Review

Cerebral palsy: elements of current orthopaedic care

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Abstract

Cerebral palsy is a permanent neurological disorder characterized by static central nervous system injury and progressive musculoskeletal adaptations that impair motor development. The interaction between neurological deficits and evolving orthopaedic changes requires continuous assessment to guide treatment. A structured diagnostic approach, incorporating neurological and orthopaedic examinations, comorbidity evaluation, laboratory testing, and standardized functional scales, is essential for accurate prognosis and individualized care. Deformities in cerebral palsy often worsen with growth, driven not only by spasticity but also by adaptive changes in muscle tissue, highlighting the need for preventive rather than solely reconstructive orthopaedic interventions. Recent insights into muscle pathology support minimally invasive surgical strategies that align with the dynamic nature of the disorder. This review explores the relationship between central nervous system injury, musculoskeletal development, and treatment planning in children with cerebral palsy, emphasizing tailored, timely, and multidisciplinary interventions.

Keywords: Cerebral palsy; spasticity; musculoskeletal pathology; muscle contracture; diagnostic matrix; deformity prevention; orthopaedic interventions; minimally invasive operative techniques; goal setting, quality of life

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Cite this paper as: Pasparakis D, Mitsiokapa E, Spanou M. Cerebral palsy: elements of current orthopaedic care. AOTH. 2026; 77(2):41-59.

DOI https://doi.org/10.69133/aoth.v77i2.538

Introduction

Cerebral palsy (CP) is a lifelong neurological disorder caused by permanent central nervous system (CNS) injury and progressive musculoskeletal adaptations. The interaction of static neurological damage with growth-related orthopaedic changes profoundly affects motor development in children.[1] Because functional abilities vary widely, comprehensive assessment is essential. A diagnostic framework that integrates neurological and orthopaedic examinations, comorbidity evaluation, laboratory testing, and functional scales enables accurate prognosis and individualized treatment planning.[2] The timing and location of interventions are guided by the recognition that the relationship between deformity and spasticity is multifactorial, with muscle adaptations during growth contributing to progressive contractures and skeletal deformities.[3] Accordingly, pediatric orthopaedic care emphasizes preventive rather than purely reconstructive strategies. Advances in muscle pathology research further support minimally invasive techniques that balance effectiveness with reduced surgical burden. We will investigate the intricate relationships between CNS damage, musculoskeletal changes, and treatment strategies for children with cerebral palsy, highlighting the need for ongoing assessment and tailored interventions.[4,5]

What is CP; a disease, a description or a diagnosis?

CP is a complex condition that is often misunderstood. It is not classified as a disease; instead, it serves as an umbrella term for a group of permanent disorders affecting movement and posture.[6] According to the International Consensus Definition: "CP describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of CP are often accompanied by comorbidities i.e. disturbances of sensation, perception, cognition, communication and behavior, by epilepsy and by secondary musculoskeletal problems".[7] CP is primarily a clinical diagnosis without a single pathognomonic sign or a definitive test for its identification[8], resulting from brain injury at different developmental stages.[9] It presents with musculoskeletal disturbances and often comorbidities.[10] CP is considered part of the Upper Motor Neuron Syndrome (UMNS), which arises from damage to upper motor neurons in conditions such as stroke, spinal cord injury, multiple sclerosis, and degenerative diseases. In CP, UMNS results specifically from a non-progressive brain injury occurring during pregnancy, delivery, or early infancy.[11]

Forms of CP

CP is categorized by movement disorder, neuromuscular appearance and topographical distribution:[12]

a) The ***traditional topographical classification*** of CP is based on limb involvement: mono-, hemi-, tri-, and tetra-plegia. The SCPE redefines these as follows: mono/hemiplegia → unilateral CP, di/triplegia → bilateral CP, and tetra/quadriplegia → total/whole-body CP.[1]

b) ***Movement disorder***: *Pyramidal tract* lesions are associated with spasticity or velocity-dependent hypertonia disorders. *Extra pyramidal tract* lesions with *basal ganglia* involvement are associated with dyskinesias i.e dystonia or choreoathetosis, whereas *cerebellar* involvement is associated with ataxia and hypotonia.[14]

c) The ***neuromuscular classification*** defines CP by movement disorder. The SCPE recognises spastic, dyskinetic, and ataxic forms; dyskinesia includes dystonia, athetosis, and chorea. Some children show mixed-tone features.[15] Since dystonia and spasticity differ pathophysiologically, they require distinct management.[16]

What is spasticity?

According to Lance (1980), "spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of stretch reflex, as one component of the upper motor neuron syndrome".[17] Thus, spasticity is characterized by excessive resistance during passive muscle stretch, which the examiner feels as a catch which is proportional to the speed at which

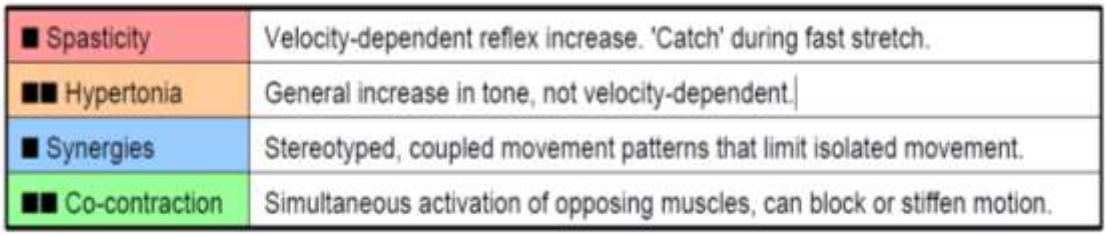

Figure 1. *Motor abnormalities in UMN syndrome.*

stretching is performed.[18] This phenomenon is due to exaggerated stretch reflexes, not merely muscle stiffness.

Common Misconception: Spasticity is not just "muscle tightness"- it is a velocity-dependent reflex phenomenon, strictly related to the exaggerated stretch reflex, caused by UMN damage. Spasticity is just ONE component of UMN syndrome. It is often mistakenly used as a broad label for all motor abnormalities seen in UMN lesions[17,11] including (Figure 1):

Hypertonia - general increase in muscle tone, not always velocity-dependent

Abnormal synergies - stereotyped movement patterns, abnormal muscle coupling

Co-contractions - simultaneous activation of opposing muscle groups

Mislabeling it as a catch-all for UMN motor problems can lead to clinical misunderstandings[19] and inappropriate treatment approaches.[20]

Impact on movement: Spasticity occurs after motion has begun, indicating that some muscle lengthening has already taken place. This suggests that spasticity is triggered by movement; it is a response to stretch rather than a precursor to movement. The exaggerated stretch reflex associated with spasticity can interfere with muscle function and disrupt normal movement patterns, leading to dysfunction in motor control. This "contamination" of movement execution can lead to a deregulation of motor actions.[21]

Clinical picture of CP; is it static?

CP is categorized as a non-progressive static encephalopathy, while the physical manifestations can evolve due to several factors.[1] Progressive musculoskeletal changes are by definition expected[7]; over time, children with CP often experience: increased muscle stiffness, progressive restrictions of joint motion, arising contractures and anatomical changes to normal alignment that can further exacerbate difficulties with movement and posture.[22] In effect, CP is static in terms of brain injury, but progressive in terms of musculoskeletal complications.

How might a static encephalopathy result in secondary progressive musculoskeletal deformities?

The "Traditional View" Children may appear normal at birth, but as they grow, muscle hypertonia and spasticity lead to progressive deformities.[23] Mercer Rang (1990) described this process as occurring in three key stages of deformity development.

Stage 1: Dynamic Contracture – Range of motion (ROM) limits are experienced during active movements, but passive ROM remains normal

Stage 2: Fixed Contracture – Permanent restrictions in joint motion become evident, transitioning to fixed contractures

Stage 3: Fixed Contractures with Joint Damage – As contractures become more severe, they lead to joint damage, further complicating movement.

This progressive model, driven mainly by spasticity as the key factor in CP complications, is termed "the traditional view".[16,24]

Consequences of the "traditional view" on CP management

While this view has been influential, it carries several consequences for the management and treatment

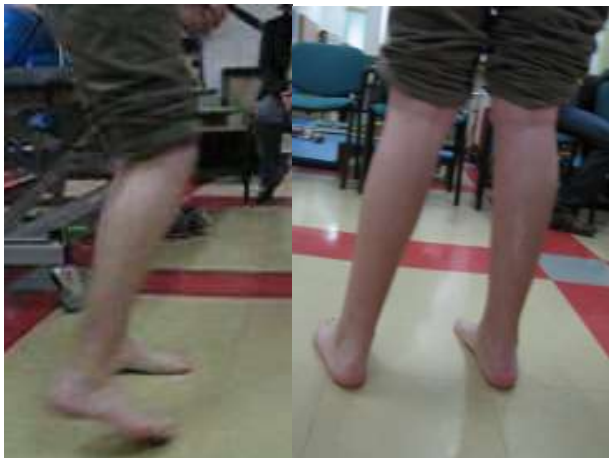

Figure 2. *Dynamic contractures in CP.*

Figure 3. *Short muscle disease in CP.*

of CP including:

Linear causation assumption: The *"Traditional View"* posits an absolute linear cause-and-effect relation between spasticity and joint contractures in children with CP. This perspective suggests that exaggerated reflex activity (spasticity) leads to prolonged muscle shortening, resulting in muscle growth failure, joint movement restrictions (contractures), and ultimately, permanent fixed deformities.[25,26]

Overemphasis on spasticity as the primary cause: Viewing spasticity as the leading cause of deformities oversimplifies CP, overlooking factors like muscle weakness, abnormal movement, and proprioceptive deficits, and risks neglecting other contributors to functional limitations.[27]

Anti-spasticity interventions: The traditional view prioritises anti-spasticity treatments like BTXA and SDR, assuming spasticity reduction will prevent contractures and improve motor skills.[28,29] Orthopaedic surgery is considered a last resort, reflecting a focus on spasticity over structural issues.

Does spasticity matter?

The role of spasticity in CP has been a subject of considerable debate among researchers and clinicians.[30-32] Historically, the term *spasticity* was applied broadly to describe a wide range of motor dysfunctions, including abnormal reflexes, impaired motor control, weakness, and even musculoskeletal deformities.[33] Lance defined spasticity as a motor disorder specifically related to *exaggerated stretch reflex activity*[17], leading to the assumption that reducing it would improve motor function. However, recent research shows that lowering spasticity does not necessarily enhance motor development or outcomes.[34,35]

Muscle strength vs. spasticity: Evidence increasingly highlights muscle weakness—not spasticity—as a stronger predictor of function. Ross & Engsberg (2007) found that muscle strength, rather than spasticity, was closely correlated with gait and functional outcomes in children with CP. Spasticity showed little or no significant correlation, suggesting interventions should prioritise strengthening over antispasticity alone.[36]

Limited impact of spasticity on functional development: Gorter et al. reported only a weak relationship between spasticity and Gross Motor Function Measure (GMFM) development, with environmental and familial influences having greater effects.[37]

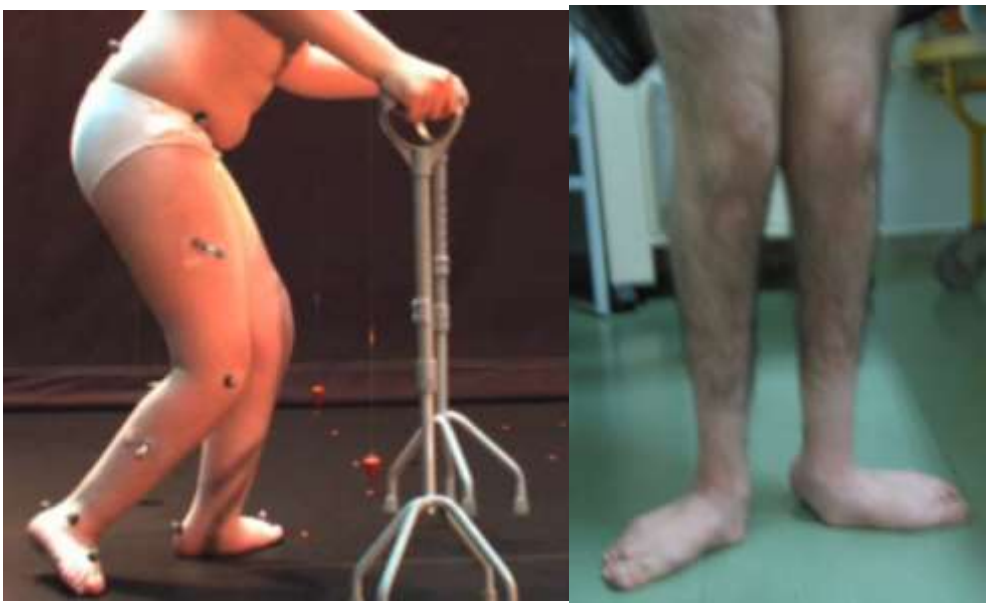

Figure 4. *Lever arm disease in CP.*

Similarly, Noble (2019) showed that selective motor control (SMC)-measured via the SCALE tool-was more predictive of GMFM outcomes than muscle volume or joint stiffness, underscoring the limited role of spasticity in functional performance.[38]

Antispasticity treatments and orthopaedic surgery needs selective dorsal rhizotomy (SDR): Long-term follow-up studies consistently demonstrate that while selective dorsal rhizotomy (SDR) effectively reduces spasticity, it does not fully prevent musculoskeletal complications. Tendrof et al. (2011), in a 10-year follow-up of 19 children with cerebral palsy, found that despite spasticity reduction, contractures continued to develop, with 16 patients requiring an average of three orthopaedic interventions.[39] In a broader review of 16 studies with ≥10 years of follow-up, Tendrof et al. (2020) reported that between 28% and 94% of patients required orthopaedic surgery for contracture correction.[40] Similarly, Park et al. (2017), evaluating outcomes of childhood SDR into adulthood, observed that 59% of patients underwent orthopaedic surgery.[41] Tu and Steinbok (2020), in a systematic review of 19 studies involving 1,054 patients, undergoing SDR with 10 years or more of outcome data reported almost identical findings, with 60% of patients requiring limb surgery.[42] More recent work reinforces these conclusions. MacWilliams, in a follow-up study of over 10 years, compared patients treated with SDR plus antispastic injections to those managed without SDR and limited injections, finding that SDR reduced spasticity and modestly improved gait but did not prevent long-term musculoskeletal complications or improve overall functional outcomes.[43] Likewise, Marron (2023) reported that although SDR may improve gait in the short term, long-term outcomes often mirror those of standard care, underscoring that musculoskeletal complications in CP arise from factors beyond spasticity alone.[44] Collectively, these findings suggest that while SDR addresses spasticity effectively, it does not eliminate the need for orthopaedic interventions, highlighting the multifactorial nature of musculoskeletal deformities in cerebral palsy.

Botulinum toxin A (BTXA): Graham et al. and Willoughby et al showed repeated BTX-A injections did not halt hip displacement.[45,46] Fattal-Valevski et al. (2008) found that up to 4 BTX-A injections over 2 years improved long-term gross motor function in children with CP, though tone reduction was short-lived, with optimal benefit after 2–3 treatments.[47] Similarly Tendroff et al. (2009) found only tempo-

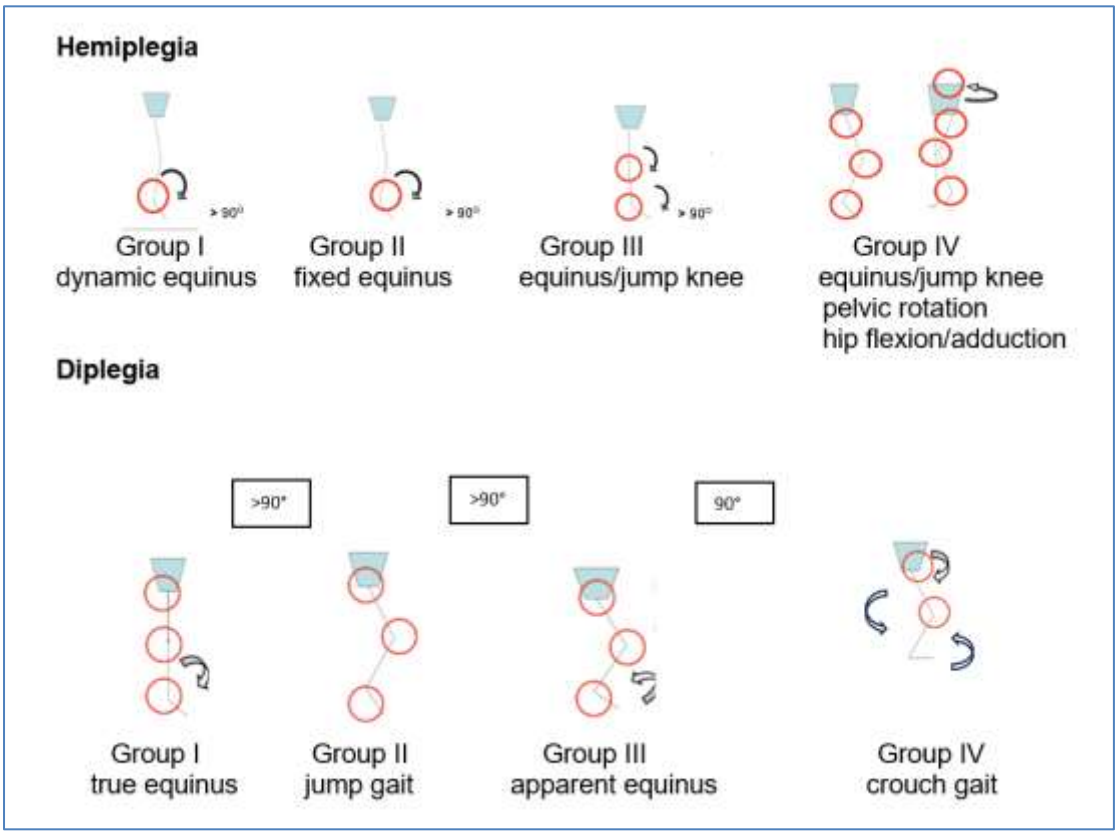

Figure 5. *The Winter-Rodda-Gage-Graham Classification in CP.*

rary range-of-motion (ROM) improvements, with contractures continuing over time.[48] A systematic review by Sätilä concluded BTXA may "buy time," but most children older than six still required surgical intervention.[49] Lin et al. also found that among 1,405 CP children (281 BTX-A/1.124 controls) followed for 5 years, outcomes did not differ between groups. BTX-A did not significantly prevent hip dislocation or scoliosis.[50]

Spasticity and ROM across development: Spasticity is not static. Lindin et al. found that spasticity increases during early childhood (first 4-5 years) but gradually declines until around age 12-15.[51] Nordmark et al., however, demonstrated a parallel loss of ROM with age, particularly at higher GMFCS levels.[52] The differing trajectories suggest that spasticity and ROM decline are not directly correlated, supporting the view that stiffness arises from mechanisms other than spasticity alone.

Muscle changes from birth to early childhood: Willerslev-Olsen et al. showed that children with CP develop muscle atrophy, reduced growth, and stiffness within the first 1–2 years of life, independent of spasticity.[53] In a complementary population-based study, Cloodt et al. found that among 2,693 children examined before age 5, frequent surgeries were required during a 10-year follow-up, with median ages of 4 for hip soft tissue, 7 for foot, and 9 for knee procedures.[54] The early onset of muscle growth reduction and progressive joint contracture implicates impaired muscle development, rather than spasticity alone. Furthermore when comparing the period after 5 years of age, when spasticity typically begins to decline, with the corresponding period of range of motion (ROM) reduction observed beyond 4 years, along with evidence that muscle atrophy and stiffness become established from 3–4 years of age onwards, it is reasonable to infer that progressive

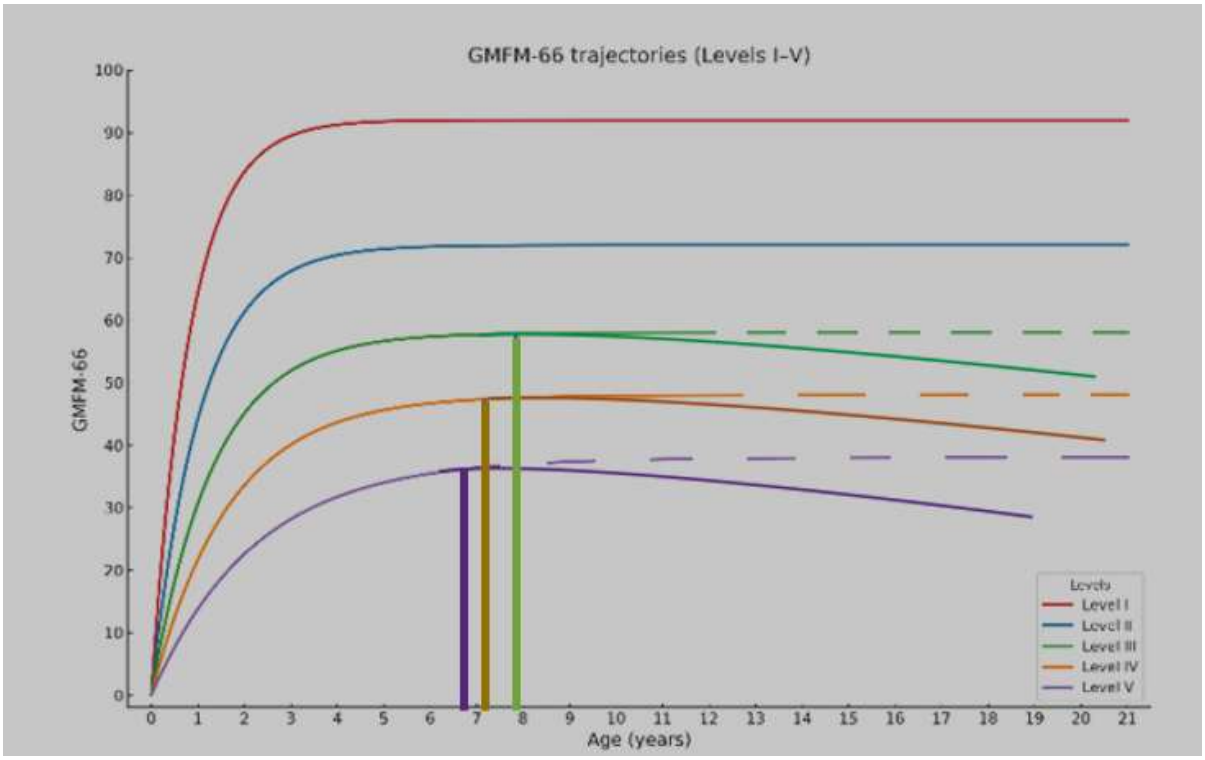

Figure 6. *Gross Motor Function (GMF) curves in CP.*

ROM restriction is more closely related to ongoing muscle atrophy and shortening rather than to decreasing spasticity. Indeed, muscle relaxation alone does not appear sufficient to prevent the development of contractures in most cases.

The spastic muscle in CP

The term "*spastic muscle*" reflects a neurological sign of upper motor neuron impairment, not an intrinsic muscle abnormality. Nonetheless, chronic exposure to disordered neural input leads to profound multi-level muscle adaptations.[55,56]

Muscle as an organ:[24] Muscle growth lags behind bone growth, leading to shorter, weaker fibers with reduced diameter.[57] This results in decreased strength, increased fatigue, and limited activity. Disuse further compounds weakness and stiffness, reinforcing a cycle of immobility.[58]

Muscle as a tissue; sarcomere adaptations: Fewer sarcomeres in series, with remaining sarcomeres overstretched, increase passive resistance and stiffness.[59]

Extracellular matrix (ECM) alterations: Hypertrophy of ECM, with excess and abnormally cross-linked collagen, reduces elasticity and heightens stiffness.[60,61]

Cellular level: Children with CP have fewer satellite cells, essential for muscle regeneration. Impaired proliferation and maturation further limit hypertrophy and repair capacity.[62]

Molecular level: Transcriptomic studies reveal increased expression of ECM-related genes and reduced oxidative metabolism genes in CP muscle, contributing to contracture formation and reduced muscle efficiency.[63,64]

Spasticity and abnormal muscle stiffness reflect distinct, partly dissociating mechanisms. Importantly, contracture is not necessarily linked to spasticity, and reducing spasticity alone does not reliably prevent deformity. Evidence indicates that, although spasticity is clinically relevant, it is not the primary driver of long-term motor impairment in cerebral palsy (CP). Instead, muscle weakness, impaired selective motor control, altered muscle

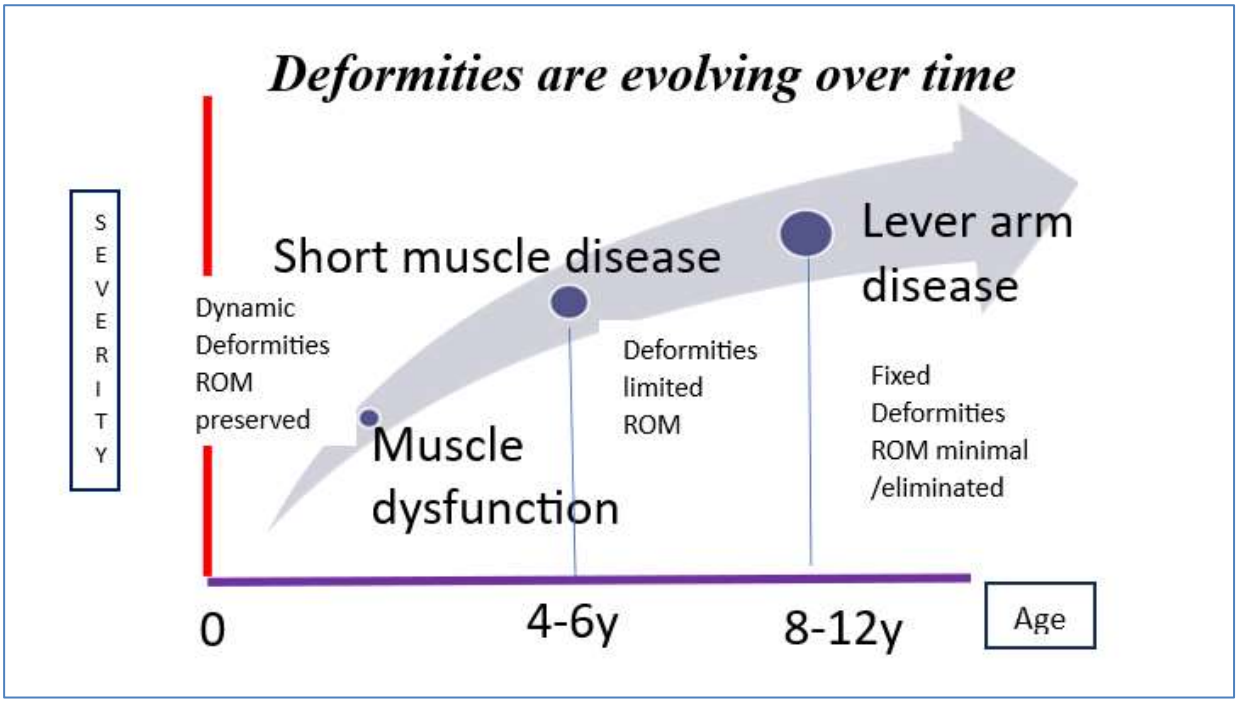

Figure 7. *Orthopaedic intervention based on child's age and nature of deformities in CP.*

growth, and extracellular matrix (ECM) remodeling appear to play larger roles. Consequently, interventions aimed solely at reducing spasticity (e.g., selective dorsal rhizotomy [SDR], botulinum toxin A [BTXA]) often yield only temporary or partial benefits. More comprehensive approaches that address muscle strength, motor control, and structural adaptations show greater promise for improving functional outcomes and quality of life.

Is CP amenable to treatment?

CP constitutes a complex and permanent neurodevelopmental disorder for which no curative therapy is currently available, as CNS damage is irreversible. Given that the CNS represents the locus of permanent impairment, restoration of normal motor function is not feasible, as this would necessitate direct intervention at the level of the central motor drive. Accordingly, therapeutic strategies are directed primarily towards management and functional optimization rather than cure.[2] Moreover, it is essential to acknowledge that the underlying neurological deficits persist despite orthopaedic or other peripheral interventions. The primary aim of orthopaedic interventions should be to improve the existing biomechanical conditions by creating a more favorable micro-biomechanical environment, enhancing posture, and movement efficiency, ultimately promoting quality of life by correcting or reducing deformities.[65] The "novelle approach" to the pathology of deformities is based on strong evidence that loss of joint ROM is deteriorating over time because of progressive muscle stiffness and diminishing muscle length, while spasticity development is decreasing during the same growth period. Accordingly it becomes crucial the systematic long-term clinical monitoring of both clinical phenotypes aiming to distinguish between these two different pathological entities.

Orthopaedic decision-making: luck or science?

In orthopaedic decision-making, successful treatment planning depends on integrating multiple factors through both clinical knowledge and practical experience.[66,67] It is a difficult detection process involving a variety of sources of information from multiple components. A clinician needs to invest significant time and effort into assimilating all the

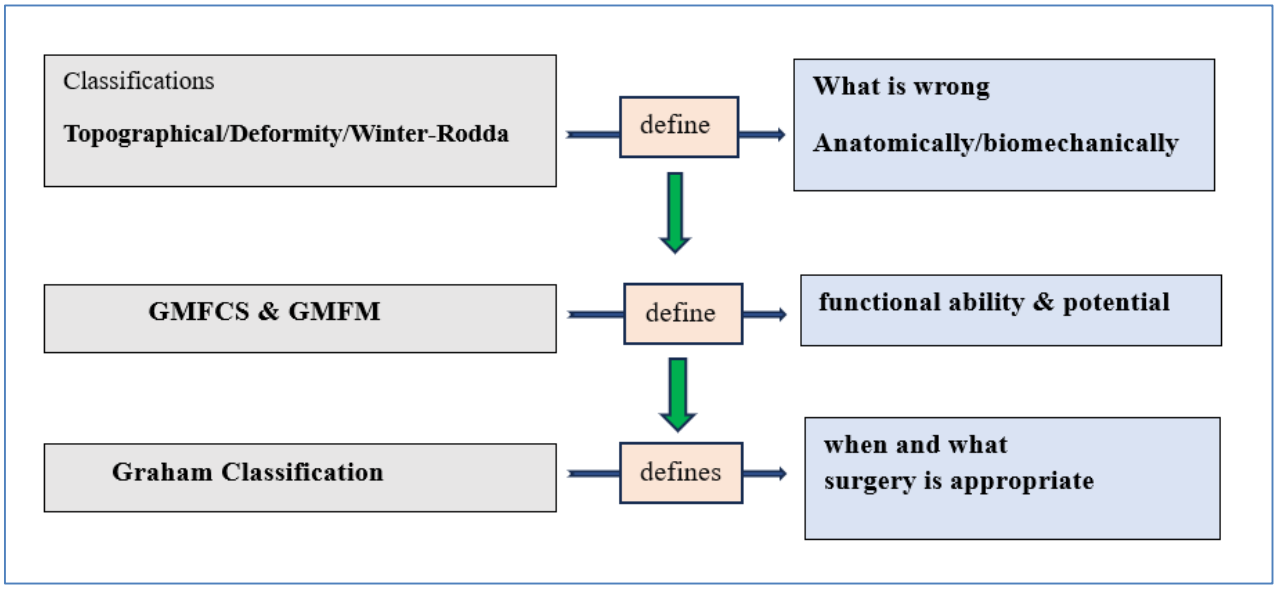

Figure 8. *Comprehensive preoperative planning in CP.*

available clinical information to determine the impairments that affect a child's capacity to walk.

This complex process requires identifying key elements, which are organized within a diagnostic matrix.[2] The matrix includes several pivotal components:

1) Clinical history[68]

2) Standardized neurological and orthopaedic clinical examination

3) Evaluation of spasticity levels

4) Assessment of soft tissue contractures and deformities: Joint ROM should be regularly assessed with standardized techniques, documenting changes in muscle length for longitudinal tracking. A key challenge is distinguishing ROM limits caused by spasticity versus structural contractures, since both are tested with passive stretch. To improve accuracy, assessments should be performed at slow and fast stretch velocities in standardized positions, reducing reflex interference [69]. Correctly differentiating spasticity from contracture is crucial, as it determines whether conservative or surgical approaches are most appropriate.

5) Measurement of muscle strength: Muscle weakness, often more impactful than spasticity, is a key contributor to musculoskeletal deformities. Beyond flexor contractures, extensor insufficiency also plays a critical role in limiting range of motion.[70]

6) Classification of deformities:

a) **Dynamic Contractures** (Figure 2): In this category, movement limitations are transient and occur only during specific motor tasks as a result of exaggerated stretch reflex activity. Importantly, full passive joint ROM is preserved, indicating that muscle length remains within normal limits.[71]

b) **Fixed - short muscle disease** (Figure 3): Over time, muscle tissue may undergo structural changes, becoming increasingly stiff and shortened. This process leads to a progressive loss of joint range of motion. As a result, the initially dynamic nature of contractures diminishes, evolving into a static condition that persistently restricts movement.[71]

c) **Fixed deformities - lever arm disease (LAD)** (Figure 4): Permanent muscle stiffness, shortening, and abnormal movement cause pathological forces on joints and bones, disrupting growth and leading to deformities.[72,73]

7) Winter-Rodda-Gage-Graham classification (Figure 5): This four-group system categorizes gait patterns according to the severity of joint involvement and associated deformities. It provides a structured framework for clinical decision-making, help-

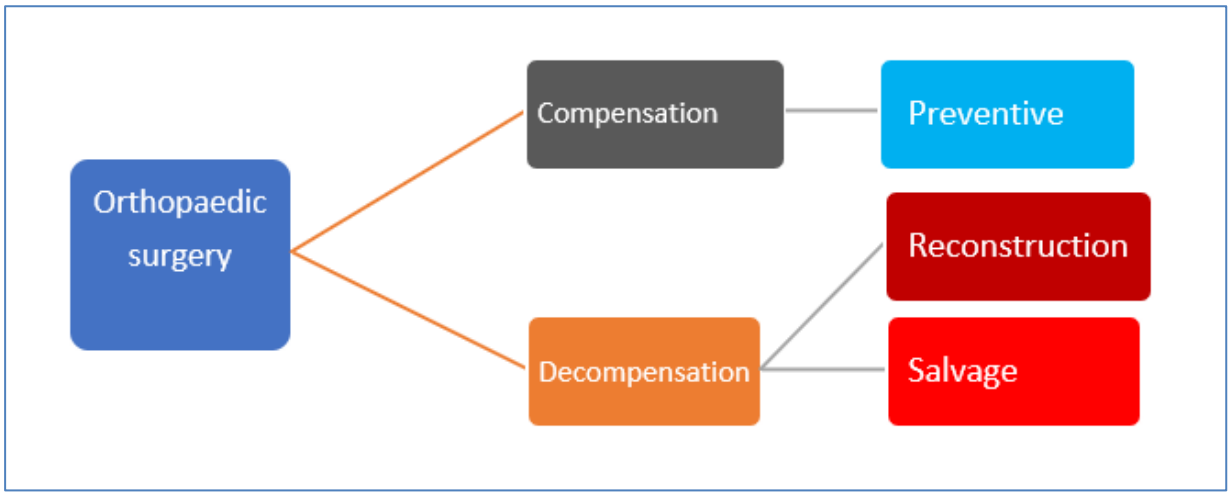

Figure 9. *Types of orthopaedic surgery in CP.*

ing to guide the selection of appropriate therapeutic interventions, including orthotic management, botulinum toxin injections, and orthopaedic surgical procedures.[74]

8) GMFM (Gross Motor Function Measure): A scoring system that evaluates gross motor function changes based on developmental milestones.[75]

9) GMFCS (Gross Motor Function Classification System): A severity grading system that correlates with clinical and musculoskeletal deformity levels. It helps in long-term prognosis and treatment planning.[75]

10) GMF curves: These curves represent typical patterns of motor development and are stratified according to the five levels of the GMFCS. Each GMFCS level has a distinct gross motor development curve, reflecting differences in the rate and extent of skill acquisition (Fig. 6). Children in Level I reach higher function and plateau later, while those in Levels IV–V show limited gains and earlier plateaus. Mapping a child's progress on their GMFM curve aids assessment, guides goal setting and intervention, and informs family counseling about likely functional outcomes.[75]

11) FMS (Functional Mobility Scale): An outcome measure system used for evaluating changes before and after surgeries.[76]

12) Diagnostic imaging: Early identification of progressive deformities in the spine, lower limbs, feet, and hips.

13) Instrumented gait analysis (IGA): This method records and classifies gait deviations, especially complex abnormalities, providing crucial information for treatment planning. These systems and tools are essential for understanding locomotion in CP patients and for tailoring effective treatment strategies.[77]

The timing of orthopaedic intervention

The timing of orthopaedic surgery is debated.[78] Traditionally, a staged approach was used starting with anti-spasticity treatments in early childhood and progressing to more invasive orthopaedic surgeries later.[79] More recent recommendations suggest delaying orthopaedic surgery until ages 7–10 (especially for levels GMFCS II & III until ages 10-12) when gait patterns are more mature and the risk of recurrence is lower.[80] At this stage, all fixed muscle contractures and skeletal deformities can be corrected in a single procedure (SEMLS), thereby improving or maintaining walking performance. In the meantime, management often focuses on conservative options such as BTXA, orthoses and physiotherapy.[81]

An alternative interpretation suggests that the optimal window for musculotendinous lengthening procedures of the lower extremities is between

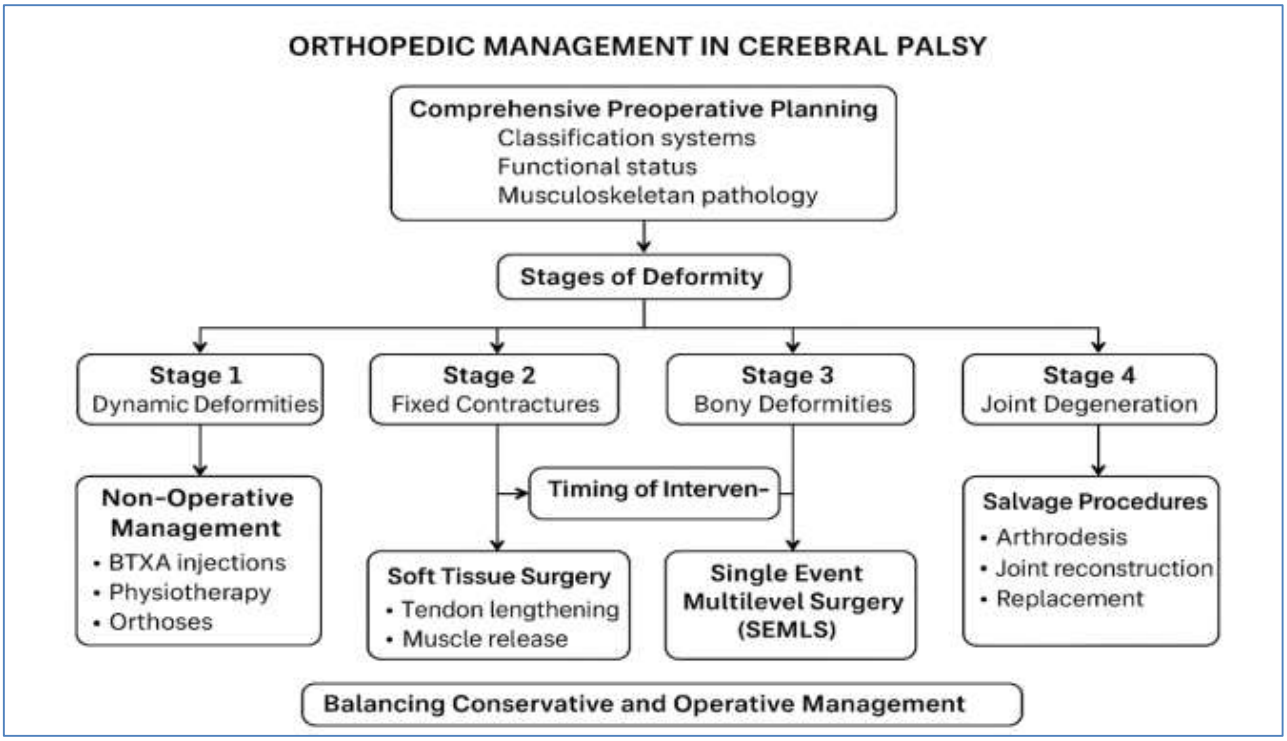

Figure 10. *Orthopaedic management in CP.*

4 and 7 years of age, given the early onset of lower limb contractures in children with cerebral palsy.[54,82] The rationale is that surgical outcomes are most favorable when performed before the development of severe fixed contractures and skeletal malalignments, which are often responsible for stagnation or regression in gross motor function.[78,83,84] Lennon et al (2024) provide further support for age-sensitive surgical planning, demonstrating that improvements in gait kinematics are strongly influenced by both age and timing of orthopaedic intervention. The most substantial and durable gains were observed in children under 10 years of age, whereas children operated on after this threshold exhibited significantly diminished improvements. Importantly, although surgery was associated with positive outcomes across all age groups, the magnitude of benefit was consistently reduced in older cohorts.[85] Conversely, delaying intervention until after these complications have developed substantially diminishes the effectiveness of surgery due to the presence of complex, decompensated joint and skeletal pathologies.[78]

In 2021, Graham et al. introduced a four-stage classification system that correlates orthopaedic intervention with the child's age and the nature of their deformities[71,86] (Figure 7):

Stage 1 (dynamic deformities, birth to age 4-6 years): Characterized by hypertonia with minimal or no contractures. The focus is on tone management, often using BTXA injections to address spasticity.

Stage 2 (contractures, age 4-12 years): After the age of 5 years, spasticity tends to diminish, but fixed contractures progressively worsen. Recognizing the transition from dynamic (spasticity-driven) deformities to fixed contractures is crucial. At this stage, orthopaedic soft tissue surgery may be indicated.

Stage 3 (bony deformity, age 4-12 years): As contractures develop, secondary bony deformities often arise, further contributing to functional impairment. Management at this stage usually requires a combination of bony realignment procedures (such as osteotomies) and soft tissue releases or lengthenings. These are often performed together during a single

operative session (SEMLS).

Stage 4 (decompensation, age 10 years to adulthood): By this stage, severe joint contractures and bony deformities make it difficult to restore optimal function, and interventions are often seen as salvage procedures.

This classification highlights that *orthopaedic intervention is not a last resort,* but rather an integral component of managing fixed deformities at stages 2, 3, and 4. In contrast, BTXA injections or SDR are primarily indicated for, when deformities are still dynamic and spasticity-driven. While non-operative methods are often prioritized initially, it is crucial to recognize that delaying surgical intervention can result in lost functional capacity. Achieving the best outcomes requires balancing conservative management with timely surgery, taking into account the time-sensitive window for optimal functional improvement.[83]

Comprehensive framework for orthopaedic management in CP

Effective surgical management of orthopaedic problems in patients with CP requires a comprehensive, individualized approach that integrates the patient's functional status, anatomical deformities, underlying muscle pathology, and the patient's developmental trajectory.[2] Central to this process is thorough preoperative planning.

Key components include:

1. Comprehensive preoperative planning (Figure 8) Effective planning involves a detailed assessment using multiple classification systems to guide decision-making and prioritize interventions:

a. Patient assessment

Topographical classification

Identifies distribution of involvement (hemiplegia, diplegia, quadriplegia, etc.)

Deformity classification

Structural vs. dynamic deformities

Identifies contractures, torsions, lever-arm dysfunction

Winter-Rodda classification

Describes specific gait patterns (e.g., true equinus, jump gait, crouch gait, stiff knee gait)

b. Functional capacity

GMFCS

Levels I–V (predicts long-term mobility potential)

GMFM Curves

Plots developmental trajectory over time

Tracks progress, monitors therapy outcomes, informs goal-setting

c. Surgical planning

Graham classification

Age-based deformity classification

Incorporates joint restriction and lever-arm dysfunction

Surgery Guides **timing** and **selection** of multilevel procedures (e.g., SEMLS)

2. Timing of intervention

Stage 1 (Dynamic deformities): Focus on non-operative methods such as BTX-A injections, physiotherapy, and orthoses. Early intervention aims to reduce spasticity and prevent contractures.

***Stages 2–4 (Fixed contractures and bony deformities)*: Soft tissue surgery** (e.g., tendon lengthening, muscle release) addresses persistent contractures.

Single event multilevel surgery (SEMLS) may be indicated to correct multiple deformities, by combining multiple orthopaedic procedures (soft tissue and bony procedures) in a single session to reduce recovery time and repeated surgeries.[68] This approach minimizes hospital stays and streamlines rehabilitation processes.[80]

3. Types of orthopaedic surgery (Figure 9) Orthopaedic surgeries can be categorized into three main types:[78]

Preventive surgery: Aimed at preventing the progression of deformities.

Reconstructive surgery: Focused on correcting existing deformities.

Salvage surgery: Intended for cases where previous interventions have failed.

4. Minimally invasive techniques Minimally invasive surgical techniques are preferred for both bony and soft tissue interventions.[87] Percutaneous muscle lengthening involves making very small incisions to release the appropriate amount of myofascial tissue necessary to lengthen the muscle, re-

ducing spasticity and increasing ease of motion.[88] Regarding osseous procedures, derotation osteotomies of the femur and tibia are optimally performed using closed corticotomy techniques with stabilization provided by titanium elastic nails or Steinmann pins.[89] Additionally, guided growth epiphysiodesis, employing either eight-plates or cannulated screws, represents a minimally invasive approach for correction of angular deformities.[90,91] These procedures reduce recovery time and minimize complications associated with larger interventions.

5. Muscle selection for interventions When selecting muscles for surgical intervention, it is crucial to focus on *Multiarticular Muscles* (psoas, rectus femoris, hamstrings, gastrocnemius). These are vital for gait propulsion and coordination but are often more affected by spasticity and weakness, while contributing less to antigravity support compared to single-joint muscles. *Monoarticular muscles* provide joint stability and essential antigravity function, and should generally be preserved to maintain posture and overall function.[92,78]

6. Consideration of specific muscle pathology: Soft tissue surgery for neuromuscular disorders should explicitly address intrinsic muscle pathology, particularly the increased passive stiffness resulting from excess extracellular matrix (ECM) hypertrophy and collagen accumulation.[93] In cerebral palsy (CP), maladaptive ECM remodeling—including elevated collagen content, altered cross-linking, and aberrant ECM architecture—contributes significantly to reduced muscle extensibility and functional impairment.[55,56] Surgical techniques like aponeurotic or fractional lengthening are designed to target accessible connective tissue layers (fascia, epimysium, aponeurosis).[94] By releasing these external ECM components, such interventions reduce the mechanical resistance to stretch, permit sarcomere lengthening, improve joint mobility, and restore more favorable force transmission, while preserving overall muscle fiber integrity and the architecture of the muscle-tendon unit.[95,92]

Aponeurotic lengthening and myofascial release: The technique involves multiple crosswise incisions of the fascia/aponeurosis (epimysium), perpendicular to its length, over the adjacent muscle belly. The muscle belly released from a stiffer sheath is allowed to be stretched in a new lengthened position and deformity corrected, while muscle integrity is preserved and muscle belly – tendon ratio improved.[96,92]

***Fractional lengthening*:** The target region is the anatomical site where the muscle belly laps into the tendon. The tendon is crosswise transected with multiple perpendicular incisions and the musculotendinous unit continuity is disrupted. The muscle in the region of the cuts is able to stretch in the new lengthened position, which can help reduce spasticity and improve range of motion.[97,95] Both techniques reduce sarcomere tension in sectioned fascia and musculotendinous regions, allowing the muscle to reset at a new resting length. This decreases spasticity and stiffness, improving joint ROM.[98]

7. Balancing conservative and operative management: Early stages focus on non-operative care to manage spasticity and maintain mobility. Recognition of time-sensitive surgical windows is essential to preserve or improve functional outcomes. Decisions should consider patient-specific goals, potential functional gains, and risks of delaying surgery (Figure 10).

8. Goal setting and family counseling: When setting goals for CP children, it is important to use the GMFCS level and GMFM developmental curves to guide realistic expectations. Families benefit from counseling that explains likely progress, timing of interventions, and long-term planning. Goals should be individualized across key domains—independence, communication, mobility, daily activities, fitness, and social participation—while also preparing for future transitions into adulthood.[4] This holistic approach ensures that therapy focuses not only on motor skills, such as walking, but also on meaningful daily life, inclusion, and long-term quality of life.

Conclusion

Cerebral palsy presents a multifaceted challenge in terms of definition, clinical manifestations, and therapeutic strategies. It encompasses a range of

disorders with varying etiologies and associated comorbidities. Although the initial central nervous system injury is static, musculoskeletal issues tend to progress during growth. Surgical interventions should be viewed as management strategies rather than definitive treatments aimed at restoring normality. They are designed to enhance mobility, fitness, and independence by addressing movement impairments due to musculoskeletal deformities. Decision-making requires careful consideration of clinical data collected throughout growth, with an emphasis on age-related factors and specific muscle pathologies. Ultimately, orthopaedic surgery aims to improve biomechanical environments to facilitate better posture and motion while anticipating potential complications from underlying neurological conditions.

Conflict of Interest

The authors declared no conflicts of interest.

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