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Gait analysis in children with cerebral palsy

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- Cerebral palsy (CP) children present complex and heterogeneous motor disorders that cause gait deviations.

- Clinical gait analysis (CGA) is needed to identify, understand and support the management of gait deviations in CP. CGA assesses a large amount of quantitative data concerning patients' gait characteristics, such as video, kinematics, kinetics, electromyography and plantar pressure data.

- Common gait deviations in CP can be grouped into the gait patterns of spastic hemiplegia (drop foot, equinus with different knee positions) and spastic diplegia (true equinus, jump, apparent equinus and crouch) to facilitate communication. However, gait deviations in CP tend to be a continuum of deviations rather than well delineated groups. To interpret CGA, it is necessary to link gait deviations to clinical impairments and to distinguish primary gait deviations from compensatory strategies.

- CGA does not tell us how to treat a CP patient, but can provide objective identification of gait deviations and further the understanding of gait deviations. Numerous treatment options are available to manage gait deviations in CP. Generally, treatments strive to limit secondary deformations, re-establish the lever arm function and preserve muscle strength.

- Additional roles of CGA are to better understand the effects of treatments on gait deviations.

Keywords: cerebral palsy; clinical gait analysis; gait deviations

Introduction

Cerebral palsy (CP) is the most frequent cause of motor disability among children in Europe representing 700 000 citizens.[1] The prevalence of CP in Europe has been stable over the last 30 years, and ranges between 1.5 and 3.0 cases per 1000 live births.[1] The motor disorders of individuals with CP are complex. They are related to primary deficits such as muscle spasticity, muscle weakness and loss of selective motor control, and secondary deficits such as muscle contractures and bony deformities. The main dysfunctions are related to motor disorders during posture and movement causing limitation in activities (e.g. walking). Around 75% of CP children are ambulatory.[2] The severity and type of impairments are variable in the CP population. Clinical classifications distinguish the topography of impairments (hemiplegia, diplegia, quadriplegia), motor disorders (spastic, athetotic, dystonic, hypotonic, ataxic and a mixed group) and functional capacities with the Gross Motor Function Classification System (GMFCS).[3]

Walking is essential for activities of daily living and social participation; therefore, it is often considered one of the most important activities in daily life.[4] Nowadays, due to the complexity of gait and especially pathological gait, clinical gait analysis (CGA) is generally used to identify, quantify and understand the deficits of a specific patient and is fully integrated into the clinical decision-making of patients with complex gait disorders.[5] Moissenet and Armand have proposed a simple algorithm for the management of a patient with complex gait disorders.[6] This algorithm has three steps: identify gait deviations; understand gait deviations by linking them with clinical impairments; and choose the best therapeutic option.

Based on these three steps, the aim of this paper is to present 1) methods to analyse gait and identify gait deviations in CP; 2) gait patterns and gait deviations in CP and their interpretation; and 3) an overview of gait deviations management in CP.

Clinical gait analysis

Clinical gait analysis (CGA) aims to determine what is causing a patient to walk in the way he/she does. To reach this aim, CGA collects and analyses a large amount of quantitative data concerning the gait characteristics of a patient. In fact, CGA provides detailed information on four main types of data recorded simultaneously: spatiotemporal, kinematics, kinetics and electromyography data.

Fig. 1 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy (video stills).

During the same session, standardised clinical videos are recorded with numerical video cameras generally synchronised with the opto-electronic system (Fig. 1).

In addition, a standardised physical examination of the lower limbs is performed to measure anthropometry, passive range of motion, muscle force and muscle spasticity.[7]

Gait can be separated into cycles. A gait cycle starts at the instant where one foot touches the floor and stops as the same foot comes into contact for the next step. It can be divided in different phases: stance phase and swing phase. Stance phase can be also sub-divided into sub-phases: first double support (both feet in contact with the floor), single support (one foot in contact with the floor) and second double support. Based on the gait cycle, temporal and spatial characteristics of gait can be calculated: walking speed, cadence, stride length, step length, step width, duration of the stance and swing phases (Fig. 2).

Kinematic data correspond to the movement of the body described in CGA by the angular variations of the different joints/segments: ankle, knee, hip, pelvis and trunk. The raw data are measured by opto-electronic systems from the position of passive (or active) reflective markers attached directly on the skin of the patient during CGA. These markers are fixed on the patient in an accurate standardised position relative to anatomical or technical landmarks.[8] These positions are dependent on the model used to compute the kinematics. The most used models in the clinical field are the conventional gait models such as 'Plug-InGait'.[9] More advanced models and methods have been developed by different research teams in order to be more accurate. For example, calibration techniques[10] or foot models[11] have been developed; standardisation of joint definitions have been proposed.[12]

The model then permits the computation of joint angles (the angle between two adjacent segments in a specific plane). For this, the rotation around mobile axis (named Euler angles) are generally used to decompose the three-dimensional rotations in three successive elementary rotations. The three common planes used in description of the gait patterns are: the sagittal plane for flexion-extension movements; the frontal plane for adduction-abduction movements; and the transverse plane for internal-external rotations (Fig. 3).

Dynamic or kinetic data describe the forces applied by the patient during his/her gait.[13] This information corresponds to the ground-reaction forces, joint moments and powers of each joint. For this computation, data measured by force-plate(s) embedded in the ground of the gait laboratory are used. Using this information, it is possible to quantify ground-reaction force, which is the force exerted by the ground on the foot. With the addition of inertial parameters and kinematic data, it is possible to calculate by inverse dynamics the joint moments and joint powers applied at each joint during gait (Fig. 4).

Parameters	Left side	Right side	Normal
Gait speed (m/s)	0.88 ± 0.05	0.88 ± 0.05	1.1-1.3
Cadence (step/min)	107.36 ± 3.45	107.42 ± 3.49	110-130
Step length (m)	0.51 ± 0.02	0.47 ± 0.03	0.65-0.75
Step width (m)	0.14 ± 0.03	0.15 ± 0.03	0.05-0.15
Stance phase (%)	60.59 ± 2.04	62.42 ± 1.64	55-65
Step time (s)	0.56 ± 0.03	0.56 ± 0.02	0.45-0.55
Cycle time (s)	1.12 ± 0.04	1.12 ± 0.04	0.9-1.1
Cycle length (m)	0.99 ± 0.03	0.98 ± 0.03	1.4-1.6
Single support (%)	37.54 ± 1.36	39.33 ± 1.36	40-50
Double support (%)	23.05 ± 2.88	23.1 ± 2.89	10-20

Fig. 2 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: spatiotemporal parameters.
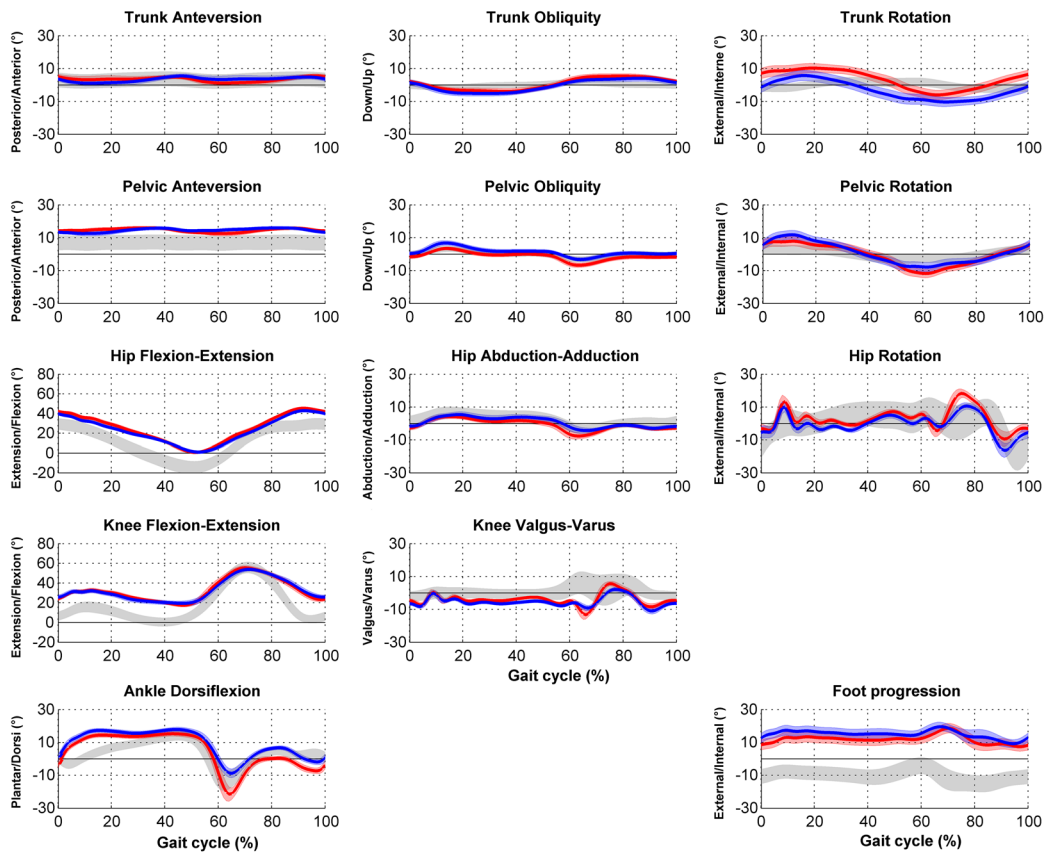
Fig. 3 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: kinematics.
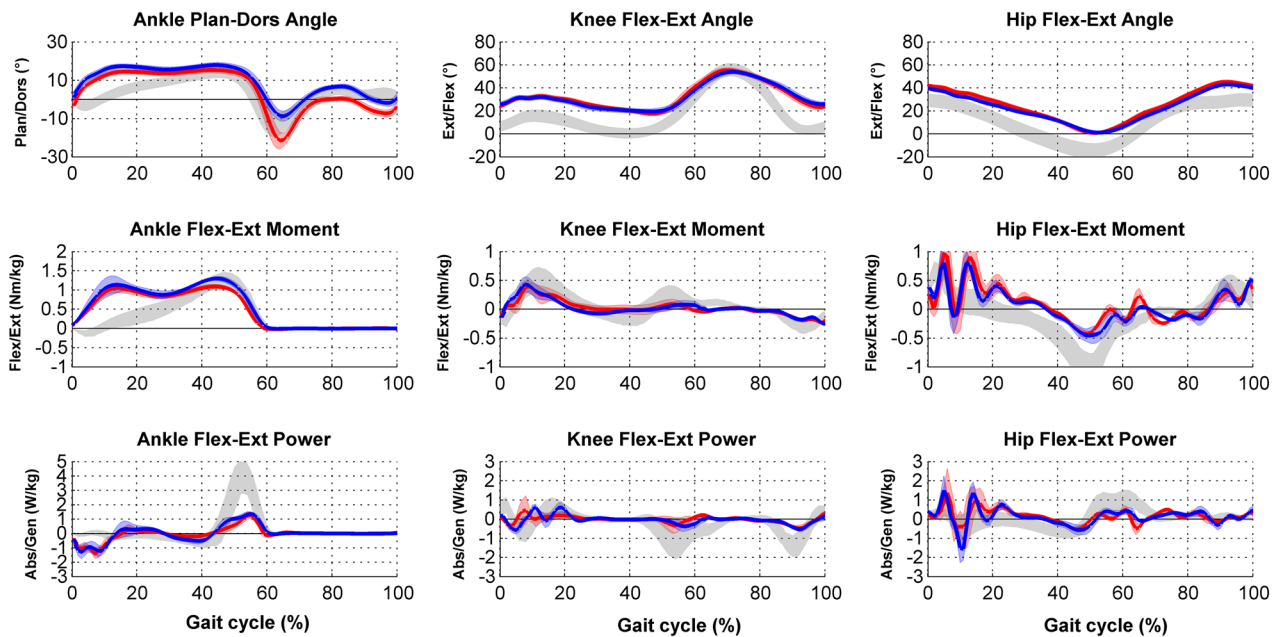

Fig. 4 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: kinetics.
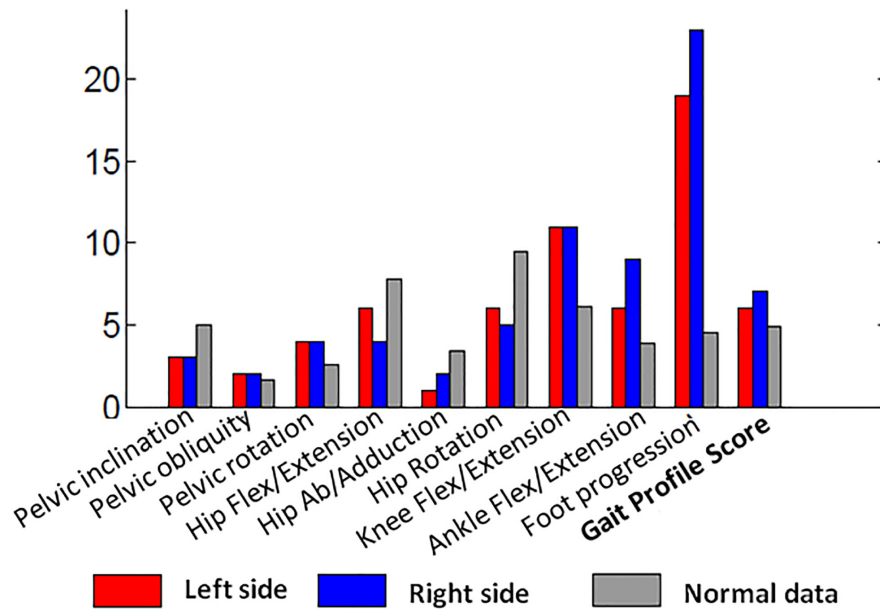
Fig. 5 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: gait scores.

Based on kinematic data, gait scores such as Gait Profile Scores and Movement Analysis Profile[14] or Gait Deviation Index[15] can be computed to summarise gait deviations (Fig. 5)

Electromyography (EMG) data show the timing and intensity of the muscle activity (Fig. 6). Thus, it is possible to describe the timing of the activity (such as lacking, delayed or permanent activity), co-contraction periods and also muscle spasticity during gait.[16] In CGA, surface electrodes are generally used with specific procedures recommended by the SENIAM.[17]

Moreover, during a CGA, it is also possible to record pedobarography measures. These data give information about contact of the feet (without shoes) with the ground during stance phase and how plantar surface pressures are distributed (Fig. 7).

Major limitations of CGA data are linked to external measurements used to estimate the movement of internal structures. These limitations include: marker placement on anatomical landmarks; skin movement (soft tissue artefacts);[18] definition of joint centres;[19] definition of anatomical axis respecting anatomical and clinical reality; and deformation of the segments (as for the foot[20]). However, CGA allows identification of gait deviations and allows further understanding of gait deviations.

To go deeper in the understanding of gait deviations, it is now technically possible to use personalised (neuro)-musculoskeletal modelling by merging data from medical imaging, CGA and clinical evaluation. Such modelling permits the simulation and testing of different hypotheses of the causes of gait deviations of a specific patient and to visualise the effect of treatments of impairments (e.g. increase

length of a muscle, correction of bony deformity).[21] Currently, the major clinical limitations of this approach are the validation of the models used and the time needed to build an accurate model of a specific patient.

Gait deviations in cerebral palsy

A large variety of gait deviations can be observed in CP patients. Classification systems to group common gait deviations in gait patterns are often used to facilitate communication[22] but are a simplification of the reality. In CP patients, we can observe a continuum between the different classes (no strict boundaries). Some gait deviations or combinations of gait deviations are not described in the classification systems. Therefore, a more accurate definition of gait deviations and related impairments is often needed to have a precise understanding of gait deviations and to better guide treatments using CGA results. The first part of this section describes the main gait patterns observed in CP. The second part describes the main gait deviations with related impairments.

Gait patterns

Classifications in gait patterns are generally different for unilateral spastic CP and for bilateral spastic CP. Most of the classifications are based the observation of the kinematics of the sagittal plane. Impairments and typical treatments are often associated with the gait patterns.

Gait patterns in unilateral spastic CP

The first pattern classification for unilateral spastic CP was proposed by Winters et al.[23] Four groups of gait patterns were identified based on observation of kinematic data in the sagittal plane (Fig. 8).
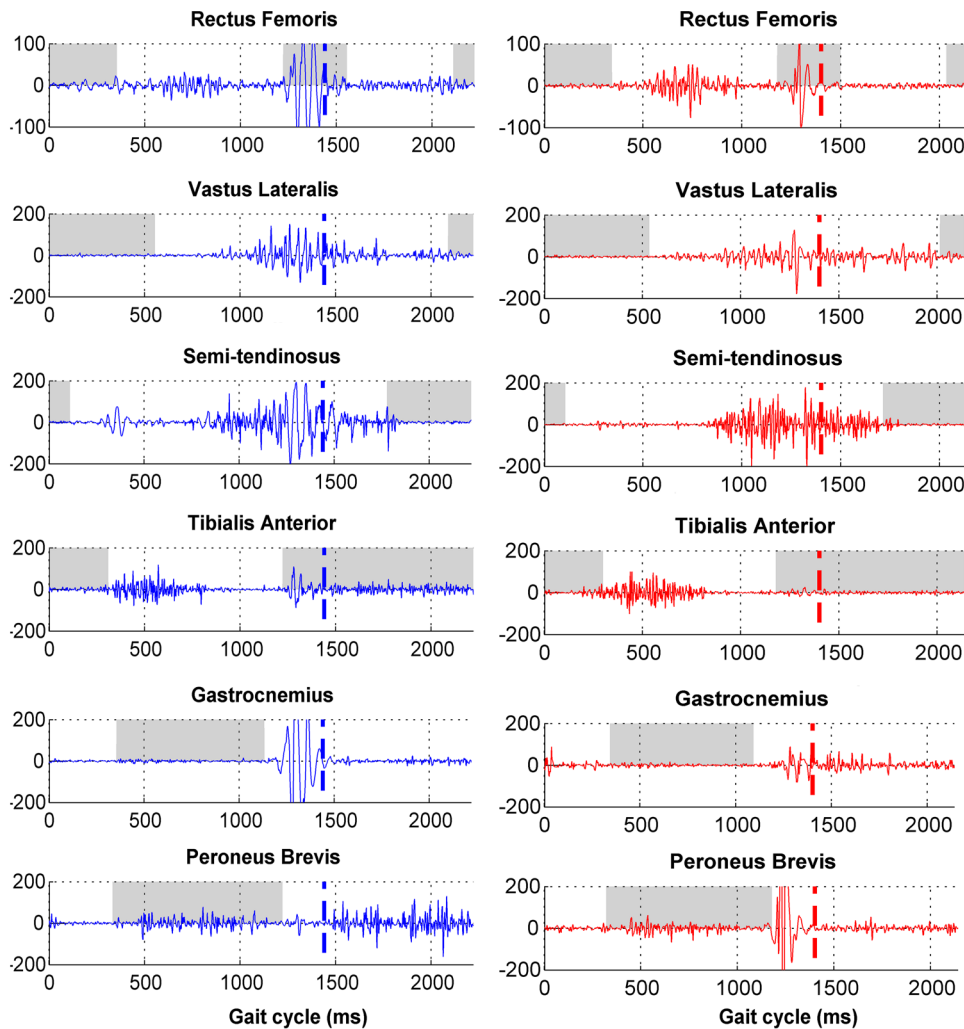
451

Fig. 6 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: electromyography.

There is progression of severity of impairments in the four groups and a progression of impairments from distal to proximal (from ankle to pelvis). Group I's main characteristic is foot drop during swing phase resulting in a lack of first rocker at initial contact. The impairments associated with group I are weakness or underactivity of the anterior tibial muscle relative to overactivity of the gastrocnemius and soleus muscles. Group II's gait pattern presents a drop foot in swing phase and a permanent plantarflexion in stance phase. Knee hyperextension is associated with this pattern. The impairments associated with group II are static or dynamic contractures of the gastrocnemius and soleus muscles. In addition to deviations of groups I and II, patients in group III have a limited flexion of the knee during the swing phase, a hyperflexion of the hip and an increased lumbar lordosis. Group IV presents all the deviations of groups I to III, plus restricted motion at the hip and knee. Patients compensate for limited motion of the hip by increasing pelvic lordosis during the terminal stance phase.

Winters' classification was refined by Rodda and Graham in 2001 to include patients with hyperextension at the knee and transverse plane deviations at the hip.[24] A schematic of gait patterns associated with possible treatments was provided by the authors (Fig. 8). Other classifications exist in the literature but have less notoriety. Some are based on subjective group placement and others on cluster analysis.[22]

Gait patterns in bilateral spastic CP
Based on Sutherland's classification of gait abnormalities of the knee in CP,[25] Rodda and Graham[24] have proposed a classification for patients with bilateral spastic CP based on sagittal plane kinematics considering the ankle, knee, hip and pelvis. Four main groups were identified: true equinus; jump gait; apparent equinus; and crouch gait (Fig. 9).
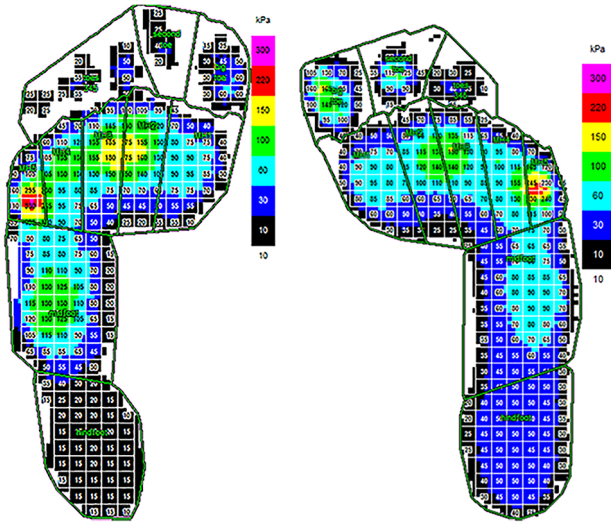
Fig. 7 Example of data in clinical gait analysis report for bilateral spastic cerebral palsy: plantar pressures.

True equinus is defined by the ankle in plantarflexion throughout stance phase and the hips and knees extended. The authors caution that the equinus can be masked by a knee recurvatum. The jump gait pattern is characterised by equinus at the ankle, flexion at knee and hip, anterior tilt and increased lumbar lordosis. Within this pattern, the stiff knee pattern described by Sutherland and Davids[25] is frequent. The apparent equinus pattern presents a normal range of dorsiflexion at the ankle, but the hip and knee are in excessive flexion throughout the stance phase leading to walking on the toes and giving an impression of equinus. Crouch gait is defined by excessive dorsiflexion at the ankle in combination with excessive flexion at the knee and hip joints (Fig. 3). The authors recognised that the two limbs can present different levels of involvement. Thus, they introduced an asymmetric gait group when the two limbs belong to two different classifications.[26] They also introduced a mild gait group for kinematics within normal limits in the sagittal plane.[26] These patterns established subjectively and qualitatively based on observation and experience were confirmed with statistical and clustering techniques without a priori.[27-29] However, numerous classifications of CP gait children were proposed involving different numbers of joints, segments, planes and clustering techniques leading to different numbers of clusters.[22]

Specific gait deviations in CP

Gait deviations in CP are a continuum of deviations rather than well delineated groups. Each CP patient is specific and generally presents with a list of impairments, with varying degrees of involvement and asymmetry between the two lower limbs. To support a precise understanding of gait deviations in CP, it is mandatory to understand the links between gait deviations and impairments. Recently, we defined kinematic gait deviations observed at the individual joint/segment/plane level with probable impairments resulting from these deviations (Table 1).[30]

Some of these gait deviations are also presented in the book by Perry.[31] We recognise that some kinematic deviations may come secondary to another problem or as compensation for another deviation. We also recognise that this table is not exhaustive and is based more on experience than evidence. Further studies are needed to confirm these links. Moreover, impairments are rarely isolated; therefore, gait deviations are often the results of combinations of impairments. This table considers only kinematic gait deviations and not kinetic, EMG and plantar pressure gait deviations.

Management of gait deviations in CP

Management of gait deviations in CP is complex and requires identification and understanding the gait deviations. Even if CGA supports these requirements, one can question the reliability of treatment decisions based on CGA.[32,33] As for many clinical diagnostic tools (e.g. imagery), CGA does not tell us how to treat a patient, but does help to identify and understand an impairment.[34] The choice of treatments depends on each physician.

Moreover, CGA helps to better understand the effects of treatments on gait deviations. This objective assessment of the patient's gait allows comparison of pre- and post-operative results. This information helps to determine if there is an improvement in patients' walking capacity. From this, the physician can critically analyse the therapeutic strategies as demonstrated by Vuillermin et al.[35] Before the introduction of CGA in their hospital in 1995, the prevalence of crouch gait was 25%. Thanks to CGA, they better understood the complexity of the walking patterns in CP patients. Thus, they realised how important the ankle-knee extension couple is. They stopped lengthening the Achilles tendon and they decreased considerably the prevalence of crouch gait in their CP patients.

Currently, there are no biological therapies to correct the brain lesion. Therefore, the management of CP children concerning motor disabilities is focussed on the primary consequences of the brain lesions (muscle spasticity and loss of selective motor control) and secondary consequences (muscle weaknesses, muscle contractures, bony deformities).

It is difficult to provide general considerations for the treatments of gait deviations in CP due to multiple
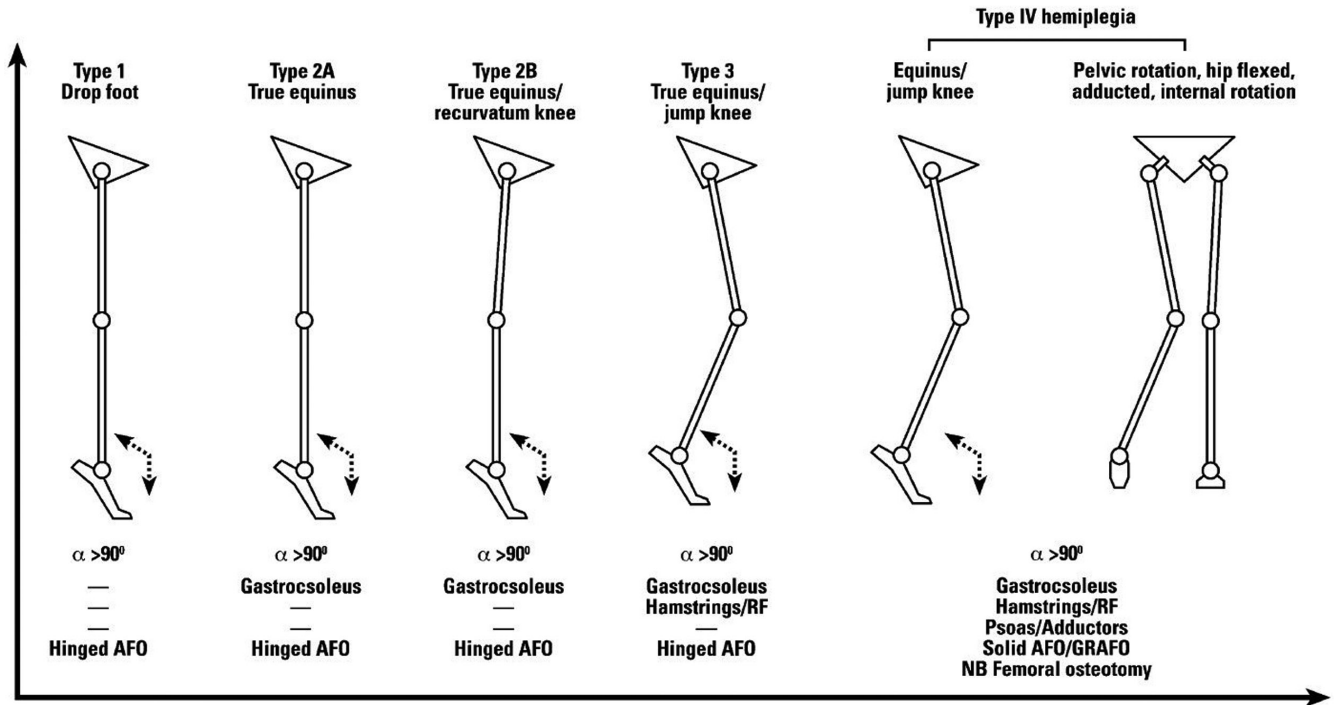
Type IV hemiplegia

| Type 1 Drop foot | Type 2A True equinus | Type 2B True equinus/ recurvatum knee | Type 3 True equinus/ jump knee | Equinus/ jump knee | Pelvic rotation, hip flexed, adducted, internal rotation |

$\alpha > 90^0$

—	Gastrocsoleus	Gastrocsoleus	Gastrocsoleus Hamstrings/RF	Gastrocsoleus Hamstrings/RF Psoas/Adductors
—	—	—	—	
—	—	—	—	
Hinged AFO	Hinged AFO	Hinged AFO	Hinged AFO	Solid AFO/GRAFO NB Femoral osteotomy

Fig. 8 Gait patterns and management algorithm for unilateral spastic cerebral palsy[24].

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Original publication: Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. *Eur J Neurol* 2001;8(suppl 5):98-108.

| True equinus | Jump knee | Apparent equinus | Crouch gait |

| $\alpha > 90^0$ | $\alpha > 90^0$ | $\alpha = 90^0$ | $\alpha < 90^0$ |

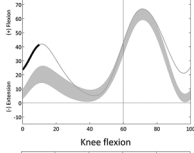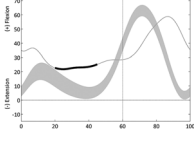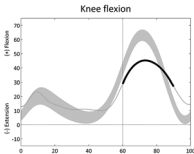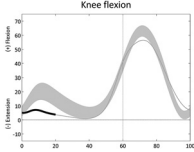| Gastroc | Gastroc Hamstrings/RF (Psoas) | (Gastroc) Hamstrings/RF Psoas | — Hamstrings/RF Psoas |
—			
—			
Hinged AFO	Hinged AFO	Solid AFO	GRAFO
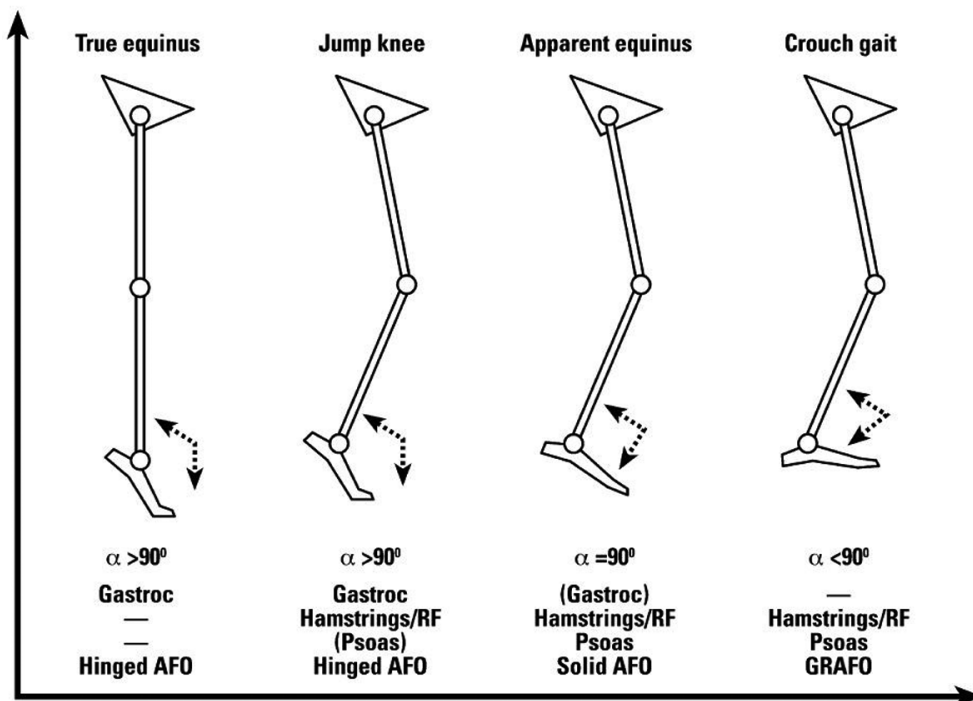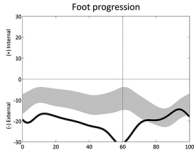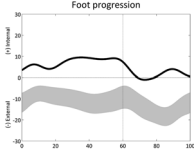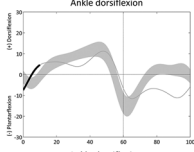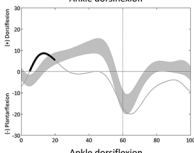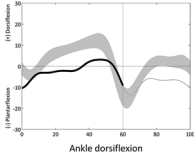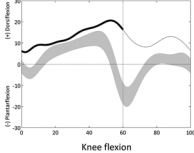
Fig. 9 Gait patterns and management algorithm for bilateral spastic cerebral palsy.[24]
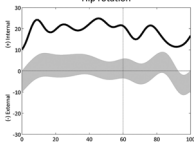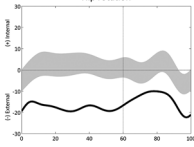
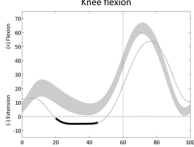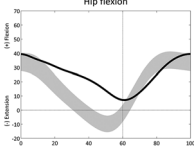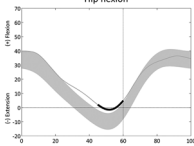Republished from *European Journal of Neurology* with kind permission of John Wiley and Sons.
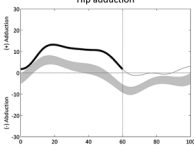Original publication: Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. *Eur J Neurol* 2001;8(suppl 5):98-108.

Table 1. Frequent single joint/plane kinematic deviations and associated impairments[30]

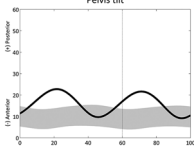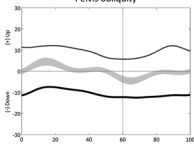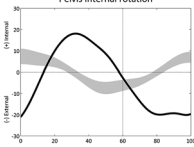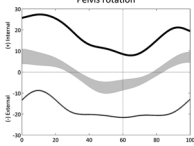| Gait deviations	Description	Impairments and coherent gait data	Confounding factor
Foot progression	External foot progression (stride)	• Increased external tibial torsion • Foot deformity o Increased ankle external rotation	• Sustained pelvic retraction • Increased hip external rotation

Foot progression	Internal foot progression (stride)	• Increased femoral anteversion
		  o Increased hip internal rotation
		• Reduced external tibial torsion
		• Foot deformity
		  o Increased internal ankle rotation
			• Sustained pelvic protraction

Ankle dorsiflexion	Absent ankle first rocker (first double support)	• Ankle dorsiflexors weakness or reduced selective motor control
		  o Excessive plantarflexion (swing)
		• Plantarflexors contracture or overactivity
		  o Excessive plantarflexion (stride)
			• Leg length discrepancy

Ankle dorsiflexion	Early ankle plantarflexion (early stance)	• Plantarflexors overactivity o Premature knee extension/hyperextension	• Leg length discrepancy or foot clearance problem on contralateral side • Increased knee flexion
Ankle dorsiflexion	Lack of ankle dorsiflexion (stance)	• Plantarflexors contracture or overactivity	
Ankle dorsiflexion	Increased ankle dorsiflexion (stance)	• Soleus weakness or soleus too long o Increased knee flexion in mid-stance	
Knee flexion	Increased knee flexion (first double support)	• Hamstring overactivity • Plantarflexors contracture or overactivity o Excessive ankle plantarflexion	

Knee flexion	Reduced knee extension (mid-stance)	• Hamstring contracture or overactivity
		• Knee fixed flexion deformity
		• Hip extensors or knee extensors weakness
		  o Excessive hip flexion
		• Ankle plantarflexors weakness
		  o Excessive ankle dorsiflexion
		• Ankle plantarflexors overactivity or contracture
		  o Excessive ankle plantarflexion
			• Cross-plane interactions (transverse - sagittal)
			  o External tibial torsion
			  o Increased femoral neck anteversion
			  o Foot deformity

Knee flexion	Reduced or delayed knee flexion (swing)	• Rectus femoris overactivity o Rectus femoris EMG activity in late stance or early swing • Stiff-knee gait, hamstring/rectus co-contraction	• Cross-plane interaction (transverse - sagittal) if retracted pelvis and hip externally rotated • Reduced push-off during ankle third rocker • Reduced speed
Knee flexion	Reduced knee flexion (loading response)	• Quadriceps weakness or patella pain o Reduced knee extensor moment (stance)	

(Continued)

Table 1. (Continued)

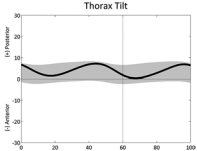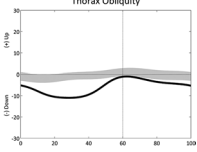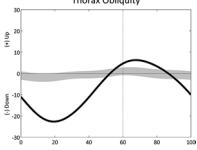| Gait deviations	Description	Impairments and coherent gait data	Confounding factor
Knee flexion (graph)	Knee hyper extension (mid-stance)	• Quadriceps weakness • Plantarflexors overactivity or contracture ○ Excessive ankle plantarflexion	
Hip flexion (graph)	Increased hip flexion (stride)	• Hip flexion contracture or overactivity ○ Anterior pelvic tilt, double bump • Hip extensor weakness	• Sustained anterior pelvic tilt (stride) • Excessive knee flexion • Leg length discrepancy

Hip flexion (graph)	Lack of hip extension (second double support)	• Hip flexor contracture or overactivity
		  ○ Anterior pelvic tilt, double bump
		• Hip reduced range of movement
		  ○ Anterior pelvic tilt, single bump
			• Leg length discrepancy

Hip adduction (graph)	Increased hip adduction (stance)	• Hip abductor weakness ○ Contralateral pelvic drop • Hip adductor contracture or overactivity	• Increased hip internal rotation • Pelvic retraction or obliquity • Leg length discrepancy
Hip rotation (graph)	Increased internal hip rotation (stride)	• Increased femoral neck anteversion • Excessive external tibial torsion	• Pelvic retraction on ipsilateral side
Hip rotation (graph)	Increased external hip rotation (stride)	• Reduced femoral anteversion • Reduced external tibial torsion	• Pelvic protraction on ipsilateral side • Obesity/large thighs • Foot deformity
Pelvis tilt (graph)	Pelvic tilt double bump (stride)	• Hip flexors contracture or overactivity ○ Reduced hip extension	
Pelvis obliquity (graph)	Pelvic obliquity down or up (stride)	• Leg length discrepancy ○ Excessive hip abduction • Adduction contracture	• Scoliosis • Hemiplegia
Pelvis internal rotation (graph)	Reversed pelvic rotation profile (stride)	• Overall weakness ○ Reversed hip adduction profile	
Pelvis rotation (graph)	Sustained pelvic pro- or re-traction (stride)	• Asymmetry in overall weakness	• Femur torsional deformity • Tibia torsional deformity • Hemiplegia

(Continued)

Table 1. (Continued)

Gait deviations	Description	Impairments and coherent gait data	Confounding factor
	Trunk tilt, double bump (stride)	• Overall weakness	
	Sustained trunk obliquity (stride)	• Hip pain (unilateral) • Abductors weakness (unilateral)	
	Excessive range of trunk obliquity, Trendelenburg (stride)	• Abductors weakness	

Table 1 is reprinted with permission from Nova Science Publishers. From: **Sangeux M, Armand S.** Kinematic deviations in children with cerebral palsy. In: Canavese F, Deslandes J, ed. *Orthopedic management of children with cerebral palsy: A comprehensive approach*. New York: Nova Science, 2015; 241-253.*The table has four columns;the first presents a graph of the deviation, the second describes the gait deviation observed, the third presents the impairments (•) and lists associated deviations (○).These confounding factors (•) are listed in the last column. One confounding factor appears several times: leg length discrepancy. Leg length discrepancy may be anatomical, when physical examination or medical imaging measures a true length difference between the legs, or functional, when the combination of joint angles during single leg support in stance phase results in altered leg length. The deviations are ordered from distal to proximal joints/segments and in the sagittal, coronal and transverse planes. In each graph, the light grey band presents the pattern of 35 typically developed children (the width equates to one standard deviation). The solid curve presents an example of altered kinematics, the part of interest is emphasised by a bolder line for the time instants of interest. Two pelvic deviations show two lines (one solid, one dashed) for the two sides of the same patient.*

gait deviations coming from different combinations of impairments. Moreover, even if the brain lesion is static, the chronic neurological impairments affect the development of muscles and bones. Therefore, CP is a progressive musculoskeletal pathology. To present the treatment options, we have divided the treatment considerations according types of treatments and gait patterns.

Main treatments in CP to address gait deviations

CP patients present numerous possible combinations of impairments that can be managed by numerous types of treatments. Physiotherapy is certainly the most common treatment in CP. The main objectives are to maintain range of motion with adequate muscle length, to preserve or restore strength, and to improve balance and co-ordination. Orthoses are also used frequently to manage gait deviations in CP, in particular ankle-foot orthoses.[36]

Spasticity management is addressed with a large spectrum of therapies that can have a focal or global effect and a permanent or reversible effect. Botulinim Neurotoxin-A (BoNT-A) is largely used to treat local spasticity.[37] BoNT-A injection should be reversible, but currently the long-term implications of denervation atrophy are not fully understood.[38] Gough et al recommends taking a cautious approach, particularly with ambulant children with spastic diplegic CP.[39] BoNT-A can also be used as a pre-operative test injection before muscle-tendon lengthening.[40] Hence, application of the drug is a possible step to test whether weakness causes deterioration in gait function prior to an operation. Selective dorsal rhizotomy has a permanent effect with a global impact on the lower limb. It is reserved for selected patients with diplegic CP.[41] Intrathecal baclofen has a general effect that can be reversible. This treatment is used to treat severe spasticity in CP.[42]

Bony deformities and muscle contractures can be addressed by surgery. One general principle is to preserve or restore lever arms. Abnormal lever arms result from bony deformities that lead to additional deformities and difficulties in efficiently-using forces exerted by the muscles.[43]

The growth of the skeleton associated with spasticity, reduced level of activity and weakness of muscles leads to muscle contractures. Muscle contractures should be evaluated and treated as conservatively as possible to prevent over lengthening and muscle weakness.

To avoid 'birthday syndrome', which is the correction of one deviation after another occurring with growth, the current treatment is to perform a single event multi-level surgery (SEMLS) to manage musculoskeletal deformities in CP.[44] SEMLS addresses all the impairments of both lower limbs in the same surgical procedure.[45-47]

Table 2. Main surgical treatments in cerebral palsy (CP) according gait patterns

Gait patterns Impairments	Treatment options	Comments
True equinus	- Gastrocnemius recession rather than tendo-Achilles lengthening	- Important to distinguish true equinus from apparent equinus - Excessive dorsiflexion can occur over time without surgical intervention, secondary to progressive weakness and increasing weight

Crouch gait	- Re-establish the lever arm function:
	  ○ Stabilise flat foot deformity to restore foot lever arm
	  ○ Correct torsional tibial deformity
	  ○ Patella advancement to reinforce the quadriceps
	(Reinforce the Achilles tendon with a floor reaction orthosis)
		- Caution with hamstring lengthening because CP patients can develop knee recurvatum and an anterior pelvic tilt

Stiff knee gait	- Rectus femoris transfer is indicated in patients functioning at GMFCS level I or II. (Patellar advancement should be considered when there is an important knee extension moment in stance)	- Rectus femoris transfer is not helpful in patients functioning at GMFCS level IV and some patients at level III, due to increased knee flexion in stance post-operatively
Torsional deformities	- Derotational osteotomy of tibia and/or femur	- Good correlation with tibial de-rotational osteotomy correction and the foot progression angle correction - Weak correlation between femoral derotational osteotomy correction and the foot progression angle correction - Soft tissue surgeries alone will not fully correct torsional deformities
Varus feet	- Tendon transfers to correct muscular forces that cause deformity	- Frequent deformities in cerebral palsy caused by tibialis anterior or tibialis posterior or a combination of both

GMFCS, gross motor function classification system

Main surgical treatments in CP according gait patterns

We chose to present the main surgical treatments according three common gait patterns in CP: true equinus,[48-50] crouch gait[51,52] and stiff knee gait;[51,52] and two impairments: torsional deformities[55-58] and varus feet[59,60] (Table 2).

Conclusion

Cerebral palsy children present complex gait deviations that evolve with growth. Having an in-depth understanding of these gait deviations is necessary to choose the most appropriate therapeutic approach. Therefore, CGA has become a fundamental part of the clinical management of gait deviations in CP patients. By using a precise quantification of a person's gait characteristics, CGA allows the identification of gait deviations and gives a better understanding of these gait deviations. Moreover, CGA has allowed better evaluation and understanding of the effects of treatments on gait deviations. Indeed, thanks to CGA, management of gait deviations in CP children has changed and continues to improve.

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