

Orthopedic management of spasticity in cerebral palsy

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Abstract

Introduction This article summarizes our experience with cerebral palsy. The primary and secondary deformities that occur with cerebral palsy are described, followed by a brief overview of the nature and role of gait analysis in the treatment of gait problems in cerebral palsy. The concept of lever-arm dysfunction is introduced.

Discussion Our current treatment program is then presented and subsequently illustrated by two case examples. Finally, an outcomes analysis of a group of patients with spastic diplegia treated with selective dorsal rhizotomy is presented to illustrate our current method of evaluating treatment outcomes and the need for team management in the treatment of this complex condition.

Keywords Cerebral palsy · Spasticity ·
Selective dorsal rhizotomy · Lever-arm dysfunction

Introduction

There has been enormous progress in the treatment of gait problems in children with cerebral palsy in the past 20 years. As such, a myriad of treatments are now available for these children that range from the standard orthopedic procedures to spasticity reduction via an intrathecal baclofen pump or selective dorsal rhizotomy. In the past,

when we treated these children, we started with a spastic child with orthopedic deformities and, as a result, walked poorly; we ended with the same spastic child who walked differently. This was because without modern technology, it was very difficult to assess the benefits or detriments of our interventions [6]. Now with the advent of gait analysis laboratories, functional assessment scales [10], patient satisfaction questionnaires, and energy cost analysis, we can assess the outcomes of treatment interventions more precisely [10]. Furthermore, third-party payers now are demanding that we do these assessments. Consequently, it is becoming increasingly clear that in the very near future, we are going to be held accountable for our treatment outcomes.

If we are going to offer these children consistent and optimal treatment, it should be apparent that we are going to have to operate under a new set of guidelines. The team of individuals who are going to treat a child with cerebral palsy must have:

1. knowledge of normal anatomy and physiology, particularly with reference to ambulation,
2. a good understanding of the functional pathology present in cerebral palsy,
3. realistic goals/objectives for treatment that are commonly shared by the patient, family, and others concerned with the child's welfare,
4. knowledge and ability to carry out any of the treatments that are needed,
5. a facility with the resources to do the necessary evaluations/treatments.

Because of its limited scope, this article cannot provide comprehensive knowledge in all areas. Rather, the goal is to provide an overview of a modern multispecialty manage-

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ment program with a focus on orthopedic surgical management of spasticity and its complications.

Attempts to improve ambulation in cerebral palsy must start with knowledge of normal gait. Normal gait is a complex topic in itself, but thanks to the work of pioneers like Vern Inman, Jacquelyn Perry, David Sutherland, and David Winter [7, 11, 14, 15, 17], it is now fairly well-understood. We will not review the principles of normal gait in this article. Because the treatment of cerebral palsy is based on the pathophysiology of cerebral palsy, our discussion will start there. Because once the reader gains this knowledge, an understanding of the appropriate treatment programs will logically follow.

Types of gait deviations in cerebral palsy

In a child with cerebral palsy, it is the central control system that is damaged. The neurological lesion may produce different tone abnormalities (spastic, athetoid or mixed). In a patient who has pure spasticity, only the corticospinal system is damaged; in athetoid cerebral palsy, only the basal ganglia and/or its connecting pathways are involved; and in a mixed pattern, both systems are injured.

Despite the fact that the effects of cerebral palsy are readily apparent at the periphery, i.e., the muscles and bones of the extremities, it is only the central control system that is damaged. The changes in length and/or structure that occur in the muscles and bones of the extremities are all secondary to the central nervous system lesion. In an individual with cerebral palsy, the primary injury (symptomatology due directly to damage to the central control system) will produce some or all of the following primary abnormalities:

1. loss of selective muscle control,
2. dependence on primitive reflex patterns for ambulation,
3. abnormal muscle tone,
4. relative imbalance between muscle agonists and antagonists,
5. deficient equilibrium reactions.

These primary problems cause secondary abnormalities that result from disordered growth. They develop over time as a child grows. Growth of bone occurs via epiphyseal plates, but it is the joint reaction forces acting on those bones that determine their ultimate shape. If those forces are appropriate, the development of the shape of the bone will follow a typical pattern. Infants are not born with adult bone alignment. Typical adult bone alignment develops primarily over the first 8 years of life. If the forces are atypical (due to spasticity or delays in gross motor function), the final shape of the bone will likely be distorted. In simple terms, bone growth follows the Star

Wars principle, “Let the force be with you!” Consequently, in conditions such as spastic diplegia, both remodeling of fetal bone alignment and forward modeling of the bone as it grows are abnormal. It is for these reasons that deformities such as hip subluxation, torsion of long bones, and foot deformities are commonly seen in this condition.

Muscle growth, on the other hand, is driven by stretch. It has been shown that in order for normal muscle growth to occur, 2 to 4 h of stretch per day are necessary [18]. Normally that stretch occurs when a child whose bones have grown during sleep, gets up, starts to run, and play. However, “normal play” demands good overall body balance, intact selective motor control, and muscles with normal elasticity (none of which are present in a child with spastic diplegia). Hence, contractures occur as longitudinal muscle growth fails to keep up with bone growth.

In cerebral palsy, gait abnormalities never occur in isolation. Rather, they are multiple and consist of primary anomalies (due to the damage to the central nervous system), secondary anomalies (from abnormal bone/muscle growth), and tertiary abnormalities. Tertiary abnormalities are those compensations that the individual uses to circumvent the primary and secondary abnormalities of gait. The tertiary abnormalities can be thought of as “coping responses”. For example, co-spasticity of the rectus femoris and hamstrings commonly produces a stiff knee in the swing phase of gait. This, in turn, leads to problems with foot clearance. Circumduction of the swinging limb, by abducting the hip, is a frequent compensation. The primary deviation of gait in this example is the rectus femoris and hamstring co-spasticity. The “coping response” is the circumduction. Much of the difficulty encountered in studying pathological gait involves the separation of the true pathology from these “coping responses”. However, good treatment demands their separation because to optimize the efficiency of gait, we need to correct the former and not interfere with the latter. The “coping responses” will disappear spontaneously when they are no longer required.

Gait analysis

In our opinion, dynamic gait analysis is required for the optimal treatment of problems relating to ambulation in cerebral palsy. Before surgery, gait analysis allows accurate dynamic assessment of the patient’s particular gait problems. An experienced gait interpreter uses the information to generate a problem list of pathologies responsible for the observed gait deviations. It is this list that is used to generate the treatment plan. Postsurgery gait analysis allows an accurate and objective assessment of outcome and a new baseline for future comparison [12]. Several

good, commercial, gait analysis systems are now available. They typically provide the user with three-dimensional kinematics and kinetics and dynamic electromyography [1].

Kinematics are those parameters used to describe the spatial movement of the body (e.g., body segment position, joint position, and joint motion) without consideration of the forces that cause the movement. Kinematics essentially tell us “what” is occurring at each of the major lower extremity joints, but not “why” it is happening. With experience, one can attribute certain kinematic patterns and deviations to specific pathologies. Comparison of pretreatment to postoperative kinematics findings indicates treatment effectiveness. For example, “Was treatment for crouch gait successful?” (Fig. 1).

Kinetics are those parameters used to describe the mechanisms that cause movement, e.g., ground reaction forces, joint moments, and joint powers. By utilizing kinetics, we can often determine why a particular gait deviation is occurring [2]. Therefore, information derived from the study of kinetics is especially useful to improve one’s knowledge of the pathogenesis of gait problems.

Many gait analysis centers assess energy efficiency by measuring oxygen consumption/cost during ambulation. This measurement of oxygen consumption/cost is an indicator of the overall severity of dysfunction [8, 16].

Lever-arm dysfunction

A lever-arm or moment-arm is best defined as a distance from a point to a force that is perpendicular to the line of action of that force. The force (measured in Newtons) times the length of the lever-arm (in meters) is equal to the

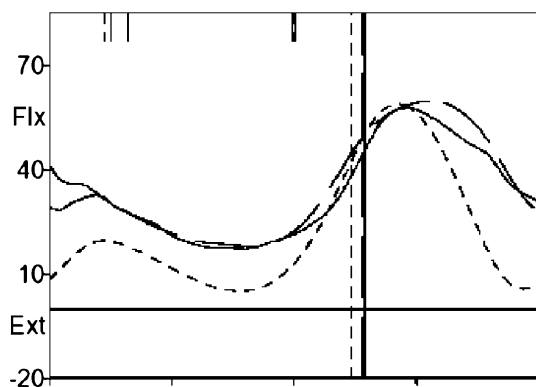


Fig. 1 Kinematic plot, knee flexion/extension sagittal plane. This plot represents one gait cycle from initial contact (left side of graph) to the next initial contact (right side of graph). Toe-off lines (vertical lines at about 65% of the gait cycle) separates stance phase to the left and swing phase to the right. Normal knee motion (short dash) shows nearly full extension in midstance. This patient’s data with diplegic cerebral palsy shows a mild to moderate crouch during stance phase and a lack of extension of the knee in preparation for initial contact in late swing phase for both the right (solid) and left (long dash)

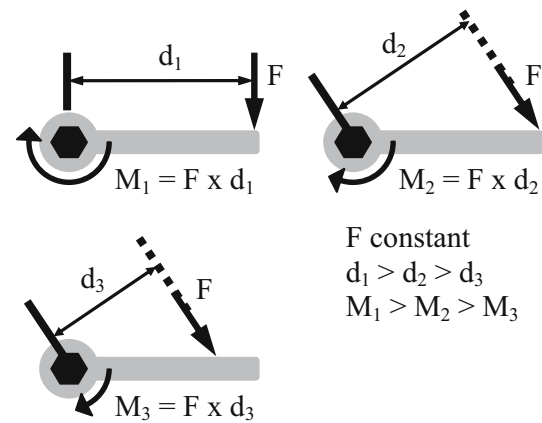


Fig. 2 Moments and lever-arms. The magnitude of a moment is the product of force times the length of the lever-arm. A lever-arm is defined as the perpendicular distance between the force and the center of rotation. A change in either the position or orientation of the applied force will cause a change in the magnitude of the moment. To create the largest moment, the force must be perpendicular to the lever

moment that acts around the center of rotation. Consequently, the units of a moment are Newton meters. In general, the lever acts along the length of the bone, and the joint at the end of that bone serves as the center of rotation or fulcrum. The magnitude and direction of the moment depends upon the point of action of the applied force (Fig. 2). Moments are perhaps understood most easily if one thinks of a seesaw in which individuals with different masses can balance one another by sitting at different distances from the fulcrum (Fig. 3). With human movement, it is the same. External moments produced by the ground reaction force and the inertial forces due to the

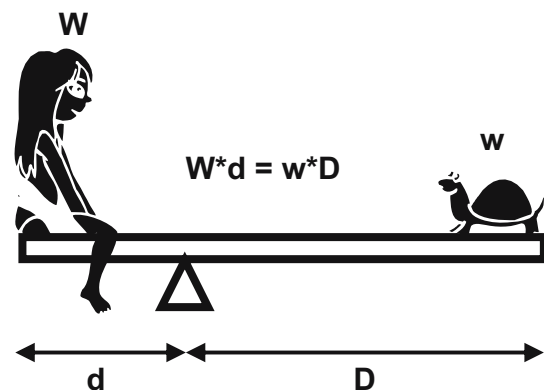


Fig. 3 A simple example to visualize moments on opposite sides of a fulcrum. Two individuals of different weights can balance a teeter-totter, so long as the product of the weight of one individual times his/her distance from the pivot point is equal to the other individual times his/her distance from the pivot. The teeter-totter can be set in motion when one of the individuals pushes against the ground. When this occurs, the effective weight of that individual on the teeter-totter is decreased. Because the moment on the other side is unchanged, static equilibrium is no longer present and so the system becomes dynamic, i.e., the teeter-totter starts to move. Changing the length of the lever, for example, if the turtle walks toward the fulcrum will also set the teeter-totter in motion

weights of the individual body segments are resisted by internal moments produced by the muscles, tendons, and/or ligaments (Fig. 4).

Lever-arm dysfunction is a term that we originally coined to describe the particular orthopedic deformities that arise in an ambulatory child with cerebral palsy [3]. However, the condition is common to any traumatic or neuromuscular problem that produces alteration of the bony skeleton. Lever-arm dysfunction, then, describes a general class of bone modeling, remodeling, and/or traumatic deformities, which include hip subluxation, torsional deformities of long bones, and/or foot deformities. These deformities are common in individuals with cerebral palsy. Because the muscles and/or ground reaction forces must act on skeletal levers to produce locomotion, abnormalities of these lever-arm systems greatly interfere with the child's ability to walk [3, 4].

Because of poor selective motor control, muscle contractures, and/or abnormalities of the bony lever-arms in a condition such as cerebral palsy, the muscle and/or ground reaction forces are neither appropriate nor adequate. With respect to the lever-arms themselves, five distinct types of lever-arm deformities exist: (1) short lever-arm, (2) flexible lever-arm, (3) malrotated lever-arm, (4) an abnormal pivot or action point, and/or (5) positional lever-arm dysfunction (Table 1). A comprehensive discussion of lever-arm dysfunction is beyond the scope of this article, but a common example of lever-arm dysfunction, which is seen in spastic diplegia, will serve to illustrate the problem.

In normal gait during the second half of stance phase, stability of the knee is primarily maintained by the ankle plantar flexors through a mechanism termed the “plantar flexion/knee extension couple”. That is, the action of the soleus at the ankle restrains forward motion of the tibia over the plantigrade foot and in so doing controls the position of

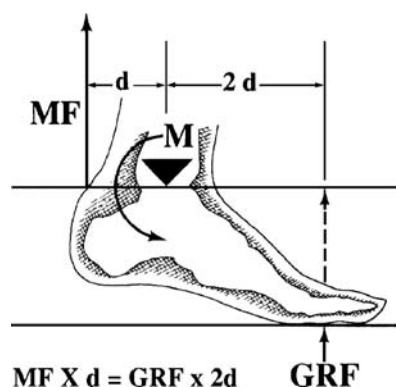


Fig. 4 Moments about the ankle. The external moment produced by the ground reaction force (measured by the force plate) multiplied by its lever-arm ($2d$) must be balanced across the fulcrum of the ankle joint by the internal moment produced by the ankle plantar flexors (MF , muscle force) multiplied by its shorter lever-arm (d). In a gait analysis laboratory, three of the variables can be measured (GRF , $2d$, and d). The fourth (MF) can then be calculated

Table 1 Examples of lever-arm dysfunction

Type	Deformity
Short lever-arm	Coxa valga
Flexible lever-arm	Pes valgus
Malrotated lever-arm	External tibial torsion
Abnormal pivot or action point	Hip subluxation/dislocation
Positional lever-arm dysfunction	Erect vs crouch gait

The types of lever-arm abnormalities that can produce gait problems are listed on the *left*. An example of each type is listed on the *right*. The first four have the effect of reducing the magnitude and/or efficiency of the moment-arm in its normal plane of action. Positional lever-arm dysfunction refers to the fact that the relative magnitude of the hamstring lever-arms at the hip and knee varies according to the position of the lower extremities. As a result of the positional changes in lever-arm length, in erect posture, the hamstrings are better hip extensors than knee flexors. However, in crouch gait, the opposite is true, i.e., the hamstrings are better knee flexors than hip extensors.

the knee joint relative to the ground reaction force. The result is that the ground reaction force produces an extension moment at the knee maintaining the joint in extension without the aid of the quadriceps (Fig. 5).

On the other hand, a child with spastic diplegia frequently has femoral anteversion in conjunction with

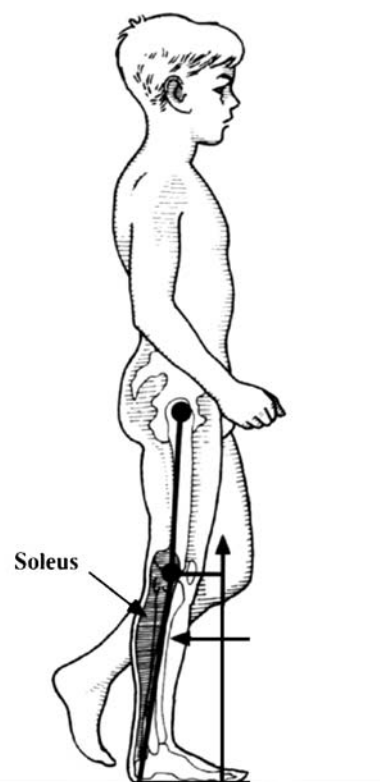


Fig. 5 Midstance. By using the soleus to slow the forward momentum of the shank (tibial segment), the ground reaction force is maintained in front of the knee. The GRF acting on the lever-arm of the foot thereby generates an extension moment on the knee that provides the needed stability without requiring quadriceps activation. This extension moment is referred to as the “plantar flexion/knee extension couple”

equinovalgus foot deformity and/or external tibial torsion. As such, the orientation of the foot can be substantially external to the axis of the knee joint. This can be compounded by the midfoot instability associated with the equinovalgus deformity. Because of the midfoot instability, the foot is too supple to function as an effective lever. As a result, even if the magnitude of the ground reaction force were normal, the magnitude of the extension moment would be greatly reduced (Fig. 6). Fortunately, lever-arm dysfunction is usually correctable with appropriate orthopedic surgery and/or bracing.

Decision-making in ambulatory cerebral palsy

In summary then, if we are going to treat cerebral palsy well, we must understand the pathological mechanisms that cause

gait abnormalities. As discussed earlier, the primary problems of deficient selective motor control, abnormalities of balance, and abnormal muscle tone drive the secondary abnormalities of inadequate muscle growth and bony deformity. The secondary abnormalities are amenable to treatment whereas with the exception of spasticity, the primary abnormalities of cerebral palsy are difficult to alter. Consequently, we must learn to analyze the pathology and determine which portions of it can be corrected and which cannot.

Inadequate muscle growth can be treated by a variety of means including any or all of the following: (1) passive stretch, (2) night splinting, (3) physical therapy, (4) botulinum toxin, (5) phenol or alcohol injections, (6) orthopedic lengthening, and/or (7) neurosurgical spasticity reduction. Bone deformity (lever-arm dysfunction) is best corrected by orthopedic surgery, but modest foot deformities may be amenable to appropriate bracing.

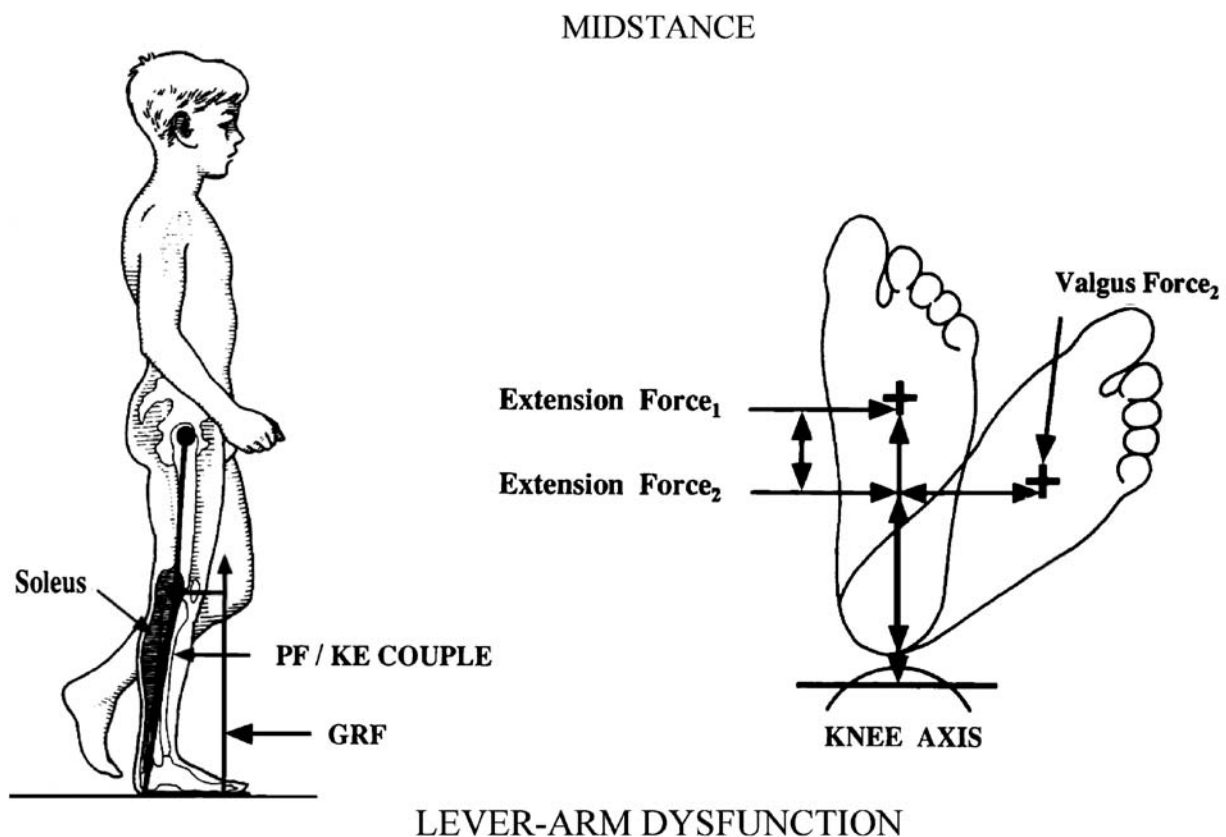


Fig. 6 Malrotated lever-arm dysfunction. With normal anatomy and alignment, note that the lever-arm for the ankle plantar flexors is in alignment relative to the knee and therefore the ankle plantar flexors are able to more effectively generate an extension moment at the knee via the plantar flexion/knee extension couple. An external tibial torsion or equinovalgus foot deformity (or both in combination) causes the center of pressure of application of the GRF to move posterior and lateral relative to its normal position. This has the effect of shortening the

extensor moment lever-arm, which means that the knee extension moment is reduced. In addition, valgus and external rotation moments are introduced. In a growing child, these abnormal forces have the potential of producing more foot deformity, worsening tibial torsion or knee deformities. In addition, the task of sustaining lower extremity extension falls to the hip and knee extensors. With increasing body mass and age, there is not enough power in these two muscle groups to assume this burden and crouch gait invariably occurs [5]

Abnormal muscle tone is a primary problem and can be challenging to evaluate and quantify. Minor degrees of tone abnormality can be managed nonoperatively. Children with multilevel, pure spasticity (Ashworth ≥ 3) at our institution are treated with selective dorsal rhizotomy provided that the other criteria for the procedure (absence of other types of abnormal muscle tone, good selective motor control, adequate underlying muscle strength, age 4–7 years, and a diagnosis of diplegic cerebral palsy due to prematurity) are met. These children typically enjoy a higher level of mobility (gross motor function classification system [GMFCS] level I/II). Children with significant hypertonia who do not meet the selection criteria for selective dorsal rhizotomy are currently being treated at our center with the intrathecal baclofen pump. Typically, these children have more severe ambulatory dysfunction (GMFCS III/IV). In general, abnormal tone emulating from basal ganglia injury is not amenable to treatment although there are a few oral pharmacological agents that can be tried.

Deficient selective motor control and abnormal balance mechanisms are permanent disabilities. Treatment with physical therapy is appropriate and useful as maturation occurs. Physical therapy should be used to maximize function in this setting, but currently there is no way to eliminate these problems. As such, poor motor control and abnormal balancing mechanisms can be the limiting factors for the overall treatment program of a particular child. Therefore, they must be recognized and discussed with the family so that appropriate goal setting can occur.

Knowledge of the pathologic gait of cerebral palsy as outlined above must be applied to treatment. To do so, the following list of management principles of ambulatory disability in cerebral palsy can be useful in determining a specific treatment program.

1. Spasticity reduction.
2. Correction of contractures.
3. Simplification of the control system.
4. Preservation of power generators.
5. Correction of lever-arm dysfunction.

The first two principles broadly guide treatment decision-making at all ages (although one would use different treatment methods to accomplish these two goals at different ages). In addition to the correction of contractures, the latter three are applied when designing the orthopedic surgical intervention.

Spasticity management and correction of contractures can be addressed in many different ways (physical therapy, orthotics, botulinum toxin injections, oral spasmolytics, selective dorsal rhizotomy, intrathecal baclofen, and/or tendon lengthening). In practice, choosing among these options depends largely on the child's age. Global spasticity management is frequently the method of choice for children

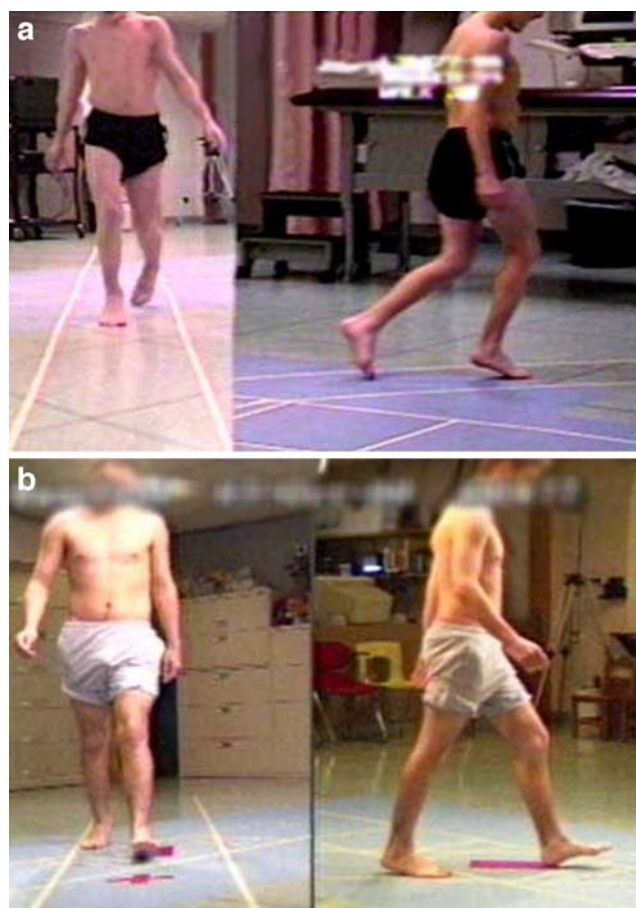
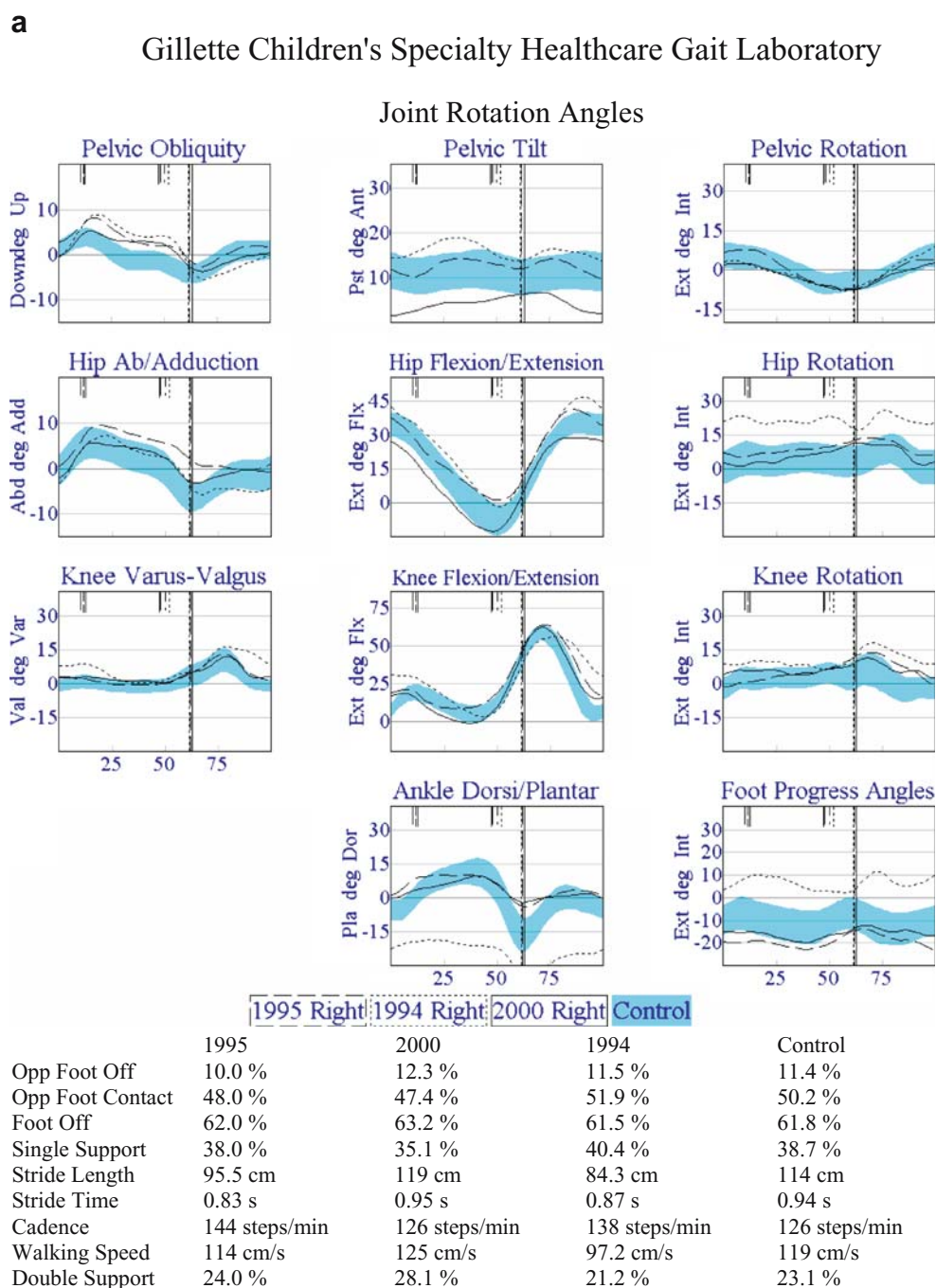


Fig. 7 Case study 1. Subject walking in gait laboratory before surgery (a) and after treatment, 6 years later (b)

between ages 4 and 7 years, whereas botulinum toxin injection and/or stretching casts are better choices for younger children and/or adolescents in the midst of their growth spurt. The reader should be aware that there is very little definitive scientific evidence comparing the efficacy of each of these methods at different ages. The overall management scheme is based largely on clinical experience and knowledge of the pathomechanics of cerebral palsy. If careful quantitative assessment and evaluation of outcomes are used as a routine part of the treatment program, optimal methods of achieving specific treatment objectives should become apparent and long-term ambulatory and joint function as an adult should be improved [9].

On the other hand, lever-arm dysfunction that interferes with function should be managed regardless of age. Consider, for example, the unstable talipes equinovagis foot that is so common in children with spastic diplegia. If we remember that moment generating capability is dependent on both the ability to generate muscle force and the presence of an intact and properly aligned lever-arm, it will

Fig. 8 Case study 1. Kinematics of the left side (a) and right side (b) on the three separate occasions: initial, 1994 (*dotted line*); 12 months after multiple lower extremity reconstruction, 1995 (*dashed line*); and 6 years post-operatively at the end of the adolescent growth spurt, 2000 (*solid line*)



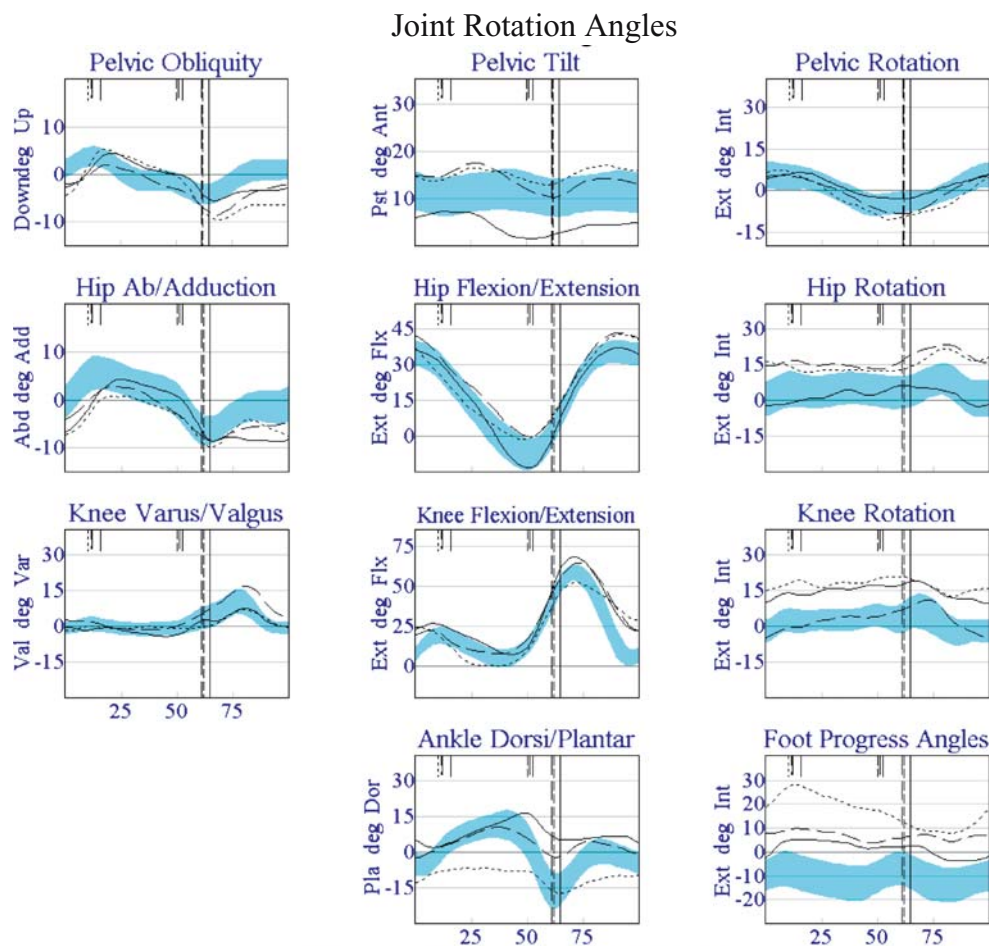
help us focus on the importance of correcting lever-arm dysfunctions such as pes valgus. For the very young child with milder deformity, physical therapy and orthoses are most appropriate. For older children, these are inadequate and surgical intervention is necessary. Choosing the correct surgical procedure(s) represents still another level of complex decision-making. For example with respect to midfoot instability, options include os calcis lengthening, os calcis lengthening plus tightening of the medial/plantar

talonavicular joint capsule with or without posterior tibial tendon advancement, and os calcis lengthening plus talonavicular arthrodesis. In addition to midfoot instability, this type of foot deformity is typically associated with hindfoot and forefoot problems. Gastrocnemius contracture is usually part of the pathology that leads to this foot deformity, so gastrocnemius recession is often required as part of the surgical treatment. Forefoot deformities are not uncommon and must be recognized and treated as well.

Fig. 8 (continued)

b

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	1995 Left	1994 Left	2000 Left	Control
Opp Foot Off	12.2 %	15.8 %	10.0 %	11.4 %
Opp Foot Contact	51.0 %	52.6 %	50.0 %	50.2 %
Foot Off	61.2 %	64.9 %	62.0 %	61.8 %
Single Support	38.8 %	36.8 %	40.0 %	38.7 %
Stride Length	96.1 cm	121 cm	77.9 cm	114 cm
Stride Time	0.82 s	0.95 s	0.83 s	0.94 s
Cadence	73.2 steps/s	63 steps/s	72 steps/s	126 steps/min
Walking Speed	117 cm/s	127 cm/s	93.5 cm/s	119 cm/s
Double Support	22.4 %	28.1 %	22.0 %	23.1 %

Plantar flexor moment insufficiency can be a debilitating cause of crouch gait at any age. For young children, botulinum toxin injection to the gastrocnemius in conjunction with a well-fitting, solid ankle-foot orthosis (AFO) to maintain foot alignment and stability may be appropriate. One must carefully assess older children with crouch gait for bony deformities such as external tibial torsion, distal tibial valgus, pes valgus, femoral anteversion, knee flexion contracture, and/or patella alta (usually associated with knee extensor lag).

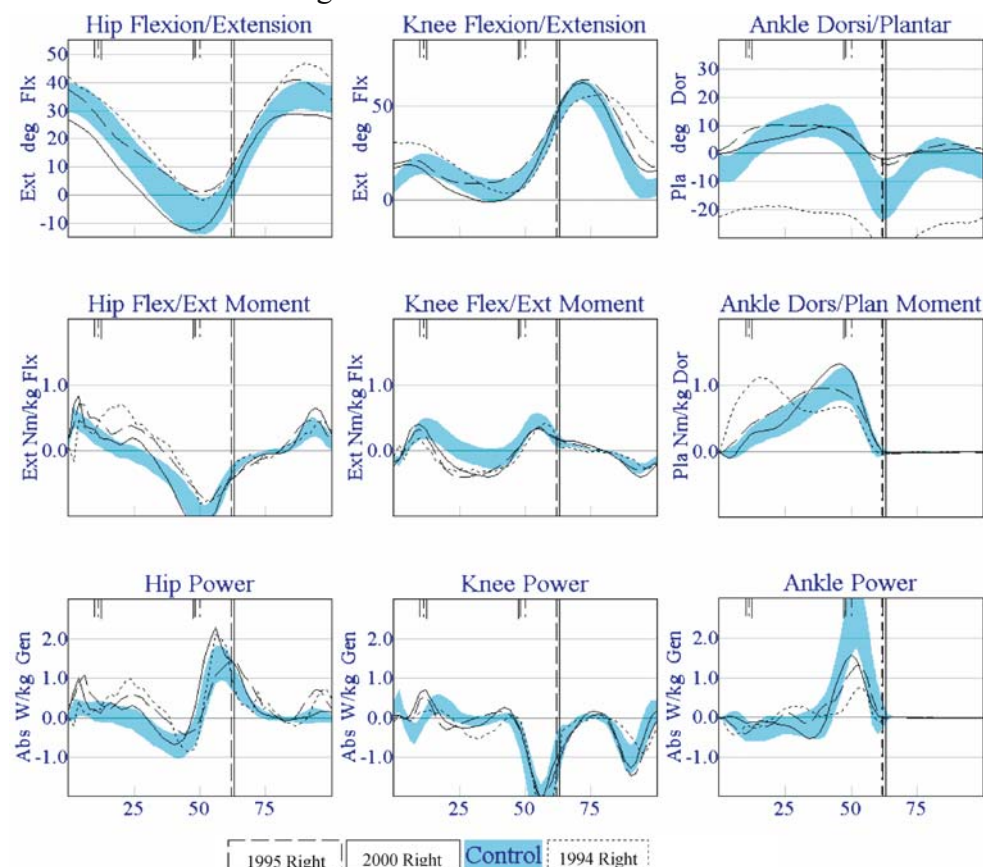
In addition, crouch gait can be due to soleus weakness. Consequently, physical therapy to strengthen the soleus should be ongoing. If this proves to be insufficient to avoid crouch, an appropriate AFO (stiff posterior leaf spring (PLS), solid or rear entry floor reaction) should be recommended to compensate for this deficiency. A fitness exercise program such as swimming or bicycling should be incorporated if possible. The benefits of improved weight management, cardiovascular health, and overall muscle strength are essential.

Fig. 9 Case study 1. Corresponding kinetic data for the right and left sides (a, b)

a

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Sagittal Plane Moments and Powers



	1995	2000	1994	Control
Opp Foot Off	10.0 %	12.3 %	11.5 %	11.4 %
Opp Foot Contact	48.0 %	47.4 %	51.9 %	50.2 %
Foot Off	62.0 %	63.2 %	61.5 %	61.8 %
Single Support	38.0 %	35.1 %	40.4 %	38.7 %
Stride Length	95.5 cm	119 cm	84.3 cm	114 cm
Stride Time	0.83 s	0.95 s	0.87 s	0.94 s
Cadence	144 steps/s	126 steps/s	138 steps/s	126 steps/min
Walking Speed	114 cm/s	125 cm/s	97.2 cm/s	119 cm/s
Double Support	24.0 %	28.1 %	21.2 %	23.1 %

Methods of treatment in the age of spasticity management are different than the prior era when tendon lengthenings alone were done. Spasticity reduction allows the child to have greater range-of-motion, less spastic response to stretch, and better potential for the development and use of voluntary muscle activity during gait. Consequently, orthotic needs are different. Frequently less rigid, posterior leaf spring AFOs can now be used. Spasticity hinders strengthening programs for children with cerebral palsy, and insufficient muscle strength can be a major cause of ongoing disability. As a result of spasticity reduction, physical therapy for strengthening may now be more beneficial than in the past. If the treating physician remembers that moment generating capability is the

key to good musculoskeletal function, it will allow him/her to identify not only muscle strength deficiencies, but also lever-arm dysfunction. For example, knee extensor lag and plantar flexor moment insufficiency can both be debilitating causes of crouch in the adolescent.

Case studies

Patient 1

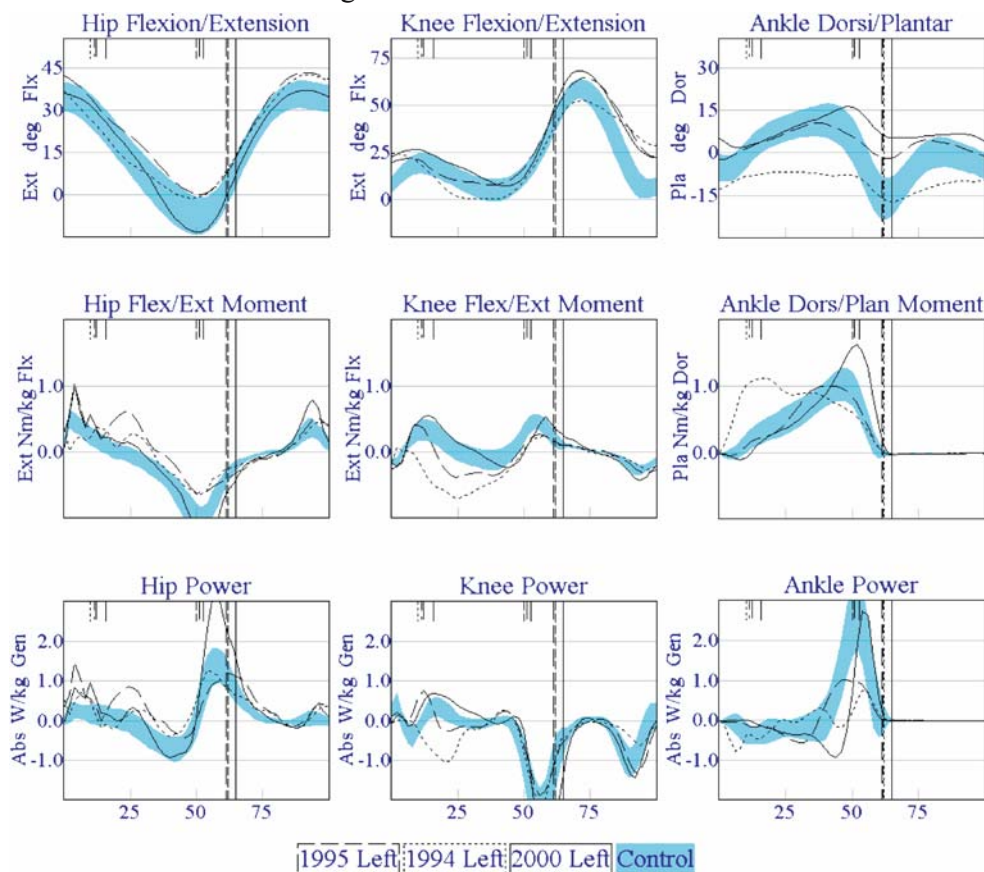
This 10-year-old with spastic diplegic cerebral palsy is an independent community ambulator without the use of

Fig. 9 (continued)

b

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Sagittal Plane Moments and Powers



	1995	2000	1994	Control
Opp Foot Off	12.2 %	15.8 %	10.0 %	11.4 %
Opp Foot Contact	51.0 %	52.6 %	50.0 %	50.2 %
Foot Off	61.2 %	64.9 %	62.0 %	61.8 %
Single Support	38.8 %	36.8 %	40.0 %	38.7 %
Stride Length	96.1 cm	121 cm	77.9 cm	114 cm
Stride Time	0.82 s	0.95 s	0.83 s	0.94 s
Cadence	146 steps/min	126 steps/min	144 steps/min	126 steps/min
Walking Speed	117 cm/s	127 cm/s	93.5 cm/s	119 cm/s
Double Support	22.4 %	28.1 %	22.0 %	23.1 %

assistive devices or orthoses. At 1 year of age, he had undergone bilateral tendo-Achilles lengthening and plantar fascia release.

Gait by observation shows toe-toe initial contact bilaterally, worse on the right. There is bilateral knee hyperextension in midstance and bilateral internal foot progression angles (Fig. 7a).

Physical examination shows mild hip flexion contractures (5°), bilateral hamstring and rectus femoris contractures, bilateral femoral anteversion (40°, right and 50°, left), a mild left internal tibial torsion, and bilateral equinus contractures (right greater than left). Motor control is good

and strength is rated 3–4/5 in general. Spasticity is felt to be mild at the hips and knees, and moderate at the ankle plantar flexors.

Kinematics of the transverse plane demonstrate excessive bilateral internal hip rotation, worse on the right, but with a more severe internal foot progression angle on the left (Fig. 8a and b). These findings suggest contributions from excessive femoral anteversion bilaterally and internal tibial torsion on the left.

Sagittal plane kinematics are consistent with multilevel spasticity/contracture. He has a double-bump pelvic pattern, restriction of hip extension in terminal stance, excessive

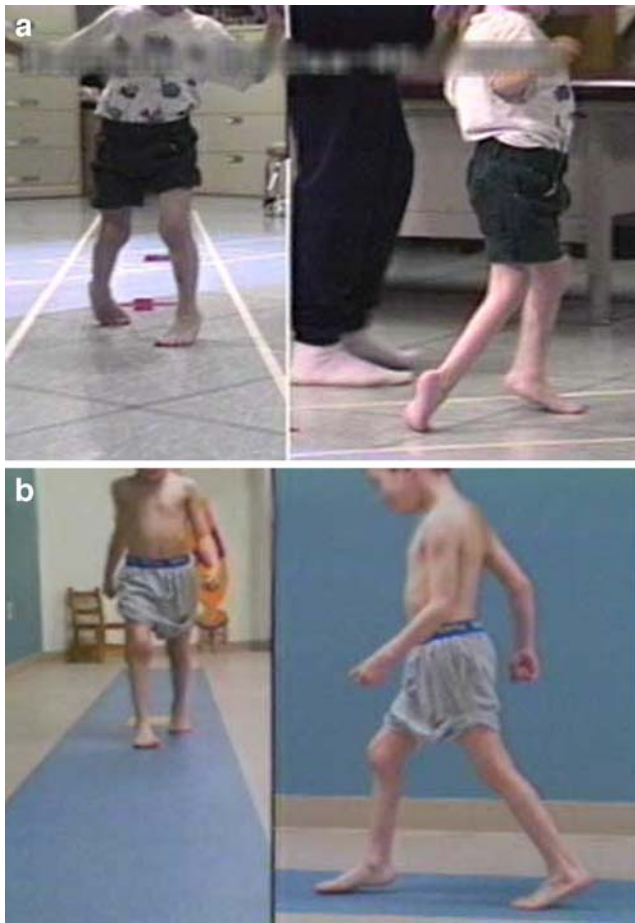


Fig. 10 Case study 2. Subject walking in gait laboratory before surgery (a) and after treatment, 2 years later (b)

knee flexion at initial contact, diminished peak swing phase knee flexion, mild genu recurvatum in midstance and equinus bilaterally (worse on the right than the left).

Coronal plane findings are normal with the exception of pelvic obliquity (right side elevated) and excessive adduction of the right hip. This is felt to be a compensation for the functional leg length discrepancy caused by the asymmetric equinus deformity (again, worse on the right).

Sagittal plane kinetics (Fig. 9a and b) evaluation shows a lack of first rocker, a very abnormal and early ankle moment, lack of power generation for push-off at the ankle, excessive knee flexion moments bilaterally (left worse than the right), which is secondary to excessive plantar flexion knee extension couples, and prolonged hip extension moments in stance (hip extensor dominance pattern). Walking speed is approximately 0.75 times normal. Oxygen consumption is just outside the normal range (1.51 times normal).

This information suggested that spasticity was not severe enough to consider selective dorsal rhizotomy. Motor control and strength were quite good, making this patient a good candidate for rehabilitation after multiple lower extremity

surgery. There were multiple contractures and bone deformities. However, it was felt that the internal tibial torsion was not severe enough to warrant surgical correction. Based on this information, the surgeries included:

1. bilateral intertrochanteric femoral derotational osteotomies
2. bilateral psoas lengthenings at the pelvic brim
3. bilateral medial hamstring lengthenings
4. bilateral rectus femoris transfers to the gracilis
5. bilateral Strayer gastrocnemius recessions
6. right soleus fascial stripping

Postoperative analysis was performed 10 months later. Preoperative to postoperative analysis showed correction of internal foot progression angle with normalization of hip rotation (Fig. 7a and b). Anterior pelvic tilt and double-bump pattern were improved. Knee extension in preparation for initial contact and at initial contact is improved (Fig. 8a and b). Knee hyperextension is resolved with normal knee extension at midstance with no evidence of crouch. Timing of peak swing phase knee flexion and magnitude are both improved due to rectus femoris transfer. The equinus contracture is resolved and pelvic obliquity is improved. Excessive hip adduction in stance remains secondary to persistent abductor weakness. On the left, there is a persistent internal foot progression angle secondary to unresolved internal tibial torsion. Transverse plane hip rotation does not appear to be improved, but in retrospect, the knee alignment device may have been placed too far internal as there is an abnormal knee varus wave in swing phase. Knee motion and equinus are similarly improved on the left side.

Ankle moments are improved bilaterally (Fig. 9a and b). The knee flexion moment in stance on the left is normalized. Power generation at the ankle is maintained/improved. There is a persistence of hip extensor dominance pattern. Oxygen consumption is now in the normal range (1.22 times normal) although self-selected walking speed for that test is slower than it was preoperatively. Femoral implants were removed as the femoral osteotomies were well healed.

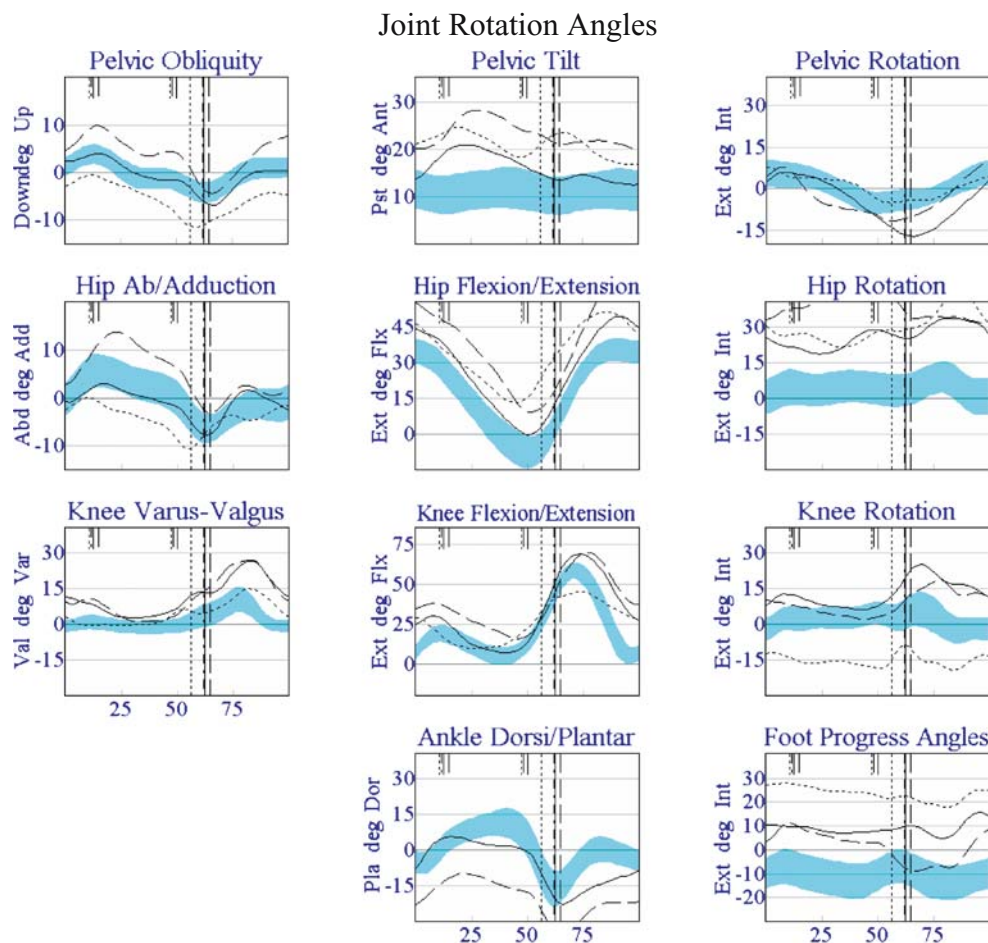
Six years after surgery, at age 16, the patient was reexamined in the motion analysis laboratory to evaluate the effects of time, growth, and therapy. Physical examination shows some hamstring contracture. The internal tibial torsion persists. There is mild gastrocnemius tightness.

Kinematics are in the normal range in the sagittal plane (Fig. 8a and b). In particular, hip extension is normal in terminal stance, knee modulation is normal, there is no equinus, and hindfoot initial contact is normal. The internal foot progression angle is persistent on the left as the internal tibial torsion has not resolved. Hip rotations are normal. Hip abductor weakness is no longer evident either by kinematics or kinetics (typically these findings do improve after plate

Fig. 11 Case study 2. Kinematics of the right side (a) and left side (b) on three separate occasions: initial, 2000 (dotted line); 12 months after SDR 2001 (dashed line); and 12 months after lower extremity orthopedic reconstruction, 2001 (solid line)

a

Gillette Children's Specialty Healthcare Gait Laboratory



	2001 Right	2000 Right	2002 Right	Control
Opp Foot Off	14.8 %	12.5 %	10.5 %	11.4 %
Opp Foot Contact	48.1 %	50.0 %	47.4 %	50.2 %
Foot Off	64.8 %	62.5 %	56.1 %	61.8 %
Single Support	33.3 %	37.5 %	36.8 %	38.7 %
Stride Length	84.9 cm	90.4 cm	51.4 cm	114 cm
Stride Time	0.90 s	0.80 s	0.95 s	0.94 s
Cadence	134 steps/min	150 steps/min	126 steps/min	126 steps/min
Walking Speed	94.3 cm/s	113 cm/s	54.1 cm/s	119 cm/s
Double Support	31.5 %	25.0 %	19.3 %	23.1 %

removal). The hip, knee, and ankle moments are in the normal range (Fig. 9a and b). Hip flexor power production at toe-off is increased, and ankle plantar flexor power production is normal. This assessment shows no losses with growth despite having gone through the adolescent growth spurt (significant because the natural history of untreated cerebral palsy is typically characterized by worsening). Oxygen consumption remains in the normal range at normal walking speed. Functionally, he is doing well.

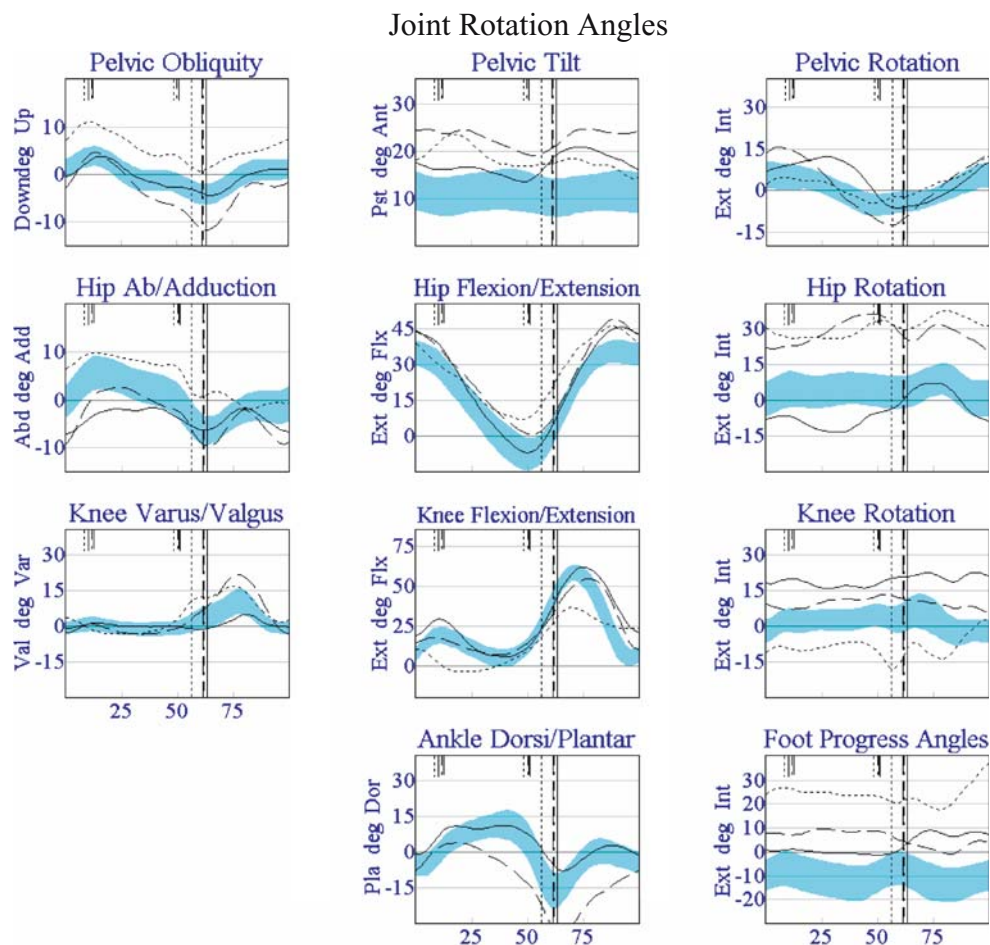
Patient 2

This 4+2-year-old boy with diplegic cerebral palsy is a limited community ambulator with bilateral hinged AFOs (Fig. 10a). He was born 6 weeks premature. Postnatal course included a 3-week stay in the neonatal intensive care unit, 1 day of ventilator assistance, apnea, and jaundice. His birth weight was 4 lbs and 10 oz. He started walking on his toes at 15 months of age. He uses a wheelchair for

Fig. 11 (continued)

b

Gillette Children's Specialty Healthcare Gait Laboratory



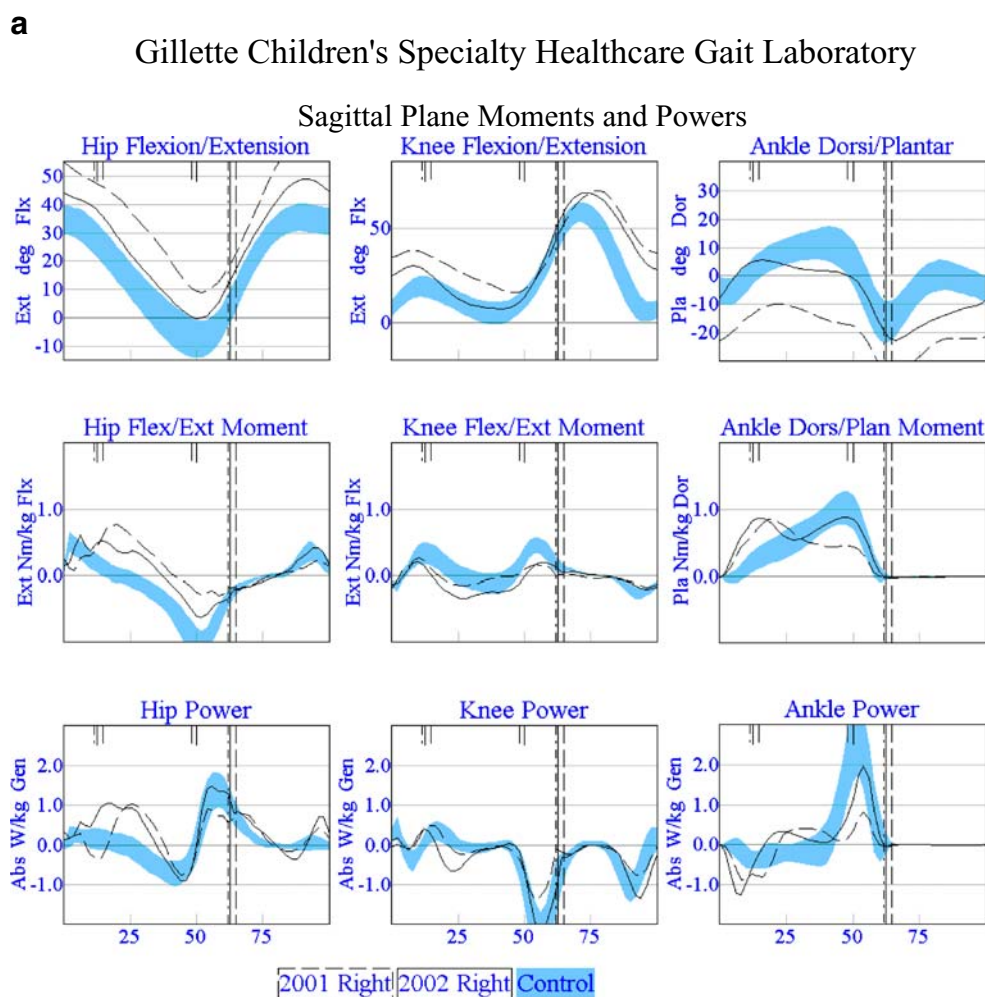
	2001 Left	2000 Left	2002 Left	Control
	2001	2002	2000	Control
Opp Foot Off	12.3 %	10.2 %	8.06 %	11.4 %
Opp Foot Contact	50.9 %	51.0 %	48.4 %	50.2 %
Foot Off	61.4 %	63.3 %	56.5 %	61.8 %
Single Support	38.6 %	40.8 %	40.3 %	38.7 %
Stride Length	87.4 cm	91.6 cm	53.7 cm	114 cm
Stride Time	0.95 s	0.82 s	1.03 s	0.94 s
Cadence	126 steps/min	146 steps/min	116 steps/min	126 steps/min
Walking Speed	92 cm/s	112 cm/s	51.9 cm/s	119 cm/s
Double Support	22.8 %	22.4 %	16.1 %	23.1 %

community distances and has ongoing physical therapy. He has had previous Botox injections to the hamstrings and gastrocnemius muscles.

Physical examination revealed bilateral excessive femoral anteversion and left pes valgus. There was multilevel spasticity including the hip flexors and adductors, hamstrings, rectus femoris, and gastrocnemius bilaterally. Selective motor control and strength were felt to be acceptable to consider rhizotomy. Spasticity is rated moderate.

Kinematics (Fig. 11a and b) showed bilateral severe internal foot progression angles secondary to femoral anteversion (hip rotation severely internal). Sagittal plane findings were consistent with multilevel spasticity with severe equinus, stiff knees, excessive hip flexion, and anterior pelvic tilt. Coronal plane findings were explainable (as in the previous case) on the basis of functional leg length discrepancy with a stiff hyperextended left knee and a flexed right knee.

Fig. 12 Case study 2. Corresponding kinetic data for the right and left sides for 2001 and 2002 (a,b)



	2001	2002	2000	Control
Opp Foot Off	14.8 %	12.5 %	10.5 %	11.4 %
Opp Foot Contact	48.1 %	50.0 %	47.4 %	50.2 %
Foot Off	64.8 %	62.5 %	56.1 %	61.8
Single Support	33.3 %	37.5 %	36.8 %	38.7 %
Stride Length	84.9 cm	90.4 cm	51.4 cm	114 cm
Stride Time	0.90 s	0.80 s	0.95 s	0.94 s
Cadence	134 steps/min	150 steps/min	126 steps/min	126 steps/min
Walking Speed	94.3 cm/s	113 cm/s	54.1 cm/s	119 cm/s
Double Support	31.5 %	25.0 %	19.3 %	23.1 %

It was not possible to collect force plate data due to short step lengths and clearance problems. Electromyography (EMG) findings showed multilevel spasticity with co-contraction. However, even though the EMG signals were prolonged, there were some definite “off” times. Oxygen consumption was 1.5 times normal at a slower than normal walking speed.

As a result of this assessment, it was felt that the patient was a good candidate for a selective dorsal rhizotomy. A 42% right and 38% left rhizotomy was done. Evaluation at 11 months postsurgery shows decreased spasticity, but

persistent equinus plus bilateral severe femoral anteversion and pes valgus foot deformity.

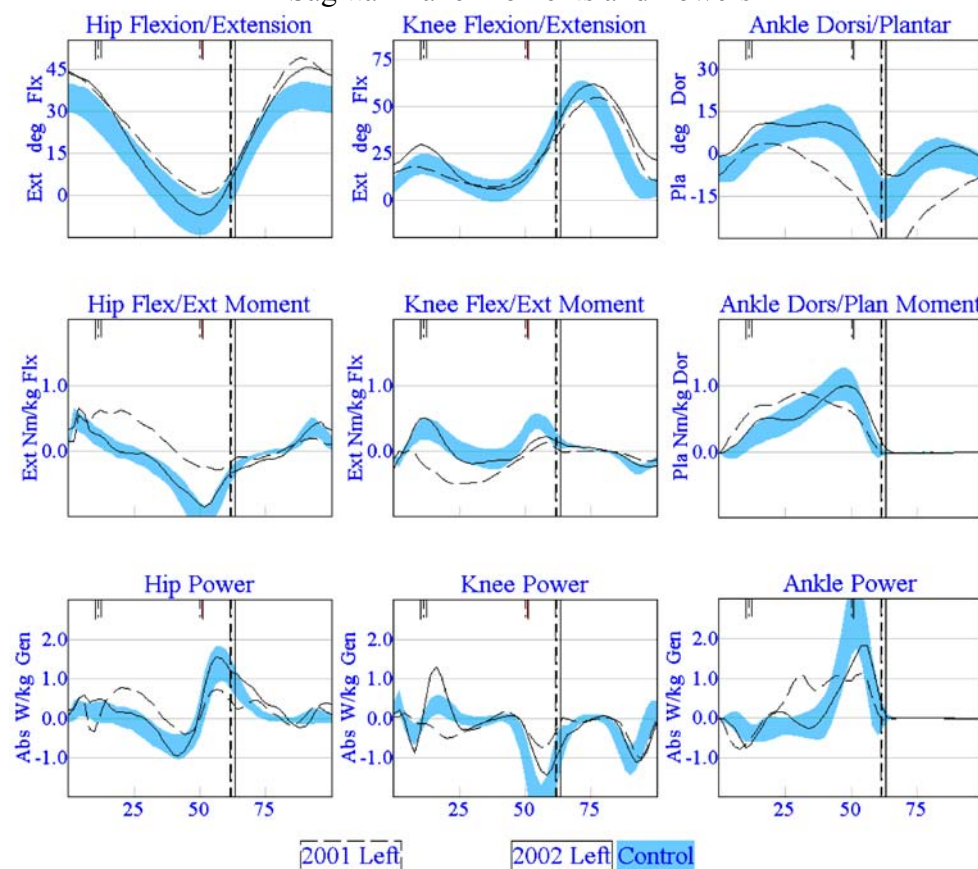
Kinematics show persistent internal foot progression angles with severe excessive internal hip rotation (Fig. 11a and b), Fig. 12. Sagittal plane findings show improved hip extension, improved swing phase knee flexion, and improved equinus as a result of spasticity reduction. However, there is persistent equinus with a drop foot in swing phase. There is persistent crouch on the right with excessive knee flexion in stance, and there is significant anterior pelvic tilt. Walking speed is significantly improved from less than 0.5

Fig. 12 (continued)

b

Gillette Children's Specialty Healthcare Gait Laboratory

Sagittal Plane Moments and Powers



	2001	2002	2000	Control
Opp Foot Off	12.3 %	10.2 %	8.06 %	11.4 %
Opp Foot Contact	50.9 %	51.0 %	48.4 %	50.2 %
Foot Off	61.4 %	63.3 %	56.5 %	61.8 %
Single Support	38.6 %	40.8 %	40.3 %	38.7 %
Stride Length	87.4 cm	91.6 cm	53.7 cm	114 cm
Stride Time	0.95 s	0.82 s	1.03 s	0.94 s
Cadence	126 steps/min	146 steps/min	116 steps/min	126 steps/min
Walking Speed	92 cm/s	112 cm/s	51.9 cm/s	119 cm/s
Double Support	29.6 %	22.4 %	16.1 %	23.1 %

times normal before treatment to approximately 0.75 times normal post rhizotomy. Energy assessment shows improved walking speed with no change in oxygen consumption.

To correct lever-arm dysfunction and residual contractures, the patient underwent:

1. bilateral intertrochanteric femoral derotational osteotomies
2. bilateral os calcis lengthenings
3. bilateral gastrocnoleus recessions
4. bilateral medial hamstring Botox injections
5. right gastrocnoleus Botox injection

Ten months later, assessment shows some persistent internal hip rotation, although this may be due to abnormal marker placement (Fig. 11a and b). The anterior pelvic tilt, incomplete hip extension, and in-toeing are all improved bilaterally. We attributed this to a correction of the lever-arm deformity that resulted from the derotational femoral osteotomies. The knees are now nearly symmetric with resolution of crouch on the right and mild hyperextension on the left. Equinus is significantly improved. Self-selected walking speed is now normal. Oxygen consumption is measured as increased although walking speed is also faster.

SDR outcomes analysis

A recent retrospective analysis of 136 children at our institution who underwent selective dorsal rhizotomy at mean age of 7.2 years showed improvement in excessive pelvic motion (“double-bump” pattern), improvement in hip extension and hip range-of-motion, improved knee extension in stance and knee range-of-motion, and correction of equinus foot deformity. Oxygen cost decreased by 34%. Spasticity improved as measured by the Ashworth score:

	Preoperative	Postoperative
Hip flexors	1.5	1.1
Hip adductors	2.7	1.3
Hamstrings	2.1	1.2
Plantar flexors	3.1	1.7

Overall function improved an average of one level on the Gillette Functional Assessment Questionnaire [13].

Summary

This article was written with the goal of illustrating the current methodology for treating children with ambulatory problems due to cerebral palsy at our hospital and clinics. The program is broadly based on two general concepts: (1) the management of spasticity and (2) management of growth deformities (muscle contractures and lever-arm dysfunction.). Identification and management of abnormalities in these two areas are central to this methodology.

Optimization of gait potential requires that both spasticity be reduced and growth deformities addressed. The neurosurgeon can offer remediation of spasticity by means of selective dorsal rhizotomy or the intrathecal baclofen pump. However, the neurosurgeon cannot address the growth deformities (bone deformities and/or residual muscle contractures). The orthopedist, on the other hand, can address the latter deformities, but cannot alter spasticity. Therefore, both disciplines must be involved and communicating with one another if optimal treatment is to be achieved. Even if addressing both the neurosurgical and orthopedic problems optimally treats the child, the result will not be good without adequate and well-directed therapy and orthotic prescription.

Gait analysis has and will continue to play a crucial role in many of aspects of this management scheme including dynamic evaluation of the unique problems of each patient, which enables improved decision-making; education to better understand normal and pathological gait; and assessment of outcome after major interventions. Before computerized gait analysis, there was no objective mea-

surement tool by which the complex dynamics of cerebral palsy gait and movement disorders could be assessed.

It should be apparent that a team of specialists, which includes physical therapists, orthotists, neurosurgeons, physiatrists, and orthopedic surgeons, are necessary to adequately evaluate and treat the multiple problems in this complex condition. Each of these specialists offers a unique perspective, which enhances the development of a comprehensive evaluation and treatment program for each individual patient.

It is also true that the not all of specific treatments outlined in this article have not been critically evaluated via double-blinded, randomized, controlled clinical trials. The use of gait analysis along with concurrently gathered functional outcomes data as objective measurement tools, however, will allow the benchmarking of this treatment methodology against others and, in so doing, will provide the basis for future progress in the evaluation and treatment for this interesting and challenging group of patients.

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