus longus muscle and the long head of the biceps femoris creates a functional predominance of the latter over the quadriceps femoris. The myofascial interaction between the short head of the biceps femoris and the adductor magnus forms a synergistic link that prevails over the hip abductors.

E. Cloodt et al. report that the fixed joint position depends on muscle length both in the initial phase and after pathological retraction [24]. In our opinion, not only the magnitude of the muscular kinematic chain and muscle mass affect contracture formation, but also the magnitude of the moment arm and the number of attachment points. The quadriceps femoris is the largest muscle of the lower limb in terms of mass; however, due to having only one fixation point and thus a single moment arm, its mechanical influence on the tibia is lower compared to the flexor muscle group. Assessment of the cross-sectional area of the flexor muscles demonstrates the predominant role of the biceps femoris (muscle mass index). The obtained data correspond to clinical findings and mathematical modeling results.

S. Herasymenko et al., studying knee joint biomechanics in flexion contractures ≥ 30°, report disruption of contact between the tibial articular surface and femoral condyles. A fixed position of the articular surfaces leads to retraction not only of muscles but also of the posterior joint capsule [25]. These changes are key factors in contracture formation and progression, as also noted by C. Martinez et al. [21]. Our ultrasound assessment of intra-articular structures demonstrated deep degenerative-dystrophic changes not only in the joint capsule but also in ligaments, menisci, and Hoffa's fat pad. It has been shown that with increasing strength of the hip flexors and adductors in children with cerebral palsy, the influence of retracted anterior cruciate and oblique popliteal ligaments increases in the development of contracture and pathological external tibial torsion. In our opinion, these ligamentous changes should be considered in prognostic assessment and in selecting treatment strategies.

F. Biligli et al. suggest that knee flexion contracture formation is closely associated with hip flexion-adduction contractures, anterior pelvic tilt, increased lumbar lordosis, and femoral neck anteversion [26]. According to our findings, deformity most often develops in a stepwise manner: first in the hip joint, then in the knee, and subsequently in the ankle joint, consistent with the "joint-by-joint" concept.

E. Ciccodicalo, P.T. Thomason et al. note that pathological external torsion of the tibia results from compensatory positioning in response to excessive femoral neck anteversion [27–29]. Our observations confirm the influence of these factors on the development of external tibial torsion and progression of knee contracture. Femoral torsion occurs as a twisting deformation with external rotation of the proximal segment and internal rotation of the distal segment, leading to compensatory external positioning of the tibia. The biceps femoris muscle also contributes to tibial torsional deformity, acting not only as a flexor but also as a physiological external rotator.

Conclusions

The mechanogenesis of knee joint contracture has a multiplanar pattern of development and depends on multiple factors:

– muscle imbalance characterized by dominance of the adductor and hamstring muscle groups due to a larger moment arm, a greater number of attachment sites, and pathological synergies;

– pain syndrome (myofascial, neurogenic, and arthralgic pain), which leads to compensatory flexion postures of the knee joints with subsequent transformation into contractures;

– altered loading of the tibial plateau, primarily affecting the frontal and medial knee complexes, resulting in overload and degenerative-dystrophic changes in intra-articular structures;

– complex torsional deformity involving multiple segments of the lower limb, including excessive femoral neck anteversion, internal torsion of the distal femur, combined with pathological external torsion of the tibia;

– dependence of the severity of knee contracture on age, duration of the pathological process, and body weight in children with cerebral palsy.

Conflict of Interest. The authors declare no conflicts of interest.

Future Research. Future studies are planned to analyze the effectiveness of knee flexion contracture treatment with consideration of the underlying pathomorphological mechanisms.

PREVIEW

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Author Contributions. Danylov O. A. — study conception and objectives, drafting conclusions, overall supervision; Shulha O. V. — data collection and drafting of the main manuscript; Horelik V. V. — analysis of study materials and compilation of the reference list.

References

1. Bohaček, I., Đapić, T., Levaj, I. & Delimar, D. (2023). Innovative method to treat patella alta in a patient with spastic cerebral palsy: a case report with literature review. *Acta Clinica Croatica, 62*(l3), 115–120. https://doi: 10.20471/acc.2023.62.s3.15.
2. Chin, E. M., Gwynn, H. E., Robinson, S., & Hoon, A. H. (2020). Principles of medical and surgical treatment of cerebral palsy. *Neurologic clinics, 38*(2), 397–416. https://doi.org/10.1016/j.ncl.2020.01.009
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. https://doi.org/10.37647/0132-2486-2019-103-4-61-72
4. Marron, A., Milnes, J., Conry, L., & Kiernan, D. (2025). Lower limb contracture definitions in children and adults with cerebral palsy: *A systematic review. Gait & Posture,* 120, 1–8. https://doi.org/10.1016/j.gaitpost.2025.03.027
5. Bezsmertnyi, Y., Branitskyi, O., Tyazhelov, O., & Karpinska, O. (2023). Modeling the work of the muscles of the lower extremity in conditions of flexion-adduction contracture of the hip joint and flexion-extension contracture of the knee joint. *Orthopaedics, traumatology and prosthetics,* (1), 55–60. https://doi.org/10.15674/0030-59872023155-60.
6. Fishchenko, V., & Obeidat, K. J. (2022). The work of the muscles of the lower limb in the case of flexion contracture of the knee joint. *Trauma, 23*(2), 17–24. https://doi.org/10.22141/1608-1706.2.23.2022.886
7. Danilov, O., & Shulga, O. (2025). Treatment of spasticity and degenerative-dystrophic changes in muscles in children with cerebral palsy. *Neonatology, surgery and perinatal medicine, 15*(2(56)), 86–94. https://doi.org/10.24061/2413-4260.xv.2.56.2025.12
8. Modlesky, C. M., & Zhang, C. (2020). Complicated muscle-bone interactions in children with cerebral palsy. *Current osteoporosis reports, 18*(1), 47–56. https://doi.org/10.1007/s11914-020-00561-y
9. Herbst, E., Glasbrenner, J., Deichsel, A., Briese, T., Peez, C., Raschke, M. J., & Kittl, C. (2023). Verletzungen des medialen Bandapparats des Kniegelenks. *Die unfallchirurgie, 127*(1), 18–26. https://doi.org/10.1007/s00113-023-01368-z
10. Kostrub, O. O., Kotiuk, V. V., Mazevych, V. B., Nikiforova, I. M., & Smirnov, D. O. (2019). MRI diagnosis of the anterior cruciate ligament damages. Modern approaches (Overview). *Visnyk ortopedii travmatologii protezuvannia, 103*(4), 104–109. https://doi.org/10.37647/0132-2486-2019-103-4-104-109
11. Meyer, P., Reiter, A., Akoto, R., Steadman, J., Pagenstert, G., Frosch, K. H., & Krause, M. (2021). Imaging of the medial collateral ligament of the knee: A systematic review. *Archives of orthopaedic and trauma surgery, 142*(12), 3721–3736. https://doi.org/10.1007/s00402-021-04200-8
12. Rokita, T. G. & Rokita, R. T. (2024). Acute injury to the medial collateral ligament of the knee: a case report. *Public health journal, 1*(5), 127–133. https://doi.org/10.32782/pub.health.2024.1.18
13. Semika, O., Reminyak, I., & Bezyazichna, O. (2020). Algorithm of rehabilitation intervention after plasty of the anterior cruciate ligament. *Physical rehabilitation and recreational health technologies, 5*(2), 61–64. https://doi.org/10.15391/prrht.2020-5(2).08
14. Ganjwala, D., & Shah, H. (2019). Management of the knee problems in spastic cerebral palsy. *Indian journal of orthopaedics, 53*(1), 53–62. https://doi.org/10.4103/ortho.ijortho_339_17
15. Danilov, O., Shulga, O., Kucheruk, O., & Bandrina, K. (2024). Causes of pain in the muscles of the lower extremities in children with cerebral palsy. *Orthopaedics traumatology and prosthetics,* (3), 41–48. https://doi.org/10.15674/0030-59872024341-48
16. Hoshko, V., Naumenko, N., Yatsuliak, M., Cheverda, A., Nemesh, M., & Martsyniak, S. (2020). The method of determining clinical and Roentgenogrammetric indicators of hip joint in patients with cerebral palsy. *Visnyk ortopedii travmatologii protezuvannia,* (4(107)), 35–42. https://doi.org/10.37647/0132-2486-2020-107-4-35-42
17. Min, J. J., Kwon, S., Kim, K. T., Choi, Y., Sung, K. H., Lee, K. M., & Park, M. S. (2021). Evaluation of factors affecting external tibial torsion in patients with cerebral palsy. *BMC musculoskeletal disorders, 22*(1). https://doi.org/10.1186/s12891-021-04570-5
18. Danylov, A., Gorelik, V., Shulga, A., & Yachna, K. (2023). Pathologic external tibial torsion as one of the causes of knee joint dysfunction and formation of pronation deformity in children with cerebral palsy. *Paediatric surgery. Ukraine,* (1(78)), 110–118. https://doi.org/10.15574/ps.2023.78.110
19. Helal, A. M., El-Negmy, E. H., & Zaky, N. A. (2022). Impact of femoral anteversion and tibial torsion on balance in children with spastic diplegic cerebral palsy. *International journal of health sciences,* 4572–4584. https://doi.org/10.53730/ijhs.v6ns7.12982
20. Mustafaeva, A. R., Novikov, V. A., Umnov, V. V., & Vissarionov, S. V. (2025). Surgical treatment of knee flexion contracture in patients with cerebral palsy: A review. *Pediatric traumatology, orthopaedics and reconstructive surgery, 13*(2), 202-214. https://doi.org/10.17816/ptors678129
21. Martinez, G., Segovia, J., & Cariola, M. (2024). Tratamiento de la contractura en flexion de rodilla en parálisis cerebral. *Revista chilena de ortopedia y traumatología, 65*(01), e40–e46. https://doi.org/10.1055/s-0044-1786541
22. Pantzar-Castilla, E. H., Wretenberg, P., & Riad, J. (2021). Knee flexion contracture impacts functional mobility in children with cerebral palsy with various degree of involvement: A cross-sectional register study of 2,838 individuals. *Acta orthopaedica, 92*(4), 472–478. https://doi.org/10.1080/17453674.2021.1912941
23. Shah, H., & Ganjwala, D. (2024). Crouch gait: Evaluation and management. *International Journal of Paediatric Orthopaedics, 10*(1), 20-25. https://doi.org/10.13107/ijpo.2024.v10i01.174
24. Cloodt, E., Wagner, P., Lauge-Pedersen, H., & Rodby-Bousquet, E. (2020). Knee and foot contracture occur earliest in children with cerebral palsy: A longitudinal analysis of 2,693 children. *Acta orthopaedica, 92*(2), 222–227. https://doi.org/10.1080/17453674.2020.1848154
25. Herasymenko, S., Poluliakh,, M., Babko, A., Herasymenko, A., Kachan, D., & Poluliakh, D. (2024). Biomechanical particularities of knee joint flexion defomation in rheumatoid arthritis patients. *Orthopaedics traumatology and prosthetics,* (4), 32–40. https://doi.org/10.15674/0030-59872024432-40
26. Bilgili, F., Temelli, Y., & Akalan, N. E. (2012). Assessment of the hip and knee flexion contractures in cerebral palsy patients with crouch gait. *Journal of academic research in medicine, 2*(2), 10–14. https://doi.org/10.5152/jarem.2012.11.
27. Ciccodicola, E., Abousamra, O., Beltran, V., Rethlefsen, S. A., & Wren, T. A. (2025). Clinical measures associated with tibial torsion in ambulatory children with cerebral palsy and Pes valgus. *Journal of the pediatric orthopaedic society of North*

REVIEW

America, 10, 100151. https://doi.org/10.1016/j.jposna.2024.100151
28. Thomason, P., Graham, K., Ye, K., O'Donnell, A., Kulkarni, V., Davids, J. R., & Rutz, E. (2025). Knee surveillance for ambulant children with cerebral palsy. *Journal of children's orthopaedics, 19*(4), 253–266. https://doi.org/10.1177/18632521251330448
29. Dan, B., Rosenbaum, P., Carr, L., Gough, M., Coughlan, J., & Nweke, N. (2025). Proposed updated description of cerebral palsy. *Developmental medicine & child neurology, 67*(6), 700–709. https://doi.org/10.1111/dmcn.16274

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FLEXION CONTRACTURES OF THE KNEE JOINTS IN CHILDREN WITH CEREBRAL PALSY: PATHOGENESIS, CLINICAL MANIFESTATIONS, ANATOMICAL AND MORPHOLOGICAL CHANGES IN BONES, MUSCLES AND INTRA-ARTICULAR STRUCTURES

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PREVIEW