Medical Research Archives

ESMED
European Society of Medicine

OPEN ACCESS

Published: April 30, 2023

Citation: Brunner R and Götz-Neumann K, 2023. A Critical View on the Importance of Treating Spasticity and New Options to Improve Function in Patients with Cerebral Palsy GMFCS I-III, Medical Research Archives, [online] 11(4). https://doi.org/10.18103/mra.v11i4.3812

DOI
https://doi.org/10.18103/mra.v11i4.3812

ISSN: 2375-1924

RESEARCH ARTICLE

A Critical View on the Importance of Treating Spasticity and New Options to Improve Function in Patients with Cerebral Palsy GMFCS I-III

Reinald Brunner[1+*], Kirsten Götz-Neumann[2+*]

[1] University Children's Hospital Basel, Spitalstrasse 33, 4056 Basel, Switzerland
[2] Observational Gait Instructor Group, O. G.I. G., 10285 Kilrenny Ave, 90064 Los Angeles, USA
+ shared first authorship

*Corresponding author: Reinald-g-h.brunner@unibas.ch or kirsten@gehen-verstehen.net

ABSTRACT
Spasticity in patients with cerebral palsy is still an unclear concept which bases on the lesion of the upper motor neuron. The consequence is a loss of reflex control. The diagnosis is made during the static clinical exam and is transported into function. Spasticity is seen as an important factor which causes functional handicap as well as functional and later structural deformities. Because spasticity is considered a deteriorating factor for function, treatment of spasticity is one of the main columns in the treatment of children with spastic cerebral palsy. However, the diagnosis of spasticity during function is not possible up to now. Recent publications even raise doubts on the efficacy of spasticity treatment. On the other hand, spasticity is seen as a factor to compensate for weakness. New studies highlight the possibility of conditioning reflexes leading to an increase or decrease of reflex expression, hence spasticity. These facts query the treatment of spasticity and pave the way to new treatment concepts where experiencing successful movements can condition control of reflexes positively, without treating spasticity. This paper looks at the recent literature on new aspects of spasticity and describes the possible therapeutic consequences. The main shift in the treatment concept is to help patients cope with their spasticity and discover movement strategies in a large spectrum of movements. We describe possible modifications of the current treatment concepts. Movements should be carried out as an automatic muscle activity to reach the goal of an action which lets the patients experience success and joy. Dynamic orthotics help to keep the body segments in a functional adequate alignment, for an optimal movement experience under gravity, while the therapist intends to activate necessary but inactive muscles including eccentric muscle contractions. The patients discover new abilities and learn to move in a more efficient way. New feed-forward movement strategies are built up which help for activities in general. The need of spasticity compensating for weakness is reduced. These principles should be transported into daily life and sports. Sportive activities involve these therapeutic aims and the patients challenge themselves when they endeavour to improve.

Keywords: Spasticity; CP; Functional treatment; New strategies for rehabilitation; Motor learning; Gait; Activity-based restorative aligned therapy

Introduction

Cerebral palsy (CP) is the most common cause of disability in childhood. It is defined as a lesion to the developing brain, which covers the pre-peri-, and early postnatal period. Predominantly one side, both legs, and the whole body can be affected. Besides ataxia and dystonia, spasticity is the most common type of motor control disorder. According to the severity of the functional impairment, patients are classified by the Gross Motor Function Classification System (GMFCS)—GMFCS I describe an almost normal situation, while GMFCS V typifies a complete loss of function [1]. In our paper, we focus on patients who still have the possibility of locomotion (GMFCS I-III). The main clinical impression is the motor dysfunction with jumpy movements, and stiffness, which results in inadequate joint positions. This symptomatology was labelled spasticity. Lance defined spasticity as "one component of the upper motor neuron syndrome which is characterised by a velocity dependent increase of the tonic stretch reflexes, with exaggerated tendon jerks, resulting from the hyperexcitability of the stretch reflex" [2]. CP was also first described as a spastic motor disorder by Sigmund Freud and Sir John Little [3]. Although other types of muscle tone disorders may be present, spasticity is considered especially problematic. These patients present with severe deformities and the strong convulsive muscle activities pull the joints in extreme positions. It is not surprising that a link is created between spasticity and deformities, and that other concomitant neurological symptoms have gained little attention. The current definition still describes CP as a paramount motor affection.

Today, the motor disorder in CP is regarded as the consequence of the UMNS (Upper Motor Neuron Syndrome) which is a combination of spasticity caused by hyperexcitability of spinal reflexes, weakness and loss of dexterity, muscle co-contraction, reduced movement selectivity, and synkinetic movements [4-7]. Spinal and brain plasticity and adaptive changes in muscle tissue modulate the clinical expression.

Spasticity as a predominant rise of muscle tone is defined as "resistance against fast movements by muscular contraction created by neuromuscular control." Besides resistance by muscle contracture, which is not spasticity, two types are distinguished: a more hyperreflexic type, and a general rise of tone with global stiffness [8]. The label "spasticity" describes these clinical aspects but remains rather imprecise especially regarding origin and pathophysiology. CP is seen as upper motor neuron syndrome, where one part concerns reflex hyperexcitability due to reduced inhibition on the second motor neuron in the spinal cord. This pathophysiological mechanism may apply in part for the increase of muscle-tendon jerks, and for jump-like movements. However, it has difficulty to explaining the general increase of muscle tone and the resulting stiffness. The various clinical pictures are explained by a various mix of tract lesions with excitatory and inhibitory control on spinal activity [5]. Plasticity of the brain partly explains the change of the clinical pictures over time [5]. Spasticity is seen today as abnormal spinal processing of normal input from the muscle spindles due to a loss of control by the upper motor neuron [6]. [Spinal] "motor neurons respond to stretch at a lower threshold than normal, with long discharges" [9]. Muscle tissue adapts at the condition of spasticity. The sarcomeres are relatively long, the connective tissue content increases, and pennation angle and fibre length change. The muscle becomes stiffer which is ascribed to titin. Contractures develop which reduce the range of motion. In the clinical setting, it may be difficult in some situations to distinguish well between the part attributable to spasticity and the part due to muscular changes [10-12]. Bar-On concludes in a review in 2015 that especially in clinical situations, it is impossible to distinguish properly between spasticity and hypertonia. The term spasticity is hence used in a broader sense [10]. Van den Noort suggests the distinction of muscular resistance due to non-neural (mainly muscular) origin and to neural origin. The latter is divided into stretch hyperreflexia (spasticity) and involuntary background activation (stiffness) [8]. Pandyan discusses the limitations of Lance's definition of spasticity in a review paper 2005 [13]: "There is insufficient evidence to support the hypothesis that the abnormal muscle activity observed in spasticity results exclusively from stretch reflex hyperexcitability." Other authors come to the same conclusion [11]. They suggest other additional pathways including afferent and supraspinal control pathways and conclude that spasticity is not a pure motor disorder [13]. Further, local sprouting from neighbouring interneurons leads to new abnormal neural connections which may explain the creation of new abnormal reflexes. This plasticity of neural connections built from local and descending pathways at spinal and brain levels is seen as one reason for the delayed appearance of spasticity [6]. Thus, the origin of spasticity is complex [7,10-12] and still unclear, and the views of experts vary. And despite of the fact that this hypothesis is actually still only partially proven, we use it as basis for our current treatment concepts.

This concept of hyperactive and unmasked reflexes is congruent in all review papers and is

generally well accepted. It is surprisingly rigid and always considers the present state of a patient as stable. Modifications by learning and reorganization of the nervous system are not considered. However, today there is evidence that even reflex control can be conditioned by a respective training program in healthy individuals and patients. This way, reflexes can get exaggerated or suppressed [14-16]. However, it does not always fit to clinical experience and picture. An abnormal reflex, the tonic stretch reflex, was introduced. This reflex was not found in animals nor in healthy humans. It remains, thus, hypothetical. Hagglund 2008 described a temporary increase of spasticity in children with CP between three and six years of age [17]. An explanation was not given. In clinical situations, we can see, that children walking while held by the mother are more relaxed than if they walk with aids or even on their own. Children with CP are more relaxed when lying down than when in an upright posture. Older, functional (CP GMFCS II and III) patients report that it is the fear of falling which makes them stiff. The basic disease is thereby not relevant: one gets this information from patients with CP or MS equally. The MS patients even state that stiffness increased the more they lost a proper feeling quality in the leg. It is far more difficult to get respective information from more affected patients with CP (GMFCS IV and V). Spasticity of syndromic or metabolic origin seems to be different from the ne in CP and less variable. However, understanding spasticity, its importance during function, and whether it is influenced during function on an objective basis is not possible today. Even gait analysis, with all its techniques for further assessing the gait data, does not provide a clear parameter. Van der Krogt related EMG activity to stretch velocity of the plantar flexors during swing phase of gait and found reduced velocity combined with increased muscle activity in spastic patients [18]. This means that even today we relate a stiff gait pattern in patients diagnosed with spasticity to spasticity. We extrapolate the clinical findings from the static assessment to function, without any proven evidence to which extent it is correct. However, we need to set a question mark on the general view on the importance of spasticity as no major change was found when a group with treated spasticity was compared with an untreated group on functional patients [19]. Not even deformities, life quality, endurance, or gait pattern were statistically different. We may have to reconsider our management of spasticity. In addition, stiffness does not always require spasticity. In cases of severe weakness and poor motor control, keeping the leg stiff during gait is a compensatory mechanism to avoid falling, and is in coherence with little or almost no EMG activity during gait.

Spasticity evolves over a few months but does not remain stable. In patients with CP, it peaks between the third and sixth year of life and decreases continuously until the end of the assessed period, at age twelve [17]. At the age of the peak, the children develop their strategies to cope with gravity. Healthy children as well show abnormal movements and control mechanisms initially at this phase of development, but much earlier in life as they are not retarded. Developing movement strategies is part of motor learning. The basic principle is trial and error: a variety of movements are carried out and evaluated which works best with one's individual anatomy. This approach enables children (and adults to a lesser degree) with even malformations to acquire a relatively good functional level. It is not only true for the locomotor system but for the individual with all aspects. If this concept is transferred to spasticity and reflex control, an improvement over time could be expected. This is in congruence with the description of the development of spasticity over time by Hagglund and Wagner [17]. There are indeed patients with CP who still present with extremely vibrant reflexes who by the age of adolescence have developed a normal pattern for walking and running, without any surgery or use training spasticity. Usually, they are GMFCS I with unimpaired cognitive functions. Other patients learn to use spasticity by hand grips or arm positions. Similar observations occur in healthy children: during the first year of life the so-called "primitive reflexes" are suppressed. Walking involves reflex activity, but these reflexes are controlled. This control can be measured by assessing the H-reflex during gait. The influence on spasticity by the upper central nervous system was already postulated in order to explain some changes in the expression of spasticity [6]. Spasticity is not a stable condition and it seems to be possible to gain some control.

As described above, motor learning requires assessing the efficacy and outcome of a new or altered movement strategy. There are two essential prerequisites: adequate sensory information and mental workup. Patients with multiple sclerosis describe the connection between loss of sensation, loss of confidence in the legs to support the body, and the increase of spasticity. For patients with CP, the affection of central sensory fibres was reported [20-23]. The affection of the sensory function concerns all qualities: the perception of pain may be abnormal, tension on muscle-tendon systems, capsules, and ligaments is altered or reduced, the representation of skin areas may be inadequate,

and the position of segments in space may be insecure. All these alterations lead to inadequate information to the central brain and impair a well-controlled and efficient motor output. Consequently, it must be far more difficult to develop an efficient movement pattern and hence, control over reflexes and muscle tone. More similar movements need to be carried out and more time is required which reflects in the developmental retardation, and in the longer time to develop a stable gait pattern—both parts of the typical sequelae of CP. Similarly, Lorentzen et al. describe the difficulty to build up feedforward programs for motion in CP children. They link it, however, more to weakness (which requires compensation), than to sensory deficits [24]. Besides the affection to the direct sensorimotor control, the lack of adequate perception results in a poor confidence in posture control under gravity. Feeling insecure with the fear of falling (with less possibilities to avoid being hurt) and the stress to cope with gravity results in a raise of muscle tone and a reduction of courage to try a wider movement spectrum as it is done by developing children. Stress hormones, therefore, have been found to be higher in children with CP than in their healthy peers [25,26]. Thus, in addition to the need for more information on movement, the movement experience gets reduced.

Muscle activity found during function which is aberrant from the normal pattern is often labelled "pathological" or "spastic." This assumption is difficult, as healthy individuals in normal postures for standing and mostly walking are compared with patients with a motor disorder due to a brain lesion which not only resulted in spasticity, but also in abnormal postures while standing and walking. As abnormal postures require different muscles to control, such a comparison can easily be erroneous. Indeed, healthy individuals mimicking the gait of hemiplegic CP were found to require the same muscle innervation pattern as seen in patients [27]. We suggest, that only the unexplained part of motor activity should be labelled "abnormal" or "spastic." One more point to consider is the concept that co-contractions of agonist and antagonist are an additional sign of spasticity and poor motor control. We scrutinize this idea as well. Agonist and antagonist act on the same joint but in different directions of motion. Ideally, these muscles are parallel with the origin and insertion on the same bones. Such a situation exists, for example, for the elbow, the biceps and the triceps brachii muscles. At the leg, however, such a situation does not exist functionally. Usually, knee extensors and knee flexors are considered to show co-contraction. This is an anatomically unique situation—where the knee extensors are to 80% monoarticular (the vasti), while the knee flexors (the hamstrings) are to 90% biarticular and bridge knee and hip joint. A co-contraction of these two muscle groups thus does not really lock the knee joint. In the case of knee flexion, the flexing effect of the hamstrings is avoided by the knee extensors, and the force of the hamstrings is shifted to the hip joint. This way, this co-contraction extends knee and hip [28]. In crouch gait this is a necessary and well controlled activity to control posture. If it is found in patients with good knee and hip extension, it can be the result of poor confidence in the legs. Similar is true for an increase of plantar flexors. This is more frequent in the case of weakness of any cause [29,30]. We explain this phenomenon in the situation of weakness and the resulting difficulty to maintain knee extension under load. The increase of plantar flexor activity pushes the tibia backwards and thus prevents knee flexion which would possibly be difficult to control with the risk of falling. This is not a spastic activity although poor motor control can lead to overactivity in these situations. In the case of foot instability, the tibialis anterior muscle can be active at the time of plantar flexor push. In this case, it stabilizes the midfoot and does not lock the ankle. Again, this is not a true agonist – antagonist situation as the two muscles differs in the insertion: the plantar flexors at the heel bone, the tibialis anterior at the basis of the first metatarsal bone. In between is the chain of midfoot joints. Again, a co-contraction is not necessarily a sign of poor motor control. However, co-contractions can be seen as reactions to poor posture.

Surprisingly, a much more general view on the situation of a patient is not common. These considerations lead to the consequence, that a differential diagnosis of abnormal muscle activity should be carried out before deciding on treatment. Regarding the new and stimulating literature, where reflexes can be modulated and spasticity including equinus and co-contractions are more seen as compensatory mechanisms for weakness and sensory dysfunction, new approaches for treatment management are offered. As treatment of spasticity has become questionable [19], and as learning to cope with spasticity is an option [15], we suggest offering a greater variability of activities in order to challenge motor control in spite of spasticity. The challenge will be to which degree struggling with spasticity can be regarded beneficial and when help is required.

Therapeutical outlook

Skeptical criticism on the current understanding and management of spasticity is of little help without an

outline of options to adapt our treatment protocol. Although a clear definition of spasticity and its causes is difficult, shown by the attempts to define it a new [31] or better [32], the importance of spasticity for gait and movement disorders seems to be greatly overestimated. New insights with a newer and wider spectrum of pathomechanisms offer new ways and concepts for treatment. In our present considerations for the causes of movement disorders, including spasticity in children with CP, we are looking only on the tip of the iceberg. When focusing on impairment and spasticity only, some simple but important paradigm shifts could help in detecting latent possibilities that are covered by challenges not only children with spastic CP have like biomechanically misaligned dynamic control patterns, but that also occur during any movement against gravity. A more thorough assessment and consecutive treatment also considers the other 90% of the iceberg. Such has been stated well by Hickam: "A man can have as many diseases as he damn well pleases“ [33].

To explore the many other causes for the dysfunction, we suggest a more diverse approach that includes biomechanical factors, mental-motor learning, and psychological factors. An important part of treatment should focus on movement discovery and movement choice instead of on impairment and on deficits. One of these deficits is spasticity. Curiosity and joy of movement could breed the necessary success in treating children with CP (Figure 1).

Figure 1 A: *Child with spastic CP GMFCS III enjoying hitting balloons.* The physiotherapist cues the patient gaining segmental control while the challenging, positive experience of dual-tasking stimulates non-voluntary motor control.
Figure 1 B: *EMG during the therapy session helps to verify that the target muscles are activated.*

The aim is to do a focused and individualized, rich, new experience of movement which greatly influences all activities. Broad, sportive activity in which the therapist will have to adapt the task to a level into which the child can learn more. Improving a child's ability by including climbing, judo, soccer, and others will provide more power to build up new movement and control strategies. Increasing the therapy effect —going above and beyond the Rehabilitation Center. With these suggestions we would like to shift the assessment and, consequently, the treatment approaches more towards discovering and acting on probably undetected latent potentials, instead of treating a symptom with all the "deficits." CP children at all GMFCS levels show plasticity for learning between age 2-5 years and reach a plateau at an age of 11-10 years in levels GMFMS levels I-II [34]. Despite the plateau concept, the brain does not lose the potential for learning. Moreover, it is cause for thought that instead of improvement, the opposite is true. Stabil levels are reported within 25 years of age, but the cohort group in a study of 6000 patients very seldom showed any

improvement. After all those years of "stable" ability, mobility declined [34] when becoming older adults (25-40 years) and getting worse [35], [36]; [37]. The concept of treating symptoms we struggle to fully understand but still try to treat, does not seem to produce stable beneficial results. Discovering hidden possibilities in children with CP to assess potentials, and acting and discovering potentials accordingly offers a new option for learning a broader motor control, which can be expected to be beneficial in the long term.

Discovering abilities versus treating spasticity

Some publications notes that children with CP will develop motor functions only up to a certain level [38] and there is hardly any change to reach a better GMFCS level for the rest of their childhood [39]. This permanently reported restriction of improvement is questioned by other publications that report groundbreaking possibilities and drastic change. Regarding spasticity as a compensation for weakness [24], the possibility of operant conditioning spastic reflexes and focusing on biomechanically challenging gait functions, our present treatment concept requires adaptations [40,41]. Working under load and with an improved alignment (from head until toe during walking) aims at discovering and recovering sensorimotor functions of individuals with CP. When spasticity as a present factor is accepted, and functional deformities are controlled as best as possible, the patient can learn the best way to cope with weakness and spasticity. If the aim of motor action is better achieved with a correct segmental alignment, this positive perception of success can motivate the patient to learn the new strategy, and can reduce the importance of spasticity and its control [6,15] (Figure 2).

The importance how the overall function of the individual with CP impairments influences the development of the nervous system, needs to be recognized. As a general principle, therapeutic strategies to recover functions of individuals with CP should comprise activities which can be accomplished by stimulation of the neuromuscular system with a variability of activity-based locomotor training, that is not restricted or inhibited e.g. by medication. This is supported by a recent review showing contradictory results of weakening the already weak muscles even more [42] or by the report of poor long term effect of spasticity treatment [19]. Rather we enable the child with targeted age-appropriate activation within meaningful self-chosen goals in order to intrinsically motivate for the new movement pattern "play" an engaging well-chosen frame – so the child can experience new movement variabilities within an intensive training of 1h a day/ 5days a week. We see an important point to guide the center of mass by trunk and hip alignment during activity based restorative locomotion therapy instead of learning to use the arms as support while using a walker too early. This will enable learning to adapt and take advantage of the control of spinal and supra-spinal feedforward motor programs which require several years of practice during childhood to become efficient and precise as in adults [24]. This is in contrast with the conventional therapy of approximately 2 x a week for a maximum of 45min. Hip flexion causing knee flexion can't break the pathological chain of a single movement choice. Instead of having a treadmill just in a therapy session, it would be important to have some more joy in gaining motor skills, even at home. In this context a rehab-buddy can be helpful, which transfers the gained new spectrum of movement choices from the therapy session into the daily life. At school, table bicycles instead of chairs could offer new movement, diminish the training deficit and weakness.

Lorentzen et al. sees co-contraction is a reasonable strategy to adopt when muscles are weak (demonstrated by most children with CP) [24]. Instead of diminishing excessive muscle activity through anti-spasticity medication, the therapy focusses in assisting the child to learn the most efficient gait patterns and remove the dynamic biomechanical constraints. In fact, we observed a change from toe walking into a heel toe gait within just one session of 1,5h treatment when addressing the biomechanically constraints (Figure 3a, b).

MacKenzie Goode-Roberts et al. described a successful case with a 15-month-old premature child with spinal cord injury, that experienced great success to transform the onset of "spasticity" into appropriate kinematics of this males in hand operation and independent stepping with hip guides, when treatment was changed into rich sensorimotor experiences with intensive, repetitive, progressive practice [43]. This treatment based on the concept of a biomechanically correct alignment, and the activity-dependent plasticity and the role of spinal cord in controlling movements [44]. In contrast to treating spasticity by weakening means such as botulinum toxin, spasticity is used as a compensation for weakness. The patients, however, need to learn to cope with their spasticity best to functionally improve.

During loading response, the ground reaction force tops body weight. As we lack perception of gravity, the external forces are underestimated in clinical surroundings. At the very beginning of the gait cycle every human needs to be able to

counteract the highest torque (within the whole gait cycle) at the hip – during Loading Response (LR) [45] in all three planes. Milliseconds of muscle activation patterns are needed to stabilize against the ground reaction forces which would pull accordingly every human naturally into hip adduction, flexion and internal rotation [46]. The muscles needed to counteract the external forces are not just abductors, extensors and external rotators at the hip, but also the medially acting M. adductor magnus. This muscle acts mainly as an extensor in cooperation of the lateral stabilizers of the hip abductors and extensors (M. gluteus max., M. gluteus med., etc.) [45,46]. In children with CP, hip internal rotation, flexion, and adduction is a common pattern during gait. Femoral anteversion and internal rotation reduce the lever arm for these muscles, and some may become long. In a recent study Thielen et al. [47] showed in healthy subjects artificially bandaged into hip internal rotation, that internal abduction forces were drastically reduced, similar to what happens within children with CP. The literature does not differentiate between different adductor muscles when "treating adductors". While the adductor longus flexes the hip in preparation for swing, the adductor magnus works as a very important medially stabilizer and extensor during Loading response in gait and hence counteracts GRF pulling the hip into flexion in sagittal plane. Sufficient neuromuscular strength is necessary to counteract GRF and stabilize the hip during LR by peak abductor strength (M. gluteus max., M. gluteus med., etc.) and adductor magnus from medial as a hip extensor. This difference should be respected when "adductor spasticity" is reduced. Once a medial collapse [45] occurs, the adductor magnus stabilizes the hip as an extensor.

Figure 2 A: *Child with spastic CP standing. Correct segmental alignment is provided, while reaching for a new movement experience (instead of collapsing into sitting or valgus)*

Figure 2 B: *Same child improved. Sufficient dynamic postural alignment and control enables her to successfully blow soap bubbles with improved breathing function.*

Figure 3 A, B: A: *Child with spastic distal CP GMFCS III, 2 years after SDR, with exercise on a treadmill. Poor segmental alignment leads to abnormal posture with higher demand to use the upper extremities for stability.* ***B****; Control of the leg segments (corrective dynamic trunk-hip-knee orthosis) improves posture and stability.*

This activity must not be regarded as "spasticity" but biomechanically necessary for hip stability. As in the paper "from spasticity to activity" described [40] [48], focusing on a more neutral alignment of the hip, especially during landing and weight acceptance phase of the gait, by avoiding excessive adduction with simultaneously focusing on activation hip extensors and external rotators showed drastic improved gait functions. Muscle activity in abnormal posture is not necessarily pathological and even more not spastic, the spot light needs to be shifted at a biomechanically correct alignment and thus optimizing muscle activity instead of deactivating or performing surgically tenotomy of all adductors to treat "spasticity."

Not every hypertonicity even in the upper extremity needs to be regarded as a result of a supraspinal problem caused by CP. Michaelsen et al. [49] showed in their study with adult patients with chronic stroke – that reaching functions with drastic improved arm range of motion was regained, by simply restraining the compensatory movement of the trunk moving forward. They suggested that there are latent "normal" movement patterns still available and not entirely lost and can be uncovered. One cost of this gaining "normal reaching pattern" and recovery would be temporally decrease in movement speed. In a chronic phase to promote compensatory strategies to maximizing function may not be the right approach. CP children in contrast did not build up a normal movement strategy as patients with an insult later in life. This does not implicate, however, that they are not capable of learning. Within children with CP, maladaptive patterns can be even worse, as their latent abilities, once discovered may improve their GMFMS or Manual Ability Classification System (MACS). But the opposite is true, instead of improving they show early on to be leveled as shown [50], or even decline [36], [39]. 70% of the kids with CP with all GMFCS levels in their first MACS classification decrease over 5 years. The MACS may not be intended to show improvements after therapy. However, when a patient tries to become more independent as adult, plateauing and decline, frustration and consequences are reported from both sides. Parents [51] as well as their children with CP become exhausted [52]. This indicates, that in the pharmacological modalities especially when

administered alone might generally "not be very effective in reducing muscle tone or stiffness or improving gait patterns" [42]. Adolph et al. [53] point out in their publication; "How do you learn to walk? Thousands of Steps and dozens of falls"; that researcher's ignore about "infants` natural experience with locomotion" and have a conceptual misinterpretation of experience. There is a difference between the number of days since walking than the test age (number of days since birth). This in fact is particularly important when designing walking rehabilitation for children with CP. The number of days since birth do not at all reflect the number of days experienced since the onset of walking for children with CP. The "Walking age" [54] in children with CP differ extremely from their typically developing people day of birth.

The adult mature walking pattern differs a lot from the one of toddlers and baby beginners walking pattern. Plantigrade walking patterns aren't unusual within human walking beginners. By trial and error, the best strategy in respect of the individual anatomical and neurological situation is found. Experience on the base of adequate sensation and perception hence is essential for developing a normal movement pattern.

When looking into walking rehabilitation in children with spastic CP (SCP) "falling", or better learning to control the forward falling center of mass, which is eccentric muscle control, needs to be trained. If children with CP only walk with walkers and other walking aids, they learn a kind of quadruped walk with their arms, but not how to control the center of mass on top of their legs. Bringing the center of mass forward, even in front of the base of support, should be included in our treatment although there may be a fear to overchallenge the child [55]. This challenge needs to be addressed within our treatment as the only way of "mastery" to learn walking into an adult or developed version of walking. Essential are early onset of activity-based treatment with biomechanically correct segmental alignment and a high load during daily activities. One case with good outcome has been reported [43], but future research needs to check the efficacy of the new treatment strategy.

Conclusion

Spasticity can be regarded at least to some degree as a compensation for muscle weakness. Without correctly aligning the body segments under load, it results in a lack of building up an appropriate feed forward strategy. The therapist can help controlling the segments correctly in order to enable better motor learning. Muscle activity deviating from reference values of healthy individuals must be distinguished into abnormal (but adequate for the biomechanical situation) and pathological (which could be spasticity). However, the diagnosis of spasticity during function has failed up to now. Motor learning as another factor manifests the pattern. Reflex activity as part of spasticity can be modulated, and treatment of spasticity is increasingly scrutinized. Improvement of understanding the background causes of spasticity and its nature opens new treatment options. One aim is to increase the movement experience with positive perception of success in general but not focusing on gait only. While a variety of sports is an option for daily life, new movement choice discovery is a therapeutic task within the treatment design.

Conflict of interest

There are no conflicts of interest to be declared.

References

1. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S. *Dev Med Child Neurol*. Apr 1997;39(4):214-23.
2. Lance JW. Symposium synopsis. In: Young RR, Koella WP, eds. *Spasticity: disordered motor control*. Yearbook Medical Publishers; 1980:485-494.
3. Schifrin BS, Longo LD. William John Little and cerebral palsy. A reappraisal. *Eur J Obstet Gynecol Reprod Biol*. Jun 2000;90(2):139-44. doi:10.1016/s0301-2115(00)00261-x
4. Burke D, Wissel J, Donnan GA. Pathophysiology of spasticity in stroke. *Neurology*. Jan 15 2013;80(3 Suppl 2):S20-6. doi:10.1212/WNL.0b013e31827624a7
5. Sheean G. The pathophysiology of spasticity. *Eur J Neurol*. May 2002;9 Suppl 1:3-9; dicussion 53-61. doi:10.1046/j.1468-1331.2002.0090s1003.x
6. Trompetto C, Marinelli L, Mori L, et al. Pathophysiology of spasticity: implications for neurorehabilitation. *Biomed Res Int*. 2014;2014:354906. doi:10.1155/2014/354906
7. Ward AB. A literature review of the pathophysiology and onset of post-stroke spasticity. *Eur J Neurol*. Jan 2012;19(1):21-7. doi:10.1111/j.1468-1331.2011.03448.x
8. van den Noort JC, Bar-On L, Aertbelien E, et al. European consensus on the concepts and measurement of the pathophysiological neuromuscular responses to passive muscle stretch. *Eur J Neurol*. Jul 2017;24(7):981-e38. doi:10.1111/ene.13322
9. Kheder A, Nair KP. Spasticity: pathophysiology, evaluation and management. *Pract Neurol*. Oct 2012;12(5):289-98. doi:10.1136/practneurol-2011-000155
10. Bar-On L, Molenaers G, Aertbeliën E, et al. Spasticity and its contribution to hypertonia in cerebral palsy. *Biomed Res Int*. 2015;2015:317047. doi:10.1155/2015/317047
11. Nielsen JB, Petersen NT, Crone C, Sinkjaer T. Stretch reflex regulation in healthy subjects and patients with spasticity. *Neuromodulation*. Jan 2005;8(1):49-57. doi:10.1111/j.1094-7159.2005.05220.x
12. Nielsen JB, Crone C, Hultborn H. The spinal pathophysiology of spasticity--from a basic science point of view. *Acta Physiol (Oxf)*. Feb 2007;189(2):171-80. doi:10.1111/j.1748-1716.2006.01652.x
13. Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disability and rehabilitation*. Jan 7-21 2005;27(1-2):2-6. doi:10.1080/09638280400014576
14. Chen XY, Chen Y, Wang Y, et al. Reflex conditioning: a new strategy for improving motor function after spinal cord injury. *Ann N Y Acad Sci*. Jun 2010;1198 Suppl 1(Suppl 1):E12-21. doi:10.1111/j.1749-6632.2010.05565.x
15. Song S, Geyer H. A neural circuitry that emphasizes spinal feedback generates diverse behaviours of human locomotion. *J Physiol*. Aug 15 2015;593(16):3493-511. doi:10.1113/JP270228
16. Thompson AK, Wolpaw JR. H-reflex conditioning during locomotion in people with spinal cord injury. *J Physiol*. May 2021;599(9):2453-2469. doi:10.1113/JP278173
17. Hagglund G, Wagner P. Development of spasticity with age in a total population of children with cerebral palsy. Research Support, Non-U.S. Gov't. *BMC musculoskeletal disorders*. 2008;9:150. doi:10.1186/1471-2474-9-150
18. van der Krogt MM, Doorenbosch CA, Becher JG, Harlaar J. Dynamic spasticity of plantar flexor muscles in cerebral palsy gait. *J Rehabil Med*. Jul 2010;42(7):656-63. doi:10.2340/16501977-0579
19. MacWilliams BA, McMulkin ML, Duffy EA, et al. Long-term effects of spasticity treatment, including selective dorsal rhizotomy, for individuals with cerebral palsy. *Dev Med Child Neurol*. Nov 10 2021;doi:10.1111/dmcn.15075
20. Trivedi R, Agarwal S, Shah V, et al. Correlation of quantitative sensorimotor tractography with clinical grade of cerebral palsy. *Neuroradiology*. Aug 2010;52(8):759-65. doi:10.1007/s00234-010-0703-8
21. Yoshida S, Hayakawa K, Yamamoto A, et al. Quantitative diffusion tensor tractography of the motor and sensory tract in children with cerebral palsy. *Dev Med Child Neurol*. Oct 2010;52(10):935-40. doi:10.1111/j.1469-8749.2010.03669.x
22. Dinomais M, Groeschel S, Staudt M, Krageloh-Mann I, Wilke M. Relationship between functional connectivity and sensory impairment: red flag or red herring? *Hum Brain Mapp*. Mar 2012;33(3):628-38. doi:10.1002/hbm.21227
23. Staudt M. (Re-)organization of the developing human brain following periventricular white matter lesions. *Neurosci Biobehav Rev*. 2007;31(8):1150-6. doi:10.1016/j.neubiorev.2007.05.005

24. Lorentzen J, Willerslev-Olsen M, Huche Larsen H, Farmer SF, Nielsen JB. Maturation of feedforward toe walking motor program is impaired in children with cerebral palsy. *Brain*. Mar 1 2019;142(3):526-541. doi:10.1093/brain/awz002

25. Duran-Carabali LE, Henao-Pacheco ML, Gonzalez-Clavijo AM, Duenas Z. Salivary alpha amylase and cortisol levels as stress biomarkers in children with cerebral palsy and their association with a physical therapy program. *Res Dev Disabil*. Jan 2021;108:103807. doi:10.1016/j.ridd.2020.103807

26. Zhao X, Chen M, Du S, Li H, Li X. Evaluation of stress and pain in young children with cerebral palsy during early developmental intervention programs: a descriptive study. *American journal of physical medicine & rehabilitation*. Mar 2015;94(3):169-75; quiz 176-9. doi:10.1097/PHM.0000000000000252

27. Romkes J, Brunner R. An electromyographic analysis of obligatory (hemiplegic cerebral palsy) and voluntary (normal) unilateral toe-walking. *Gait & posture*. Oct 2007;26(4):577-86.

28. Frigo C, Pavan EE, Brunner R. A dynamic model of quadriceps and hamstrings function. Research Support, Non-U.S. Gov't. *Gait & posture*. Jan 2010;31(1):100-3. doi:10.1016/j.gaitpost.2009.09.006

29. Brunner R, Romkes J. Abnormal EMG muscle activity during gait in patients without neurological disorders. *Gait & posture*. Apr 2008;27(3):399-407.

30. Schweizer K, Romkes J, Brunner R. The association between premature plantarflexor muscle activity, muscle strength, and equinus gait in patients with various pathologies. *Res Dev Disabil*. Jun 10 2013;34(9):2676-2683. doi:10.1016/j.ridd.2013.05.025

31. Li S, Francisco GE, Rymer WZ. A New Definition of Poststroke Spasticity and the Interference of Spasticity With Motor Recovery From Acute to Chronic Stages. *Neurorehabil Neural Repair*. Jul 2021;35(7):601-610. doi:10.1177/15459683211011214

32. Dressler D, Bhidayasiri R, Bohlega S, et al. Defining spasticity: a new approach considering current movement disorders terminology and botulinum toxin therapy. *J Neurol*. Apr 2018;265(4):856-862. doi:10.1007/s00415-018-8759-1

33. Freixa M, Simões AF, Rodrigues JB, Úria S, da Silva GN. Occam's razor versus Hickam's dictum: two very rare tumours in one single patient. *Oxford Medical Case Reports*. 2019;2019(5)doi:10.1093/omcr/omz029

34. van Gorp M, Roebroeck ME, Swan Tan S, et al. Activity Performance Curves of Individuals With Cerebral Palsy. *Pediatrics*. Nov 2018;142(5)doi:10.1542/peds.2017-3723

35. Day SM, Wu YW, Strauss DJ, Shavelle RM, Reynolds RJ. Change in ambulatory ability of adolescents and young adults with cerebral palsy. *Dev Med Child Neurol*. Sep 2007;49(9):647-53. doi:10.1111/j.1469-8749.2007.00647.x

36. Hanna SE, Rosenbaum PL, Bartlett DJ, et al. Stability and decline in gross motor function among children and youth with cerebral palsy aged 2 to 21 years. *Dev Med Child Neurol*. Apr 2009;51(4):295-302. doi:10.1111/j.1469-8749.2008.03196.x

37. Benner JL, Hilberink SR, Veenis T, Stam HJ, van der Slot WM, Roebroeck ME. Long-Term Deterioration of Perceived Health and Functioning in Adults With Cerebral Palsy. *Arch Phys Med Rehabil*. Nov 2017;98(11):2196-2205.e1. doi:10.1016/j.apmr.2017.03.013

38. Jan Lieber, Petra Marsico, Labruyère R. Interventionen früh beginnen. *physiopraxis Das Fachmagazin für Physiotherapie*. 2021;09.2021doi:DOI: 10.1055/a-1582-6009

39. Alriksson-Schmidt A, Nordmark E, Czuba T, Westbom L. Stability of the Gross Motor Function Classification System in children and adolescents with cerebral palsy: a retrospective cohort registry study. *Dev Med Child Neurol*. Jun 2017;59(6):641-646. doi:10.1111/dmcn.13385

40. Götz-Neumann K. Von Spastizität zu Aktivität: Exzitation statt Inhibition. *physioscience*. 2014;10(03):115-125. doi:10.1055/s-0034-1384954

41. Mrachacz-Kersting N, Kersting UG, de Brito Silva P, et al. Acquisition of a simple motor skill: task-dependent adaptation and long-term changes in the human soleus stretch reflex. *J Neurophysiol*. Jul 1 2019;122(1):435-446. doi:10.1152/jn.00211.2019

42. Kaya Keles CS, Ates F. Botulinum Toxin Intervention in Cerebral Palsy-Induced Spasticity Management: Projected and Contradictory Effects on Skeletal Muscles. *Toxins (Basel)*. Nov 8 2022;14(11)doi:10.3390/toxins14110772

43. Goode-Roberts M, Noonan K, Stout D, et al. Case Report: Capitalizing on Development and Activity-Dependent Plasticity, an Interaction With Pediatric-Onset Spinal Cord Injury. *Front Pediatr*. 2022;10:804622. doi:10.3389/fped.2022.804622

44. Roy RR, Harkema SJ, Edgerton VR. Basic concepts of activity-based interventions for improved recovery of motor function after spinal cord injury. *Archives of physical medicine and*

rehabilitation. Sep 2012;93(9):1487-97. doi:10.1016/j.apmr.2012.04.034

45. Götz-Neumann K., Gehen Verstehen Ganganalyse in der Physiotherapie, Georg Thieme Verlag, 4. Auflage 2016, page 124 Medialer Kollaps.

46. Burnfield JPaJM. SLACK Health Care Books and Journals releases 'Gait Analysis: Normal and Pathological Function' Second Edition. *Orthopedics Today*. 2010;30(3):70.

47. Thielen M, Waible D, Krautwurst BK, Wolf SI, Dreher T. Effects of artificially induced bilateral internal rotation gait on gait kinematics and kinetics. *Gait & posture*. 2022;95:204-209. doi:10.1016/j.gaitpost.2022.05.003

48. Götz-Neumann K. From Spasticity to Activity: Excitation instead of Inhibition
Accurate Observing Gait Analysis and O. G. I. G. Gait Therapy
in Infantile Cerebral Palsy. *physioscience*. 2014;10:115–125. doi:DOI http://dx.doi.org/10.1055/s-0034-1384954

49. Michaelsen SM, Luta A, Roby-Brami A, Levin MF. Effect of trunk restraint on the recovery of reaching movements in hemiparetic patients. *Stroke*. Aug 2001;32(8):1875-83. doi:10.1161/01.str.32.8.1875

50. Ohrvall AM, Krumlinde-Sundholm L, Eliasson AC. The stability of the Manual Ability Classification System over time. *Dev Med Child Neurol*. Feb 2014;56(2):185-9. doi:10.1111/dmcn.12348

51. Silverio APM, Mancini MC, Antunes FIT, Figueiredo PRP, Bueno KMP, Brandao MB. 'Thinking about myself?' Experiences of parents of adolescents with cerebral palsy: A qualitative study to guide the implementation of a service for families. *Child Care Health Dev*. Jan 3 2023;doi:10.1111/cch.13094

52. Freeman M, Stewart D, Cunningham CE, Gorter JW. "If I had been given that information back then": An interpretive description exploring the information needs of adults with cerebral palsy looking back on their transition to adulthood. *Child Care Health Dev*. Sep 2018;44(5):689-696. doi:10.1111/cch.12579

53. Adolph KE, Cole WG, Komati M, et al. How Do You Learn to Walk? Thousands of Steps and Dozens of Falls per Day. *Psychological science*. 2012;23(11):1387-1394. doi:10.1177/0956797612446346

54. Clark JE, Whitall J, Phillips SJ. Human interlimb coordination: the first 6 months of independent walking. *Dev Psychobiol*. Jul 1988;21(5):445-56. doi:10.1002/dev.420210504

55. Han D, Adolph KE. The impact of errors in infant development: Falling like a baby. *Dev Sci*. Sep 2021;24(5):e13069. doi:10.1111/desc.13069