HHS Public Access

Author manuscript

Neurol Clin. Author manuscript; available in PMC 2021 May 01.

Published in final edited form as:

Neurol Clin. 2020 May ; 38(2): 397–416. doi:10.1016/j.ncl.2020.01.009.

Principles of Medical and Surgical Treatment of Cerebral Palsy

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Keywords

Cerebral Palsy; Movement Disorders; Management Principles; Complex Care

Overview: Nature of the Problem

Cerebral palsy (CP) describes “a group of 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 disturbances of sensation, cognition, communication, perception, and/or behaviour, and/or by a seizure disorder”[1]. Cerebral palsy is the most common cause of physical disability in children (2–3/1000 live births worldwide)[2]. After decades of variability in prevalence rates of CP, advances in prenatal and neonatal care including therapeutic hypothermia for perinatal hypoxic-ischemic encephalopathy[3] and antenatal magnesium for preterm delivery[4] finally appear to be improving rates in developed countries[5].

The underlying etiology of CP ranges from brain malformations to preterm white matter injury, hypoxic-ischemic injury, pre-, peri- or postnatal stroke, genetic disorders, CNS infection, or early traumatic brain injury. In developed countries, only 2–10% of cases are attributable to perinatal hypoxia-ischemia[6]. A majority of cases are associated with periventricular white matter injury[7] attributable to hypoxic-ischemic and neuroinflammatory preterm insults. While the initial brain insult is considered “static,” the manifestations over the lifetime are dynamic processes that require lifelong management. Etiologies of motor deficits that instead follow a progressive course with continuing central nervous system injury are known as “masqueraders” [8] or “mimics” [9] of CP, which include a variety of genetic and metabolic disorders.

As the diagnosis of CP permits widely heterogeneous etiologies and anatomic patterns of brain abnormality, motor phenotypes in individuals with CP are often equally complex.

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Qualitatively, recognizable movement disorders range from spasticity to dystonia, choreoathetosis, tremor, and myoclonus (and often combinations of the above). While spasticity is the most commonly recognized movement disorder in CP, roughly one quarter of individuals demonstrate a clinically-significant additional or alternative movement disorder[10]. In currently accepted nomenclature, motor phenotypes of CP are classified by the dominant movement abnormality into spastic, dyskinetic, ataxic and mixed subtypes. However, even within a physiologic CP subtype (e.g. “spastic” or “dyskinetic”), there can be wide variation in severity, co-existing movement disorders, and treatment response.

None of the etiologies of CP impact motor pathways in isolation, and co-existing diagnoses are common. The primary insult/malformation itself frequently (but variably) impacts a number of additional neurologic and neurocognitive domains:

- Epilepsy: 22–40% of children with CP develop epilepsy; children with hemiplegic or quadriplegic motor impairment are at particular risk[11].
- Cognition: Approximately one half of individuals with CP also have intellectual disability[11]. Many others have communication difficulties[12], attentional difficulties[13], or specific learning disabilities[14]. -Somatosensation: Cortical sensory deficits are ubiquitous[15], and measurable hypoaesthesia and hyperalgesia are prevalent[16].
- Chronic pain is also prevalent but poorly-characterized to date[17].
- Other sensory deficits: A majority of individuals with CP have visual impairment, and 12–25% have hearing impairment[11].
- Oromotor dysfunction: 80% of individuals with CP (particularly individuals with dyskinetic components) require non-oral feeding at some point in their lifetime.

Additional complications of CP arise not from the initial brain injury but from resulting physiologic or psychologic stressors:

- Orthopedic alignment: Children with CP are at high risk for hip deformity or scoliosis. Both are rare in children with mild motor impairment but prominent in the majority of severely-affected individuals by adulthood[18,19].
- Disuse-related complications: Decreased mobility itself is linked to complications spanning many organ systems ranging from muscle atrophy to cardiometabolic disorders (including obesity, diabetes mellitus, hypertension, and hyperlipidemia), restrictive lung disease, skin breakdown, a decrease in gut motility, and a systemic hypercoagulable, pro-inflammatory state[20].
- Mental health: Diagnosed prevalence of anxiety and mood disorders in individuals with CP each reach 20–30% in adulthood[21].

There is no cure for CP, and treatments used are largely symptomatic with a focus on quality of life and participation in society[22]. As described above, individual needs vary widely along a number of dimensions. As such, individual management plans must be equally diverse and

versatile. In this brief review, we summarize general principles of treatment of CP and refer elsewhere for more comprehensive discussion of individual topics.

Management Goals and Patient Evaluation

Defining achievable and meaningful goals of care should represent the beginning of any management discussion. While goal selection is highly individualized, current and developmentally-predicted level of gross motor functioning can help shape discussion (Table 1).

Following careful assessment, specific motor phenotypes may be established and targeted. Caveats include:

- In individuals with mixed CP, the “dominant” movement disorder may not cause the most impairment.
- Movement disorders may be positional, intermittent, or stimulus or activity-induced, and so tailored examination with careful observation is beneficial.
- Movement quality evolves over development both in neurotypical people as well as in individuals with CP. Changes over time may be particularly pronounced in individuals with dyskinetic CP[23]. -Superimposed epileptiform movements can vary in semiology and can be complex, and the clinician must maintain a high degree of suspicion for seizure-related movements.

Identification of specific etiologies is essential for early diagnosis (including ruling out masqueraders) and for guiding treatment selection. Brain MRI is the accepted standard screening evaluation for all individuals with CP[24] and represents the first step in the etiologic evaluation. Macroscopic sequelae of brain injury/malformations are apparent in 83–86% of individuals with CP [25].

Genetic and metabolic testing are not currently routinely recommended for individuals with CP, but suspicion of a masquerader or a discordance between clinical and imaging findings should prompt further investigation[26]. Currently available whole exome sequencing now shows a 14% yield for clear pathogenic variants even without case pre-selection when obtained by a specialty clinic [27]. Increasing clinical utility for genetic/metabolic testing may lead to more common use in the future. Early identification of masqueraders is particularly important as prompt treatment can mitigate or prevent symptoms in selected disorders (e.g. dietary modification in glutaric aciduria type 1, supplementation for cerebral folate deficiency, or enzyme cofactor replacement for molybdenum cofactor deficiency type A; reviewed in [28]).

Appropriate identification of coexisting medical and developmental diagnoses is equally imperative to successful management-- both via selection of appropriate therapeutic goals and identification of barriers to care implementation.

- Medical complexity in CP can span multiple organ systems, and physiologic stability is essential for any successful treatment program.

- Neurocognitive impairments can affect patient and family ability to understand or implement a management plan. As such, formal evaluation of cognitive and communicative strengths and weaknesses is important[29]. Of note, verbal clarity does not necessarily reflect cognitive ability, and up to one quarter of children with severe dysarthria have normal or borderline cognition[12].
- Mental health diagnoses and environmental stressors (affecting the patient and/or caregivers) are also important determinants of participation in society, quality of life, and in ability to carry out a demanding treatment program[30].

As such, successful treatment frequently requires consideration of cognition, language, learning, and behavior with interdisciplinary support from families, medical providers, therapists, educators, and other members of the community.

Pharmacologic Treatment Options

Systemic medications remain a mainstay of medical treatment for individuals with CP. To date, treatment selection remains largely symptom-driven and directed at specific movement disorders (Table 2), though hints at targeted, pathogenesis-based treatments are beginning to emerge. Systemic medications remain commonly used for generalized hypertonia (e.g. oral baclofen, diazepam and tizanadine for spasticity) but are side effect-limited. Further, normalization of tone does not necessarily normalize motor control [31]. Medical management of dystonia and choreoathetosis seen in dyskinetic CP is particularly challenging-- mechanisms are not wellunderstood, and medications used have little supporting evidence and variable efficacy[32].

Pathogenesis-targeted treatments offer a possible road to personalized medicine. Evidence toward targeted treatments is building along two directions: 1) correction of the underlying genetic/metabolic disorder, and 2) identification of idiosyncratic (either positive or negative) treatment responses to specific modalities for particular etiologies. Gene-modifying and metabolic therapies are rapidly evolving and beginning to show the capacity to modify the natural history of specific genetic movement disorders (see [9,33] for further discussion). Currently detailed phenotypic descriptions including response to treatment are available for many genetic etiologies (Table 3), which can be helpful for “tailoring” treatment regimens.

Targeted injections/infusions also warrant discussion. For example, for severe refractory spasticity or dystonia, intrathecal baclofen is often beneficial, especially when goals include improved comfort and ease of care. For focal/segmental spasticity, local botulinum toxin injection is a widely-used approach [34]. Botulinum injections reliably decrease tone and strength in the targeted muscle and when used judiciously can help support gait and function in children without fixed contractures. However, efficacy declines with age, and muscle atrophy accumulates with repeated doses-- limiting long-term usage[35].

Nonpharmacologic Therapy-based, Surgical, and Combination Management

Physical therapy and other non-pharmacologic treatments have long been cornerstones of habilitative and rehabilitative care in CP. A wide range of nonpharmacologic treatments are available, and treatment approaches are constantly evolving. Again, heterogeneity in populations and in techniques used has limited high-quality study, and there is little consensus on optimal treatment paradigms. That said, large therapeutic trials now offer convincing evidence for or against some specific therapies (Table 4), and useful principles are beginning to emerge (Box 1)[36].

Selective dorsal rhizotomy (SDR) is a widely utilized neurosurgical procedure, which reduces spasticity primarily in the legs by interrupting the sensory component of the deep tendon reflex. It is most beneficial in children with spastic diplegia (without dystonia) commonly secondary to PVL, who have good antigravity strength and selective motor control, who are able to participate in prolonged rehabilitation, and for whom less invasive interventions have not been beneficial[37]. Transient short-term side-effects may include sensory or bowel/bladder symptoms, but long-term complications are rare[38].

Deep brain stimulation (DBS) involves implanting electrodes within deep gray structures including the globus pallidus. Stimulation is controlled by a generator placed subcutaneously in the upper chest. DBS has been very successful for patients with *DYT-1*-related genetic dystonia; more generally, outcome data in patients with dyskinetic CP is limited and heterogeneous[39]. Genetic diagnosis (Table 3) and movement disorder characteristics (dystonia vs. chorea and/or athetosis) likely contribute to this variability[40].

Surgical orthopedic interventions are commonly employed for the secondary musculoskeletal pathology that develops over time in many children with CP. Surgical procedures include tendon lengthening to correct contractures, tendon transfers to reestablish muscle balance, rotational osteotomies for torsional deformities, and spine, hip, and/or foot stabilization. Careful pre-op planning from a medical perspective can be of benefit[41], including optimization of nutritional status and tone. Controlled trials of specific orthopedic surgical interventions, including single-event multilevel surgery (SEMLS, in which multiple joints are operated on in one procedure to reduce burden of hospitalization/recovery) are needed to fully demonstrate efficacy[42]. In regard to scoliosis surgery, while full consensus has not been reached[43], the recommendation leans toward operating at curves less than 90 degrees[44,45].

Adjusting Treatment and Evaluation of Outcome

As most treatment is symptomatic and marked by variable efficacy and side effect profiles (as well as possible rebound effects), the authors use the following principles (with guidance from[46]): -Acknowledge uncertainty about benefit and potential side effects when initiating treatment. Safety and efficacy studies have not been formally evaluated for many medications (particularly for children). Further, even routine therapy sessions may not be well-tolerated for some people. A majority of children and young adults report pain during

physical or occupational therapy, and parents of children with CP report that stretching is the most frequent routinely painful activity[47]. Therapeutic standing, assisted walking, assisted sitting, and splint use have also been reported as painful [48].

- Identify clear, measurable goals **prior** to initiating titration. If goals are not reachable with tolerable side effects, all involved should be willing to consider discontinuing a therapeutic trial if side effects outweigh benefits.
- Start with a low dose (with the initial dose at a time and place with careful observation to monitor for idiosyncratic reactions), increase slowly and avoid abrupt discontinuation.
- Objective outcome measures (Table 5) remain somewhat limited at present. While subjective, patient and parent report an important measure of efficacy. Therapists are important allies in this regard-- depending on the situation, their functional measurements may be best used either as blinded observations of treatment effects or an integrated component of multidisciplinary assessment.
- Providers must be thoughtful when treatment plans do not produce the expected results (Table 6). Unexpected treatment failure should prompt re-examination of clinical status, benefit/risk profiles of applicable treatments, and diagnoses.
- A broad spectrum of neurorestorative therapies are under pre-clinical investigation but are currently unproven[49]. Despite biologic plausibility, many clinical trials have suffered from a lack of a standardized, systematic approach--making comparison difficult[50]. However, patients and families often turn to experimental complementary, alternative, or integrative medical therapies when conventional therapies do not provide the results desired-- and enthusiasm has outpaced effective regulation. For example, cell-based (stem cell) clinics have arisen promising dramatic benefits beyond those suggested by current evidence. Treatments are generally very costly to families, and there have been several reports of serious adverse effects (including death) following treatments[51]. As such, patients, families and clinicians should discuss evidence of benefits/risks (as well as, often, lack of evidence) when considering using unproven treatments[52].

Treatment over the Lifespan

Functional status and adaptive needs change over the lifespan for individuals with CP. During childhood, the primary focus is placed on optimizing developmental gains in motor and cognitive ability. Motor abilities for children unable to ambulate independently can peak at age 7–8 and may decline during adolescence[53]. The transition to adulthood is also a particularly challenging period, and establishing independence and maintaining health/quality of life are key goals for adolescents and adults with CP.

Surveillance guidelines reflect these changing needs. While some considerations such as pain should be evaluated at every visit[54], other surveillance (such as screening for orthopedic deformity) requires a more complex schedule [55] based on age and functional status. Motor,

cognitive, language, and academic development as well as vision, hearing, and mental health should be closely monitored in all children with CP[29].

Community participation remains a significant challenge for adolescents and adults with CP. In one cohort, 79% of respondents aged 18–30 finished 12 or more years of school, but only 23% held a driver's license or were competitively employed (though continued schooling and lack of available work were included in reasons for unemployment). Only 5% were married, though 74% reported having friends. Roughly one-third of respondents regularly strolled around the neighborhood or engaged in sports[56]. That said, one study reported that over 90% of children and adults with CP reported overall life satisfaction, and even more predicted overall life satisfaction 5 years in the future [56].

Conclusion / Summary

CP is a complex, heterogeneous disorder. Successful management requires accurate assessment and treatment of both motor and non-motor manifestations. Prenatal and perinatal neuroprotective strategies offer the promise of decreasing prevalence and severity of CP, but neurorestorative strategies remain experimental. Advanced imaging modalities and genetic testing frequently provide etiologic clarity and are proving increasingly helpful for guiding management plans.

In the setting of inter-individual heterogeneity, individualized treatment plans with close monitoring of effects and tolerability represent the gold standard of care. Well-designed, controlled studies are beginning to build evidence for and against specific therapies to achieve specific goals. To date, evidence for medical and surgical treatments (e.g. selective dorsal rhizotomy) largely relates to body structure-level endpoints (e.g., tone reduction), while therapy trials have largely targeted functional (activity level-based) outcomes. As such, no treatment to date has high-quality evidence on both the body structure level and on the functional level[36]. Bridging this divide should be a priority for therapeutic trials in order to maximize individual participation at home, in the community, and in the workplace.

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Box 1:

Principles of therapy in CP.

Types of therapy used in management of CP are too numerous and varied to list. The following principles reflect existing evidence as well as the authors' approach when prescribing courses of therapy.

Dose of therapy. The "dose" may be more important than the method used for some therapies. As an example, constraint-induced movement therapy (CIMT) is a form of intensive therapy for individuals with hemiplegic CP in which the less-impaired hand is restrained for (typically) 14 days during which focused repetitive tasks and shaping activities are performed for 6 hours per day in order to overcome "learned [or] developmental non-use"[79]. Comparisons between studies suggest that more therapy (up to 90 hours over 15 days) yields more and longer-lasting benefits for individuals with hemiplegic CP. However, for both CIMT and bimanual therapy (in which coordinated use of both hands together), dose/response relationships are seen[80,81]. Combination with adjuncts ranging from functional electrical stimulation[82] to transcranial magnetic stimulation[83] or virtual reality programs[84] also shows promise. There remains marked variability in individual outcomes.

In practice, the authors assume that there are diminishing returns for ever higher doses of therapy, but optimal doses, durations, and intensities (high-intensity "bursts" vs. distributed over time) are generally not known[79]. As such, we recommend setting realistic, measurable, individualized goals to be accomplished over defined time periods with periodic assessment of therapeutic gains vs. burden of increased care.

Types of therapy. Evidence is building to support use of specific therapies for specific indications and against efficacy for others[36]. Again using the example of hemiplegic CP, intensive CIMT and bimanual therapy both have evidence supporting efficacy[80,81], and neurodevelopmental therapy has evidence against its efficacy. However, evidence for many treatments is limited and inadequate to make strong recommendations for or against them.

One emerging principle is that "top/down" task-oriented therapy goals may be more effective than "bottom-up" goals focused on basic functions or prerequisite skills[80].

Timing of therapy:

Transition points. Permanent (e.g. surgery) and long-lasting (e.g. botulinum toxin therapy) treatments intuitively alter biomechanics (e.g. decreased muscle strength in the period following multi-level orthopedic surgery [85]). As such, guidelines emphasize the importance of intensive postsurgical (and post-botulinum [80]) therapy [86] throughout the recovery period, while specific evidence-based protocols are only beginning to emerge [85].

Early intervention. Much current policy and medical practice prioritizes early detection of CP and early initiation of therapy. Evidence has emerged to support benefits of some early interventions [87]. However, early childhood brain plasticity is likely better considered a dynamic period of rapid change than an open-and-shut critical period [88]. While early diagnosis and therapy may represent an opportunity, submaximal early intervention should not be seen as a reason to lose hope.

Synopsis

Cerebral palsy is the most common cause of childhood motor disability, affecting 2–3/1000 children worldwide. Clinical abnormalities in tone, posture, and movement are the result of brain dysgenesis or injury early in life, and impairment varies in type, distribution, and in severity. The underlying brain disorder may also lead to other associated neurologic and systemic impairments. Variability in functional impairments, which can change during development, necessitates an individualized treatment plan. Treatment options are primarily symptomatic and directed toward optimizing independence, function, and/or ease of care-- while limiting side effects. New promising disease--preventing and modifying treatments are emerging.

Key Points

- In cases of cerebral palsy, early brain injury or dysgenesis results in a wide variety of movement disorders as well as associated non-motor impairments.
- Successful management to optimize independence, participation, and quality of life requires individualized multidisciplinary care throughout the lifespan.
- Many symptomatic treatments of specific manifestations are available and widely used, including physical therapy, medications, and neurosurgical and orthopedic interventions. Most lack strong evidence of efficacy. For any intervention, open dialogue between patients, families, and providers regarding therapeutic options and expected benefits and potential side effects is essential.
- Modern imaging and genetic investigations have significantly advanced understanding of etiology and can improve treatment in some individual cases.
- It is important to identify co-morbid medical, cognitive, and mental health disorders as these can contribute significantly to outcome.
- Neuroprotective therapies are employed to prevent or mitigate the effects of early brain injury.

Neurorestorative therapies are also being developed and may help ameliorate long-term sequelae.

Table 1:

Common treatment goals.

Treatment goals are highly personal but are often constrained by an individual's current level of gross motor functioning. The GMFCS E&R is a commonly used classification framework for individuals with CP. Note that goals should reflect thoughtful individualized discussion and do not neatly fall on a rigid spectrum -- for example, chronic pain is common for individuals with CP across the spectrum of motor impairment (GMFCS E&R I-V; [17]).

Gross Motor Functional Classification System, Extended and Revised (GMFCS E&R; from[57])		**Spectrum of treatment goals**
I	Walks without Limitations	***Less motor impairment*** Increased motor precision (e.g. in writing, driving) Improved gait efficiency/community ambulation Improved gait stability/household ambulation More independent self-care (e.g. dressing, bathing) Improved standing/exercise ambulation More stable sitting Prevention of orthopedic deformity Ease of care Comfort ***More motor impairment***
II	Walks with Limitations	
III	Walks Using a Hand-Held Mobility Device	
IV	Self-Mobility with Limitations; May Use Powered Mobility	
V	Transported in a Manual Wheelchair	

Table 2:
Commonly used medications for the treatment of specific movement disorders in CP.

Green squares indicate first-line medications for specific movement disorders. Yellow squares indicate additional medications that may be considered for the movement disorder shown. Recommendations reflect existing evidence [58] as well as the authors' clinical experience.

MEDICATION	Dose [46]	Frequency	SPASTICITY	DYSTONIA	CHOREOATHETOSIS	MYOCLONUS	TREMOR	Notes
Baclofen	**0.2–2 mg/kg/day (up to 120 mg/day)**	**BID-TID**	[59]					
Benzodiazepines								
Diazepam	**0.5–7.5 mg/dose**	**2–4x daily PRN**	[60]					Beneficial perioperative orthopedic surgery and for nighttime spasms.
Clonazepam	**Start 0.02 mg/kg/day**	**BID**						Beneficial for exaggerated startle
Lorazepam								Useful for associated anxiety
Other antiepileptics								
Valproic Acid	**Start 10 mg/kg/day**	**BID-TID**						
Levetiracetam	**2.5–10 mg/kg/day**	**BID**						
Carbamazepine	**5–20 mg/kg/day**	**BID-TID**						
Gabapentin	**Start 5 mg/kg/dose**	**BID-TID**		[61]				Also helpful for neuropathic pain
Dopaminergic medications								
Levodopa/carbidopa	**0.5–2 mg/kg/day**	**BID-TID**						
Other oral medications								
Tizanidine Hydrochloride	**4–24 mg/day**	**TID**						
Dantrolene Sodium	**0.5–10 mg/kg/day**	**BID**						
Tetrabenazine	**12.5 to 25 mg**	**BID-TID**						
Trihexyphenidyl	**Start 0.25 mg/day**	**BID-TID**						

MEDICATION	Dose [46]	Frequency	SPASTICITY	DYSTONIA	CHOREOATHETOSIS	MYOCLONUS	TREMOR	Notes
Primidone								
Propranolol								
Cannabinoids (e.g. CBD, THC)	**Not established**							**Insufficient data for tone management** [62]
Injected/infused medications								
Botulinum toxin A and B	**Variable**	**q3–6 months as required**	[36]					Local/ segmental treatment
Intrathecal baclofen pump (ITB)	**50–1600 micrograms/day**	**Higher doses for dystonia**						

Abbreviations: BID: twice daily; TID: Three times daily; CBD: cannabidiol; THC: tetrahydrocannabinol

Table 3:
Treatment considerations for selected childhood-onset movement disorders.

As cohorts of individuals with monogenic movement disorders are increasingly well-described in the literature, descriptions emerge of the extent of the neurologic phenotype as well as response to specific interventions. It should be noted that most evidence along these lines is at the case series level with a few small randomized controlled trials.

Gene and corresponding condition	Description of movement disorder	Treatment considerations
***TOR1A*: Early-Onset Isolated Dystonia (DYT1)**	Isolated dystonia typically beginning distally in middle childhood frequently progressing over months to years sparing the larynx and neck [63]	Oral/injected medication is similar to that in dystonia more broadly. GPi DBS often produces sustained efficacy for refractory cases [64]
***GCH1*: Autosomal Dominant Dopa-Responsive Dystonia (DYT5a)**[65]	Classically with diurnally-varying childhood-onset dystonia beginning distally with gradual progression to generalized dystonia with pyramidal symptoms though an expanding recognized phenotypic spectrum	Typically dramatic and sustained response to relatively low doses of orally administered levodopa (20–30 mg/kg/day)
***SGCE*: Myoclonus-dystonia (DYT11)**	Myoclonus frequently with focal/segmental dystonia beginning in childhood with clinical course ranging from spontaneous remission to gradual progression[66]	Response to many therapies has been reported including benzodiazepines, antiepileptic drugs, L-dopa, zolpidem, and VIM or GPi DBS [67]
***GNAO1*-related movement disorder**	Episodic choreoathetotic movement disorder. Early hypotonia with later dystonia is common. Frequently progressive motor and cognitive deficits [68]	Hyperkinetic movements can be very treatment-resistant. Topiramate has been suggested as first-line for chorea[69]; cases have also reported response to tetrabenazine, GPi DBS[68], or intrathecal baclofen[70]
***GCDH*: Glutaric Aciduria Type 1**[71]	Stepwise motor decline during early childhood (prior to age 3) with development of dystonia, axial hypotonia, and later rigid parkinsonism	Frequently preventable with oral carnitine supplementation early in life. However, symptoms that do arise can be very refractory with little evidence of benefit for treatments beyond baclofen (oral or intrathecal) and benzodiazepines[72]. Response to DBS has been discouraging to date [73]
***DDC*: Aromatic L-amino acid decarboxylase (AADC) deficiency**[74]	Variable degrees of (typically non-progressive) axial hypotonia with limb hypertonia; typical movement disorders include oculogyric crises, dystonia, or hypokinesia	Response to dopaminergic agents and pyridoxine have been reported, and trials are recommended, but data on efficacy is limited

Table 4:
Selected non-pharmacologic therapies used for symptomatic treatment of cerebral palsy.

Four commonly-used treatments are outlined here with summaries of evidence for specific indications as well as typical contraindications. Evidence is derived from a systematic review[36] and, as in that reference, uses the evidence alert traffic light system.

Treatment	**Major effects and level of evidence (Evidence alert traffic light system: Green light: "Effective, therefore do it"; Yellow light: "Measure: Uncertain effect"; Red light: "Ineffective; therefore don't do it")**[36]	Contraindications for use
Non-invasive treatments		
Serial casting	Improves range of motion at the ankles. Effects may be short-lived but may be functionally helpful in selected partially-ambulatory individuals. Other indications (e.g. use at knees) lack adequate evidence. Not physiologically plausible as a means of directly impacting spasticity	heterotopic ossification, bone fracture/ dislocation, occlusive venous/arterial disease[75]
Orthotics (e.g. ankle-foot orthotics, or AFOs)	Low-to-very low quality of evidence for/against benefit on gait parameters, limb function, or prevention of contracture.	insufficient voluntary dorsiflexion control, fixed equinus deformity, insufficient heel strike, hypertonic reflex foot activity, lack of ambulation[76]
Surgical treatments		
Selective Dorsal Rhizotomy (SDR)	Effective for reducing spasticity and improving gait kinematics Evidence for improved gross motor functioning but no evidence regarding translation to improved participation in activities	dystonia, ataxia, fixed contractures [77]
Single-event multilevel surgery with associated therapy	Low-quality supporting evidence of improved long-term functional mobility	severe weakness, uncontrolled spasticity or dystonia, progressive neurologic disorder, inability to perform postoperative rehabilitation[78]

Table 5:

Examples of Assessments in CP.

Many instruments are used to measure and quantify various aspects of motor functioning in CP. This table is not exhaustive but illustrates types of assessments that may be used. In most cases, multiple types of assessments are used in a complementary manner (e.g. to comment on single-joint function, qualities of movement disorders present, and on effects on functional status).

Assessment type	Examples	Uses
Overall functioning	Gross Motor Function Measure[89] (gross motor) WeeFIM[90] (self-care, mobility, cognition)	Global assessment of motor functioning
Segmental	Medical Research Council (muscle strength)[91] Modified Ashworth Scale (spasticity)[92] Tardieu (spasticity) [93]	Phenotyping, longitudinal monitoring, assessing treatment response
Movement disorder-specific	Hypertonia Assessment Tool[94] Barry-Albright Dystonia Scale[95] Scale for the Assessment and Rating of Ataxia [96]	
Infant evaluation	Prechtl's Assessment of General Movements[97] Hammersmith Infant Neurological Exam[98] Alberta Infant Motor Scale[99]	Early identification/diagnosis/risk stratification

Table 6:

Why Treatment Plans Can Fail.

Treatment plans, even when well-planned and well-coordinated, may fail to achieve expected results. Potential pitfalls are outlined here.

Reason	Comments/Examples
Incorrect/Incomplete diagnosis	Neuromuscular and neurometabolic disorders may initially appear similar to individuals with CP and only later demonstrate clear progressive decline. Within a diagnosis of CP, dystonia can easily be overshadowed by spasticity, which can lead to suboptimal medical management[100].
Insufficient treatment	There may be limitations from an insurance or psychosocial perspective
Inappropriate treatment	Children with dystonia are not good candidates for SDR[77].
Limited effective treatment options	Hyperkinetic movements are notoriously difficult to treat.
Overlooked comorbidity: pain, anxiety or mood	Patient/family motivation is critical for the therapies that are required to complement and make medical or surgical interventions successful
Patient expectations differ from provider goals	Benefit from intervention is typically limited. Realistic, concrete goals should be set jointly between providers and families, communicated clearly, and re-evaluated regularly.
Side-effects > benefits	Some surgical interventions, both orthopedic and neurosurgical, can produce unwanted reduction in motor strength. Paradoxically, spasticity can be useful in some situations for scaffolding postures[100].