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# The impact of derotation orthosis (twister cables) on muscle activity in spastic muscles and their antagonists during gait in a child with cerebral palsy – a single case study.

by

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**SUMMARY**

Orthopedic aids have rarely been subjected to scientific scrutiny and have thus been rejected or accepted based on subjective opinions. The purpose of this study was to evaluate the impact of derotational orthosis on spasticity during walking in a child with cerebral palsy. The research questions addressed whether muscle activity changed during walking with the use of derotational orthosis compared to walking without it, which spastic muscles were affected, and whether and how antagonist muscles to the spastic muscles were affected. An experimental study was conducted on a six-year-old girl with cerebral palsy, diplegia-ataxia. EMG recordings of the gastrocnemius, tibialis anterior, gluteus medius, and adductors were performed during walking for 2x15 meters without derotational orthosis, with it immediately after application, and after 30 minutes of use. Video recordings of walking with or without derotational orthosis were made. The results showed that muscle activity in the spastic gastrocnemius was reduced to normal values with the use of derotational orthosis for 30 minutes. A trend was observed indicating that over time, spasticity decreased and the ability to use hip abductors increased with derotational orthosis. The conclusion drawn was that derotational orthosis was a valuable walking aid for the child measured and that the rotational component's ability to achieve spasticity reduction should be considered in children with cerebral palsy.

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## INTRODUCTION

Families and therapists have attempted in various ways to help children with cerebral palsy (cp) learn to walk. Independent walking can lead to not only greater physical well-being but also to a better ability to navigate their surroundings and explore their environment. Cerebral palsy is the term used to describe a range of conditions involving motor dysfunction due to a non-progressive brain injury early in life (Levitt, 1982). Despite the injury being non-progressive, the clinical picture changes as the nervous system develops and the child grows (McCarthy, 1986). The functional abilities and symptomatology can vary greatly among different children with cerebral palsy. Even if children are at the same level of motor development for their age, the treatment to improve their functional capacity may vary as their impairments differ. For example, enhancing the walking ability of a child with CP requires different solutions for different children.

The term "muscle tone" has an uncertain meaning but is used here to refer to muscle activity. Disorders of muscle tone are common in children with CP, often involving a combination of hypo- and hypertonia. This can be partly explained by the distinction between two types of muscle tone: intrinsic tone and postural tone (Shumway, 1986). Intrinsic tone refers to the reactivity of the stretch reflex and can only be triggered in healthy individuals by a tap to the tendon. Postural tone is "a state of continuous and untiring contraction of postural muscles necessary to overcome gravity and maintain posture" (Foley, 1977).

Consequently, there may be a simultaneous decrease in postural tonus if postural mechanisms are impaired and an increase in intrinsic tonus in the absence of inhibitory control over the stretch reflex arc.

Hypertonia is a common feature in the symptomatology of a child with CP. To describe the quality of different types of hypertonia, terms such as spasticity, spasm, rigidity, and dystonia are used (Carr & Shepard, 1980). The physiological differences between the various clinical manifestations of hypertonia are not fully elucidated, but it can be assumed that they are all likely caused by different mechanisms through which the brainstem is released from control by higher centers (Carr & Shepard, 1980).

### Spasticity

Spasticity of central origin is the form of hypertonus caused by damage to the motor cortex or its extrapyramidal outflow, which normally exerts an overarching, controlling influence on brainstem activity. Spasticity is thus a clinical manifestation of abnormal tone caused by disrupted supraspinal control over the stretch reflex

arc (Carr & Shepard, 1980). Central spasticity is distinguished from spinal spasticity, which can occur with a complete spinal cord injury. Spinal spasticity is characterized by a greater release of flexor reflexes due to the cessation of inhibitory control by the dorsal reticulospinal pathways. In an injury above the brainstem, the reticular system retains its inhibitory control over flexor reflex afferents. Central spasticity is commonly found in children with cerebral palsy. Campbell (1984) suggests that spasticity in a growing child may secondarily cause abnormal muscle fiber development because it affects coordination, strength, speed, and duration of movements in a musculoskeletal system undergoing developmental changes. Spasticity can ultimately lead to weakness in the antagonist muscles to the spastic muscles. Poor endurance, compensatory posture, contractures, and skeletal deformities may also occur (Campbell, 1984).

Also, the spastic muscles can be weak when spasticity is eliminated. Some children rely on spasticity to stand and walk, and they would lose these abilities if it were eliminated. However, in other children, spasticity causes reduced function by contributing to misalignments or joint deformities. For example, spasticity causing plantar flexed feet and significant scissoring of the legs can impair walking function in a child with CP. These children are often discouraged from walking to prevent an increase in spasticity (Levitt, 1982).

Abnormal muscle tone is just one of the issues in CP. Absence or lack of postural reactions is another common symptom that doesn't necessarily correlate with the degree of spasticity. Levitt (1982) argues that the development of independent functions depends on the child's level of postural reactions, not solely on how spastic they are. For example, a child with spasticity may develop independent walking while a child with mild spasticity may not be due to the former's better postural reactions. This aligns with Paine (1978), who demonstrated that postural reflexes do not necessarily correlate with an increase or decrease in spasticity. Pederson (1980) suggests that spasticity also doesn't necessarily correlate with voluntary movements. Eliminating spasticity can either strengthen, weaken, or abolish voluntary movements. Nwaobi et al. (1983) & Nwaobi (1987) suggest that the relationship between muscle hyperactivity and voluntary movements is unclear but have shown in their studies that more spastic sitting leads to poorer voluntary fine motor skills. New studies emphasize the position of the center of gravity in space as a crucial factor in the extent of spasticity (Myhr & von Wendt 1990 & 91, Nwaobi et al. 1983 & Nwaobi 1986, Steen et al. 1990), and for the ability to improve postural control (Myhr & von Wendt 1990). Hirschfeldt (1990) has shown that it's the center of gravity of the trunk, or its displacement, that triggers the postural response. Although postural control may be the determining factor in the

development of independent walking, there are cases where spasticity, through abnormal movement patterns, hinders walking.

**Normal and spastic gait**

The so-called infant stepping is a walking pattern that can be elicited in newborn babies and usually disappears by 2 months of age. Infant stepping was described by Forsberg (1985). It is characterized by the leg tending to move as a unit in flexion and extension. The leg remains relatively flexed throughout the walking cycle. Flexors are active during the stance phase. During the swing phase, the leg is lifted by a powerful flexion in all joints as the forefoot is placed on the support surface with a backward movement of the leg. Flexors are co-activated during the swing phase and are thus active for a large part of the walking cycle. During push-off, the horizontal force at the foot is directed medially, causing the contralateral foot to be placed in front of the support leg. Both vertical and forward propulsion are minimal or absent during the toe-off due to the lack of propulsion firing in the calf muscles. The leg is rotated forward with plantar flexion instead of push-off. From 2 months to 7 months of age, a so-called astasia stage occurs when the child does not support on their feet. This is followed by the child supporting on their feet and the development of voluntary standing and walking begins. The maturation of muscle activity patterns during walking occurs during the first 2 to 3 years after the onset of independent walking. By 4 years of age, children exhibit a muscle activity pattern like adults (Tata & Peat, 1987; Leonard et al., 1988). It is established that EMG patterns show significant intersubject variability in normal individuals, but according to Forsberg et al. (1989), mature adult gait has certain characteristic features that distinguish it from the newborn's digitigrade pattern (infant stepping) to a plantigrade gait. What characterizes plantigrade gait is:

1. Heel strike
2. Knee flexion during the stance phase
3. Asynchronous EMG patterns
4. Pelvic rotation, tilting, and translation

An important aspect of plantigrade gait is the control of foot-ankle movements, which results in a clear heel strike. Forward propulsion is generated by ankle extension followed by knee flexion and hip flexion (Forsberg, 1989). Leonard et al. (1988) have shown that instead of developing plantigrade determinants, children with cerebral palsy retain a pattern resembling infant stepping. They do not develop a clear heel strike but initiate calf muscle activity at the end of the swing phase and begin plantar flexion before foot placement. At the end of the stance phase, there is no increase in calf muscle activity, leading to poor forward propulsion. The swing phase begins with active hip flexion followed by knee flexion and, finally, ankle extension. The ability to dorsiflex the ankle during the swing

phase to lift the foot off the ground is poorly developed. Co-activation in antagonistic muscles and short-latency spikes immediately after foot placement persist. Children often exhibit abnormal internal rotation from the hip, which, combined with increased adduction, leads to scissoring of the legs. Toe walking combined with scissoring results in an unbalanced and difficult-to-master gait. Electromyographic studies have shown that seemingly similar movement patterns can be caused by several different patterns of muscle activity (Chong, 1978; Sutherland, 1969). Chong et al. identified three types of abnormal muscle activity patterns in abnormal hip internal rotation. The most common pattern was activation of hip adductors and internal rotators during the normal part of the walking cycle but with phasic prolonged medial hamstring activity so that muscle force creating internal rotation was applied at an inappropriate part of the walking cycle. Sutherland et al. (1969) found that internal rotation was primarily caused by medial hamstrings and adductors, emphasizing the importance of a thorough examination of each child to identify the causes of internal rotation.

**Treatment of spastic gait**

A variety of treatment methods exist for treating spasticity, and more specifically spastic gait. Previously, there was a tendency to perform lower limb surgeries on young children more liberally, but now it is understood that the child's disability profile constantly changes and reaches a plateau around the age of 7. Therefore, surgical interventions should be postponed whenever possible. Otherwise, there is a risk of correcting issues that would have resolved on their own, leading to further complications (Patrick, 1989). In recent years, a new surgical method called rhizotomies has been introduced abroad, and in some cases, they have been able to reduce spasticity (Cioffi et al., 1990). Medications are only sparingly used for reducing spasticity in CP (McCarthy, 1984). Hershler et al. (1989) demonstrated how constant cerebellar stimulation can improve gait in an adult with CP. Horton and Taylor (1989) describe how behavior therapy combined with physical therapy can increase the ability to walk independently in children with CP and mental retardation.

Gordon (1987) describes how physical therapy treatment methods have evolved from so-called "muscle reeducation" theories, which focused on individual muscles, to the so-called neurotherapeutic methods, which aimed to treat the central nervous system itself. Neurotherapeutic theories suggested that it is possible to change or facilitate a patient's movement patterns by applying specific patterns through sensory stimulation, primarily via proprioceptive afferent pathways. The assumption was that sensory stimulation would produce lasting effects, facilitating changes in the CNS. Gordon argues that neurotherapeutic methods have significantly improved the treatment of neurological patients but that the

neurotherapeutic paradigm has encountered a crisis as patients have been unable to transfer the movement patterns they trained with facilitation to functional activities. Gordon calls for a paradigm shift to the so-called "Motor control perspective". In this perspective, the emphasis on neurophysiology is abandoned in favor of integrating neurophysiology, anatomy, muscle physiology, biomechanics, and behavioral science. Instead of focusing on how the CNS produces movements, the emphasis is on how the CNS gains control over the musculoskeletal system. Movements are used to solve specific problems or meet needs. Therefore, it is important for the patient to learn to solve problems in meaningful environments.

The physical therapy treatment methods often aim to inhibit spasticity and facilitate normal movements. Inhibition in patients with MS can be achieved, for example, through prolonged stretching of spastic muscles (Odeén, 1981), and this method is also used in the treatment of children with CP. Inhibition can also be accomplished through vibration (Eklund and Hagbarth, 1966), the use of rotational movements (Levitt, 1982; Atkinson, 1986), or placing the child in tone-inhibiting positions where spasticity is controlled from "key points of control" (Bryce, 1979). If the child can move with a more normal movement pattern after inhibition, it is believed that these patterns in themselves reduce spasticity (Levitt, 1982). Orthopedic aids are used to facilitate walking and correct misalignments in spastic children. Foot orthoses, shoe inserts, and derotation orthoses are examples of such aids. Both physical therapy treatment methods and orthopedic aids are rarely subjected to critical evaluation. They are often accepted or sometimes rejected based on tradition or subjective opinions.

**Derotation orthosis – (Twister cable orthosis)**
The derotation orthosis (Figure 1) has been used in Sweden since the 1970s. Initially, it was primarily used for children with spina bifida and later also for children with CP. In Sweden, the derotation orthosis had relatively little spread during the 1980s. In 1989, only 12 units were sold, and by 1990, sales increased to 30 units. In some parts of the country, the orthosis has been discontinued precisely because of subjective claims that it increases spasticity. The interest in studying the impact of the derotation orthosis on spasticity originates from:

1. Experience from children and parents who find the derotation orthosis to be a useful aid. It has been assumed that children and parents can evaluate and eliminate non-functional aids themselves.
2. Clinical observations indicating that the derotation orthosis improves children's walking patterns, primarily by reducing toe walking and scissoring tendencies.

3. An interest in the inhibitory effect of the rotational component in the treatment of spasticity.

According to the production description of the derotation orthosis, the orthosis positions the foot in the correct position for functional walking without hindering normal mobility in the hip and knee. It is suitable for correcting internal and external rotation. The derotation orthosis consists of a pelvic brace where a wire with heat shrink tubing attaches laterally on each side. The wire runs down to the child's shoe where it attaches to a fixed bracket on the lateral side. The wire is attached to the bracket on the shoe when the shoe is rotated outward approximately 90 degrees, adjusted according to the degree of inward rotation in the individual child. The wire is held against the child's leg with the help of Velcro straps around the calf and thigh. The brackets can be designed for permanent attachment to a pair of shoes or with a quick-release mechanism for interchangeable shoes. The derotation orthosis is produced as a semi-finished product and is customized in length by an orthopedic workshop and equipped with Velcro straps.

## PURPOSE OF THE STUDY

The purpose of this study was to investigate whether the derotation orthosis could alter muscle activity during walking in a child with cerebral palsy and how the derotation orthosis affects step length, step frequency, and walking speed. The research questions were as follows:

- Does muscle activity in spastic muscles and their antagonists change during walking with the derotation orthosis compared to walking without it?
- If muscle activity in spastic muscles changes, which specific muscles are affected - gastrocnemius and/or adductors - internal rotators?
- Are non-spastic muscle groups, such as abductors and dorsiflexors, affected in terms of their ability to activate during walking with the derotation orthosis?

## METHOD AND MATERIALS

The investigation took the form of an experimental study of one child. The child was selected from among those who had been using a derotation orthosis at Folke Bernadotte Home since 1985. It turned out that only 2 children were available for measurement, they were twins, and the older, slightly less affected girl was chosen due to her good cooperation and better walking ability.

**Participant**
A six-year-old girl, with normal cogntition diagnosed with moderate diplegic ataxic cerebral palsy. Her gross motor development level corresponded to approximately one to one and a half years, as she could stand alone and walk 6 to 7 steps with the derotation orthoses. Spasticity predominated on the left side with elements of hypotonia, especially on the right side. Grice arthrodesis had been performed on her right foot in March 1990. The girl had a tendency toward spastic adduction contracture in the left hip, which could be reduced after stretching and vibration treatment. She had been using the derotation orthosis since the age of 2. At the time of measurement, the girl was wearing a T-shirt, shorts, and sandal shoes connected to the derotation orthosis. Lange inserts were placed in the sandals. The sandals were rotated outward by 90 degrees when the orthosis was put on.

**Reference to Normal**
Reference to "normal person" in the results refers to adults who walked using the same measurement equipment also at the Tonus Laboratory at Karolinska Hospital (I Odeén 1990a).

**Experimental setup for electromyography (EMG)**
The electromyographic activity was wirelessly recorded using the Medinik IC-600 biotelemetry system and Beckman surface electrodes. The electrodes were embedded in soft plastic plates and fixed along the muscle belly with tape and elastic bandage. The muscles were manually palpated, and the attachment points were marked with a marker pen. The corresponding transmitter was attached with Velcro straps next to the electrodes (Odeén 1981). Muscle activity in the gastrocnemius, tibialis anterior, adductors, and gluteus medius was recorded. In the study, it was assumed that the adductors functioned as internal rotators, which has been commonly observed in children with CP (Chong 1978, Sutherland 1968). The participant walked 2 x 15 meters both with and without the derotation orthosis, always using a Key walker rollator. She was instructed to walk calmly at a normal pace, with minimal directives to preserve the naturalness of the gait.
Measurements were taken on the left leg:

1) Without derotation orthosis (trial 1 = T1)
2) With derotation orthosis immediately after T1 (trial 2 = T2)
3) With derotation orthosis 30 minutes after T1 (trial 3 = T3)
4) Resting muscle tone was recorded before T1 and after T3.

Between T2 and T3, the participant was free to engage in activities. She sat and ate ice cream in her father's lap, walked around the room, and played with a toy animal.

**Gait parameters**
To identify the gait cycles, two plates with four pressure-sensitive foot switches were used. These were taped directly onto the skin at the back edge of the heel and on the medial side of the forefoot. The girl wore her regular shoes during the gait registration. The foot switches were connected to a DC transmitter (Medinik). The transmitter was worn by the father in a waist belt, who walked slightly behind the girl during the measurements. The signals were transmitted telemetrically to a receiver, transformed through an amplifier (Grass 7 PIF), and printed on recording paper. A gait cycle was defined as the period between two consecutive heel strikes on the recorded leg. The total distance walked was marked successively, and the signals were stored in a matrix form and then printed out (Ericsson PC/printer) (Myrenberg et al. 1990).

**Video filming**
Video footage from the measurement occasion on May 29, 1990, and a video taken at the participant's home on September 17, 1990, were used to analyze the movement patterns in the hip, knee, and ankle joints. The video from the measurement occasion showed gait in the frontal plane, i.e., from the front and back, while the later video also showed gait in the sagittal plane. The filming was not standardized but was used only as a supplement to the EMG recording.

## RESULTS

**EMG-registrations and gait parameters**
Due to foot deformities, orthopedic inserts, and the participant's low weight, it was difficult to obtain well-defined signals from the foot contacts. Therefore, in the study of recruitment patterns, it was necessary to rely on the abductors, which showed a relatively normal pattern, as is common in individuals with CP. A ruler was placed vertically from the activation of the abductors at the beginning of the step down over the other 3 muscles. Evaluation of step length and step frequency could not be performed. The total walking time at the different measurement occasions is presented in Table 1. Resting activity before Trial 1 and after Trial 2 is reported in Table 2.

EMG recordings showed that the greatest activity in the spastic muscles occurred without the derotation orthosis. The reduction in spasticity was greater after 30 minutes than immediately after the application of the orthosis. Muscle activity in the Gastrocnemius was within normal limits after 30 minutes. The antagonist muscles to the spastic muscles could work better with the derotation orthosis in F3, and in F2, muscle activity was unaffected. For clarification of the following results, please refer to Table 3 and 4.

Gluteus medius abductor
Results showed that activity was more consistent and increased with derotation orthosis (F 2 and F 3). The recruitment pattern closely followed that of a normal person, with the highest activity occurring at 30% of the gait cycle, corresponding to the initial phase of the stance phase. The lowest activity was observed at 85% of the gait cycle, corresponding to the first half of the swing phase.

Adductors - internal rotators
Compared to a potential normal person (NP), the recruitment pattern deviated significantly. Specifically, there was much higher activity during the stance phase and co-contraction. A gradual reduction in peak activity was observed from F1 to F3. In experiment 3, the peak was reduced by 5.5 mm (10 mm = 50 microvolts) compared to F1.

Tibialis anterior
The muscle exhibited a reverse recruitment pattern compared to the normal person, which is common in individuals with CP (Odeén, 1990b). In normal individuals, the highest activity occurs at toe-off and initial heel strike. It then rapidly decreases and drops to 11.3 at 55% of the step - the end of the stance phase - before quickly increasing again during the swing phase to 31.5 mm at toe-off. In cp, there is a rising high activity during stance phase and lower activity during swing phase, which was also observed in this subject. The measurements showed that the tibialis anterior was minimally affected by the derotation orthosis. Also, in F3, the activity was uneven.

Gastrocnemius
In F1 and F2, the trials were difficult to interpret due to spasticity and reflexes. By F3, the pattern had normalized, and the activity had significantly reduced. The measurements fell completely within the normal range. The activity was also more consistent compared to F1 and F2. Figure 2 illustrates the EMG activity from the Gastrocnemius during the different trials.

**Video filming**
Without the derotation orthosis, the participant walked with internally rotated legs and on tiptoes. During turning, the toes of the left foot dragged on the ground. Occasionally, the knees collided during walking. The girl could put her entire foot down either by hyperextending the knee or by flexing the knee and hip joints. As a result, hip extension was less than when walking with the derotation orthosis. Sometimes, the hip abductors lost their stabilizing ability in the stance phase, and the child exhibited a Trendelenburg gait.

With the derotation orthosis, the girl walked on her entire foot. She had difficulty dorsiflexing the foot actively, even though the triceps surae was not spastic. This seemed to be related to her inability to fully extend the knee joint in the swing phase to achieve heel strike at the beginning of the stance phase. The hips were not internally rotated, hip extension was good, and the pelvis was stable.

**Figure 1**
Derotation orthosis (Twister cable orthosis)

**Figure 2.**
EMG-registration during gait from Gastrocnemius without derotation orthosis (F1), with derotation orthosis (F2) and with derotation orthosis after 30 minutes of use (F3). (note: figure is copy from original report)

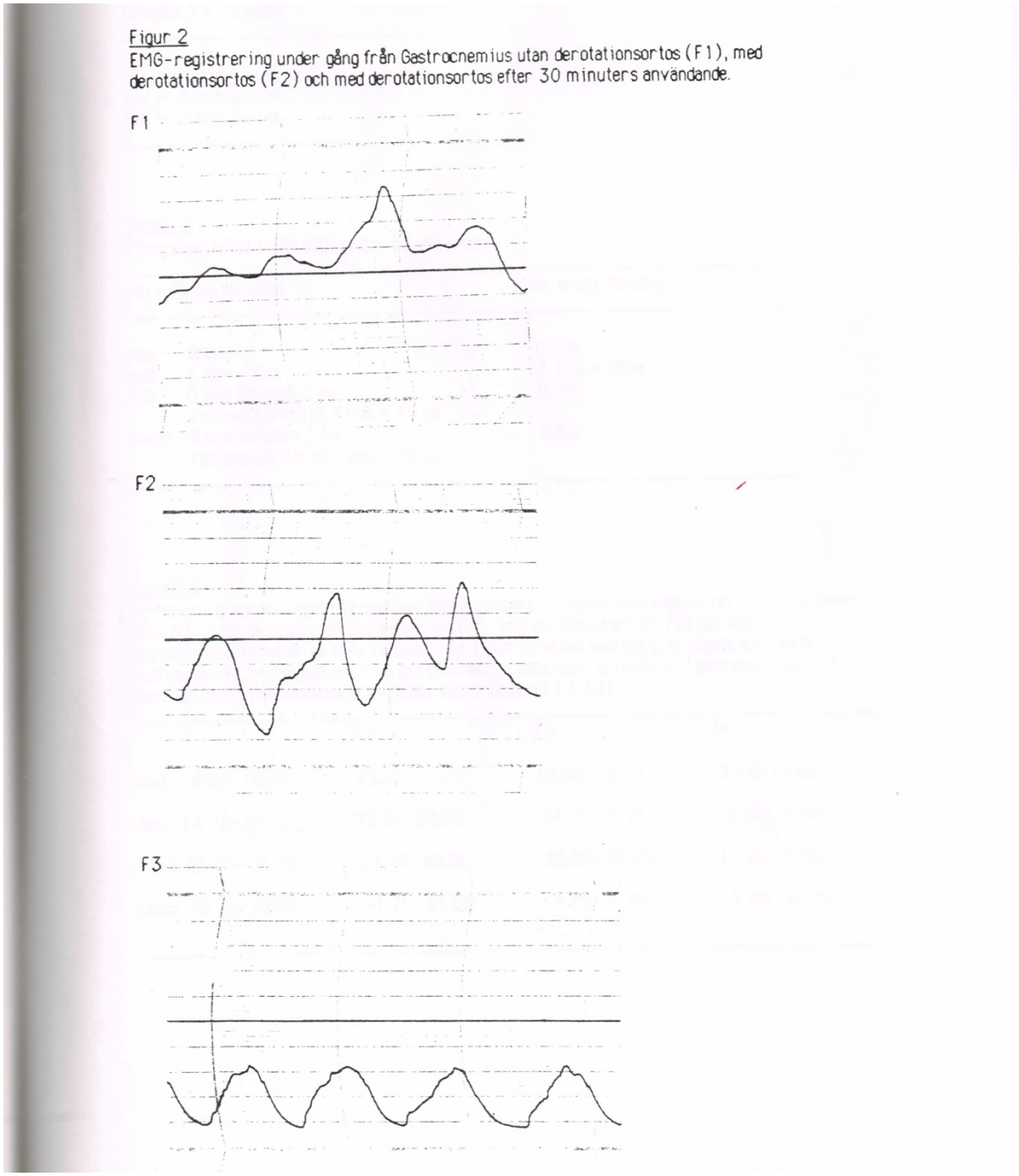


**Table1**

| | **Walking time 2x15 meters** |
|---|---|
| **F1** | 3 minutes 16 seconds |
| **F2** | 3 minutes 30 seconds |
| **F3** | 2 minutes 54 seconds |

**Table 2.**

| Muscle activity measured at rest during 20 seconds | | |
|---|---|---|
| | Before first trial | After third trial |
| Abductors | 0 mm | 0 mm |
| Adductors | 1 mm = 5 uϒ | 3 mm = 15 uϒ |
| Tibialis Anterior | 0 mm except 2 sec reflex activity at 3 mm = 15 uϒ | 0 mm |
| Gastrocnemius | 0 mm except 2 sec reflex activity at 3 mm = 15 uϒ | 0 mm |

**Table 3**

The arithmetic mean of the lowest and highest muscle activity in mm (10 mm = 50 uV) during walking without derotation orthosis (F1), with derotation orthosis (F2), and with derotation orthosis after half an hour (F3). The mean was calculated using the highest and lowest measurement values for 24 walking cycles. For example, a significant reduction in activity in Gastrocnemius is observed in F3, as well as an increase in abductor activity with the orthosis (F2 & F3).

| | F1 | F2 | F3 | NP |
|---|---|---|---|---|
| Abd | 8.50-16.25 | 13.50-18.25 | 13.00-18.00 | 5.50-17.00 |
| Add | 14.75–30.75 | 15.50-28.50 | 14.25-25.25 | 5.00-8.50 |
| Tib.A | 25.50–38.75 | 33.75–40.00 | 26.25–39.25 | 11.25-33.50 |
| Gastr. | 12.75–33.25 | 11.75–31.50 | 8.25-12.00 | 5.00-16.25 |

**Table 4**

The variation in muscle activity levels in different steps - the lowest (A) and highest (B) levels of muscle activity in mm (10 mm = 50 uV) during walking in a 6-year-old girl with CP (F1-F3) is compared to the highest and lowest levels of muscle activity in a normal person (NP). For example, completely normalized muscle activity is observed in the gastrocnemius in F3.

| | F1 | | F2 | | F3 | | NP | |
|---|---|---|---|---|---|---|---|---|
| | A | B | A | B | A | B | A | B |
| Abd | 2–8 | 3–18 | 2–11 | 12–19 | 2–15 | 2–21 | 5.5 | 17 |
| Add | 11–25 | 18–38 | 8–14 | 18–29 | 13–17 | 16–21 | 5 | 8.5 |
| Tib A | 19–35 | 17–45 | 11–40 | 27–45 | 15–22 | 20–31 | 11.25 | 33.5 |
| Gastr | 2–6 | 6–27 | 9–11 | 8–28 | 1–6 | 5–10 | 5 | 16.25 |

**DISCUSSION**

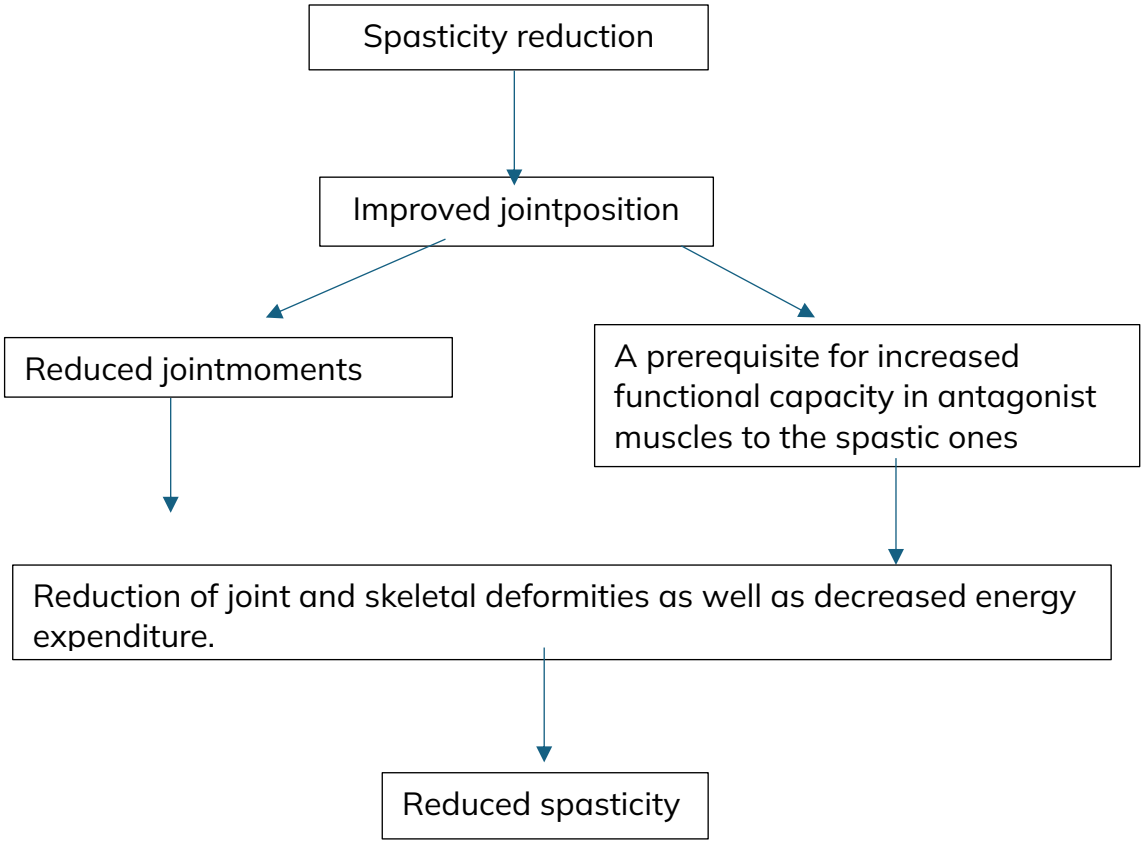
This experimental study only involved one child, so general conclusions cannot be drawn from the results. However, there is a need to critically evaluate orthopedic aids without preconceived notions (Currier 1984). In this case, it was difficult to conduct a larger study due to the limited availability of derotation orthoses. As previously mentioned, the study assumed that the adductors functioned as internal rotators, which is common in children with CP (Chong 1978, Sutherland 1968). Internal rotation can secondarily cause a valgus position of the foot (Villani et al 1989), but it does not directly cause plantar flexion of the ankle. Therefore, it is interesting to note how the derotation orthosis, by counteracting internal rotation, simultaneously normalized muscle activity in the foot's plantar flexors after 30 minutes of use. This reinforces claims that rotation has a generally spasticity-reducing effect, which has been clinically observed and mentioned in books on the treatment of children with cerebral palsy (Levitt 1982, Atkinson 1986) but has not been scientifically studied (Atkinson 1986).

The fact that the positive effect did not occur immediately after donning the orthosis but after 30 minutes raises speculation that the reduction occurred due to stretching of the internal rotator musculature. Odeén 1981 demonstrated that spasticity reduction of spastic muscles was achieved after 30 minutes of stretching in patients with MS. In Romanini's and Sabadini's study (cited in Villani 1989), it was shown that rotational deformities highly correlate with foot deformities and that foot deformities are much less common in those without rotation problems. These facts suggest that therapists who often try to address toe walking with various treatment methods around the ankle joint, such as shoe inserts, vibration, or taping, may potentially find a way to influence ankle position by counteracting internal rotation in the hip joint.

It is widely accepted that studies of gait must illuminate the foot, knee, and hip joints, as misalignment in one joint creates a dynamic compensatory misalignment in the other two joints (Lai et al. 1988). For example, toe walking can be countered

with knee flexion and hip flexion or with hyperextension of the knees and hyper lordosis of the lumbar spine.

Following this, if muscle activity is normalized, and for example, toe walking is addressed, not only the ankle joint but also the knee and hip joints will have less need to compensate for the foot misalignment. The gait pattern in children with CP is often a result of adaptation to the clinical condition (Lai et al. 1988). Lai et al. (1988) describe how dynamic misalignments associated with gait in CP produce significant increases in flexion and extension moments in the hip and knee, as well as higher moments during plantar flexion in the ankle joint. These high stresses lead to a significant increase in the force affecting the joints and soft tissues around them, and ultimately, soft tissues may be stretched, and skeletal deformities may become permanent. Therefore, it is important to identify the cause of toe walking, among other gait abnormalities, to treat it correctly. Toe walking in children with CP can sometimes be caused by excessive muscle activity in the triceps surae and sometimes by ankle contracture (Tardieu et al. 1989). These facts suggest that the ability of the derotation orthosis in this case to normalize gastrocnemius muscle activity and to assist the hip abductor muscle in working more effectively may yield more positive secondary effects. These secondary effects can be hypothetically described as follows:



In this study, four muscles were examined, but it may be valuable in future evaluations to increase the number of muscles studied. However, this must be weighed against the potential impact of the EMG equipment on the gait pattern. Yong et al. (1989) have shown that both surface electrodes and intramuscular electrodes affect the gait in children with CP. The use of surface electrodes showed fewer disturbances in the gait pattern than internal electrodes. With surface electrodes, the number of steps per minute (cadence) decreased. With internal electrodes, there was a decrease in stride length for both the measured and non-measured legs, cadence, and the number of centimeters walked per second. Therefore, surface electrodes may be preferred over internal electrodes both for obtaining a more accurate measurement of the gait pattern and from an ethical standpoint. In further evaluation, it would be beneficial to also compare the magnitude of joint moments with and without the derotational orthosis.

Evaluating from the perspective of a normal adult should not necessarily be a source of error. Csongradi et al. (1979) demonstrated in an EMG study that there were no significant variations in muscle activity related to gender or age in children aged between 3 and 17.8 years. One might speculate that an adult, due to better-developed gait automatism, walks more energy-efficiently than a child. The results regarding walking speed in this study should be interpreted cautiously as the participant in Experiment 2 stopped momentarily when disturbed by passersby. As mentioned, there are significant variations in symptoms and their causes in CP. Access to laboratories where children can be studied in motion is necessary to map the causes of symptoms (Patrick 1989). This should provide better conditions for accurately determining treatment interventions for gait training than a general treatment program can offer.

As previously mentioned, it is unclear whether spasticity or the development of postural reactions determines whether a child becomes an independent walker or not. This is valuable to consider when establishing a reasonable prognosis and realistic expectations for the child's functional ability. However, it does not diminish the importance of attempting to eliminate spasticity and thus the complications it secondarily causes. The goal must be to maximize the child's functional capacity and minimize secondary problems, regardless of whether the child develops independent walking ability. The conclusion of this study is that for the child measured, the derotation orthosis is a valuable walking aid and that the rotational component's ability to reduce spasticity should be considered in children with cerebral palsy.