Gait impairments after stroke

Towards optimized assessment and tailored use of medical-technical interventions



Bente Bloks

DONDERS SERIES

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Gait impairments after stroke

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Chapter 1

General introduction

General introduction

Stroke is one of the major causes of disability in the world, resulting from vascular occlusion or hemorrhage in the brain [1, 2]. This may cause damage to the upper motor neurons within the central nervous system [3]. As upper motor neurons play a key role in initiating and modulating voluntary movements, an upper motor neuron lesion leads to significant motor dysfunction (Box 1) [3, 4]. In the case of cerebral hemisphere stroke, a distinct set of clinical symptoms typically manifest on the contralesional side, including muscle weakness, impaired motor selectivity, and spasticity [3-5]. These motor deficits often lead to gait impairments [4]. As gait capacity is a critical component of ambulatory physical activity [6], social participation [7], and overall well-being [8], disruptions in gait capacity can have a significant effect on quality of life [9].

Box 1. Upper motor neuron lesion following stroke

Upper motor neurons in the central nervous system are responsible for transmitting signals related to voluntary movements. The primary neural pathway for these signals is the pyramidal tract, which comprises the corticospinal tract [3]. This tract extends from the cortex down to the spinal cord, enabling precise movements of the limbs and trunk. Most (about 90%) of the corticospinal neurons cross to the contralateral side at the level of the medulla (lateral corticospinal tract), allowing them to innervate the contralateral limb muscles (Figure 1) [10].

In the case of damage to the lateral corticospinal tract due to stroke, compensation for the loss of motor control can occur through the corticoreticulospinal pathway [4, 11]. This pathway comprises the corticoreticular tract, which originates from the cortex and projects to the reticular formation in the brainstem, and the lateral reticulospinal tract, which descends from the reticular formation down to the spinal cord. However, compared to the lateral corticospinal tract, the lateral reticulospinal tract exhibits reduced motor selectivity, limiting its ability to produce refined movements [12]. More specifically, the corticoreticulospinal pathway is much stronger connected to the ankle plantar flexors (triceps surae muscles) and knee extensors (quadriceps muscles) compared to the ankle dorsiflexors (tibialis anterior muscle) and knee flexors (hamstring muscles), resulting in a bias towards ankle plantar flexors and knee extensors in the recovery of muscle strength and control [4, 13]. In addition, the corticoreticular fibers from the affected hemisphere are often damaged by stroke as well, leading to reduced facilitation of the lateral reticulospinal tract. As the lateral reticulospinal tract also exerts a strong inhibitory effect on the lower limb stretch reflexes, damage to the corticoreticular fibers often results in contralaterally increased muscle tone and other spasticity-related symptoms [12, 14].

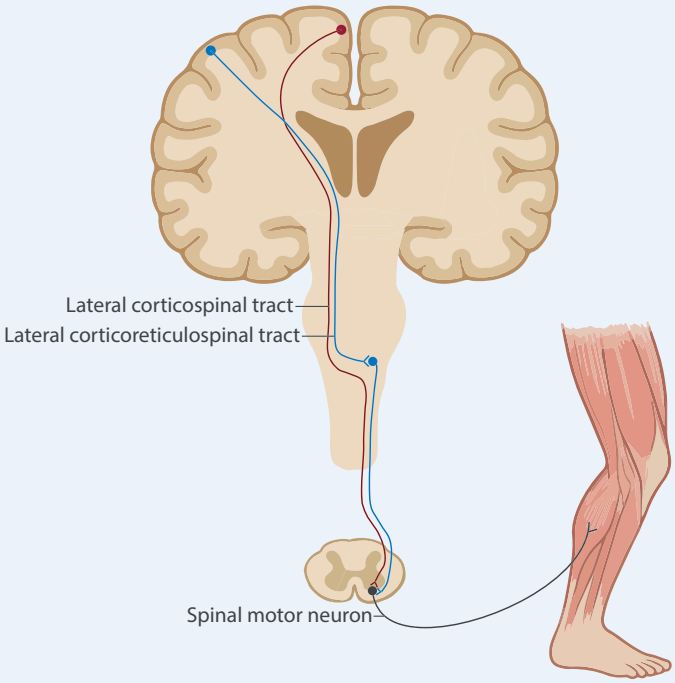


Figure 1. Innervation of spinal motor neurons to the lower limb by the lateral corticospinal and corticoreticulospinal tracts.

Post-stroke gait

Efficient and safe gait in people with stroke is determined by three fundamental prerequisites: 1) gait independence, 2) gait adaptability, and 3) gait pattern (Figure 2) [15, 16]. In turn, these prerequisites are (to different degrees) influenced by trunk and standing balance control, as well as motor control of both the paretic and non-paretic leg.

Gait independence is defined as the ability to walk without assistance or supervision from others, either with or without using a walking aid. The primary factor contributing to gait independence is standing balance control, which is strongly influenced by the ability to maintain trunk stability ("axial muscle control") [17, 18]. The gait pattern is characterized by a sequence of repetitive and coupled movements of body segments (Box 2) [19]. For achieving a proper gait pattern, adequate motor control of both legs is essential. This requires sufficient muscle strength and joint range of motion, and

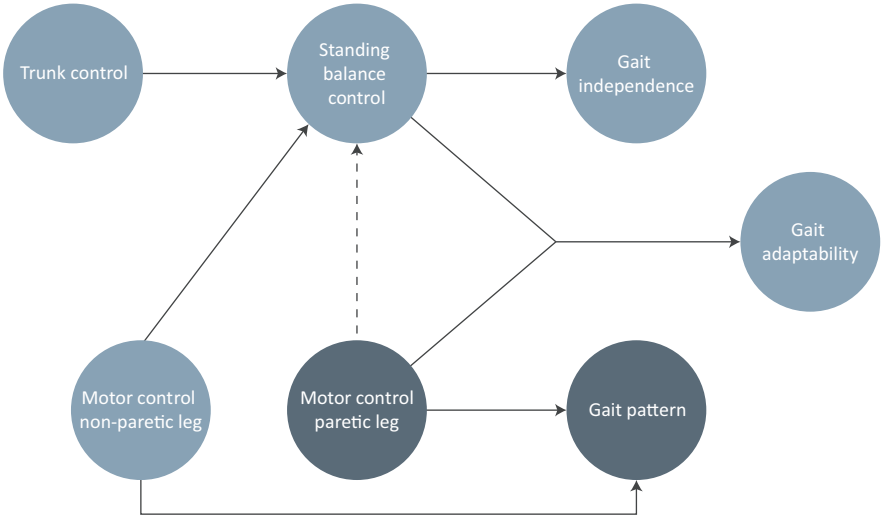


Figure 2. Theoretical framework on gait after stroke. Adapted from: Weerdesteyn et al. [15] and Van Duijnhoven [16].

effective muscle coordination ("leg muscle control"). Gait adaptability refers to the capacity to adapt gait in response to environmental challenges, such as navigating around obstacles or walking on uneven terrain [20]. According to the theoretical framework displayed in Figure 2, this involves a complex interaction between axial and leg muscle control.

In many individuals with stroke, impaired motor control of the paretic leg directly affects the gait pattern (Box 2), leading to an asymmetric, so-called "hemiparetic gait" [13, 21]. Depending on the severity of paresis, it also affects gait adaptability, as adjustments of gait require sufficient control of both the stepping and supporting leg. For the same reason, impaired motor control of the paretic leg also has an influence on dynamic (perturbed) standing balance control, but relatively little on so called "steady state" (unperturbed) balance control. That is why the vertical arrow in Figure 2 is interrupted.

Box 2. Gait pattern

The gait pattern can be described as the repetitive kinematic characteristics of the gait cycle, which starts at initial foot contact and ends with the subsequent foot contact of the same leg (Figure 3) [19]. The gait cycle can be divided into two phases: the stance phase and the swing phase.

In unaffected gait, the stance phase starts at initial foot contact, which is characterized by a heel strike with the ankle joint in a neutral position, the knee extended, and the hip flexed. Subsequently, an ankle plantar flexion movement can be observed, allowing the foot to achieve full contact with the ground. At this point, the contralateral leg starts the swing phase. In the ipsilateral leg, an ankle dorsiflexion movement occurs, accompanied by knee and hip extension, to facilitate forward progression of the body over the stance leg. As the heel lifts off the ground, the leg prepares for the swing phase by generating a rapid ankle plantar flexion movement, known as "push-off". Subsequently, the hip flexors initiate a "pull-off", causing the knee to flex and the foot to be lifted off the ground. During the swing phase, the leg moves forward through hip flexion, while simultaneous knee flexion and ankle dorsiflexion ensure adequate foot clearance from the ground. At the end of the swing phase, the ankle returns to a neutral position, the knee extends, and the hip remains flexed, prepositioning for the subsequent foot contact that marks the beginning of the next gait cycle.

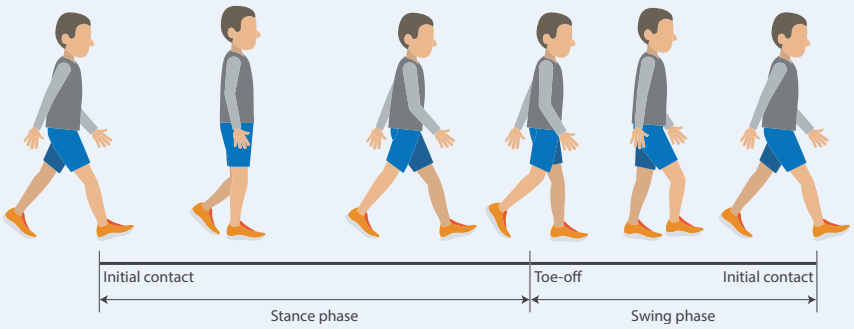


Figure 3. The gait cycle.

In hemiparetic gait, several deviations in the gait pattern can be observed compared to unaffected gait. The three most prominent deviations include circumduction during the swing phase, knee instability during the stance phase (due to either excessive knee flexion or knee hyperextension), and reduced knee flexion during the swing phase. The latter will hereafter be referred to as "stiff knee gait", which is defined as reduced knee flexion during the swing phase, regardless of its underlying motor deficit. The three most prominent gait deviations can be attributed to

three key motor deficits resulting from stroke (Figure 4). As illustrated in Figure 4, circumduction can result from pes equinovarus or reduced knee flexion during swing caused by triceps surae weakness or spasticity of the quadriceps muscles. Knee instability during stance may also arise from pes equinovarus or triceps surae weakness.

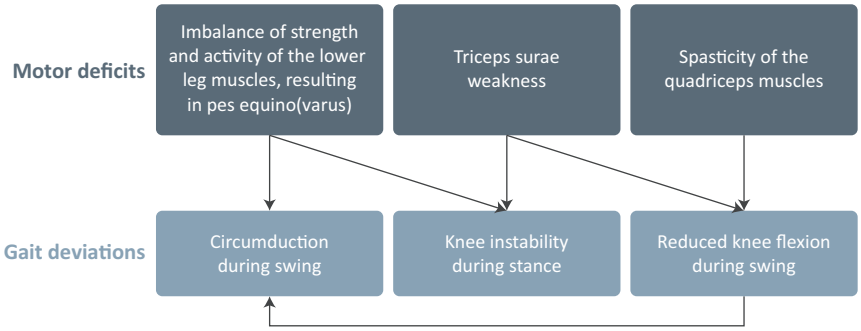


Figure 4. Key motor deficits and resulting gait deviations following stroke. Adapted from: Nonnekes et al. [4].

Pes equinovarus deformity is one of the key motor deficits following stroke and is caused by an imbalance of the lower leg muscles around the ankle and foot (Box 3) [4, 22, 23]. In turn, this muscular imbalance results from a combination of muscle weakness, spasticity and/or contractures. Pes equinus during the swing phase may hinder foot clearance, thereby increasing the risk of tripping and falling. As a result, a compensatory circumduction movement at the pelvis (lift) and hip (abduction) is often observed. If equinus deformity persists during the stance phase, it can compromise leg stability by inducing an external knee extension moment resulting in knee hyperextension. Additional pes varus during the stance phase can further compromise stability and increase the risk of ankle sprains. Furthermore, when equino(varus) deformity is present during the transition from the swing to the stance phase, abnormal initial foot contact may occur, potentially leading to instability during loading. Hence, pes equino(varus) can significantly impair gait, often leading to substantial limitations in daily functioning. The specific limitations vary from person to person and may include problems with walking barefoot, leading to dependence on assistive devices or orthopedic footwear, and to balance problems.

Box 3. Pes equinovarus

Pes equinovarus deformity is characterized by a plantar flexed position of the foot (pes equinus) in combination with a supinated position of the foot, which includes a varus tilt of the calcaneus (pes varus) (Figure 5).

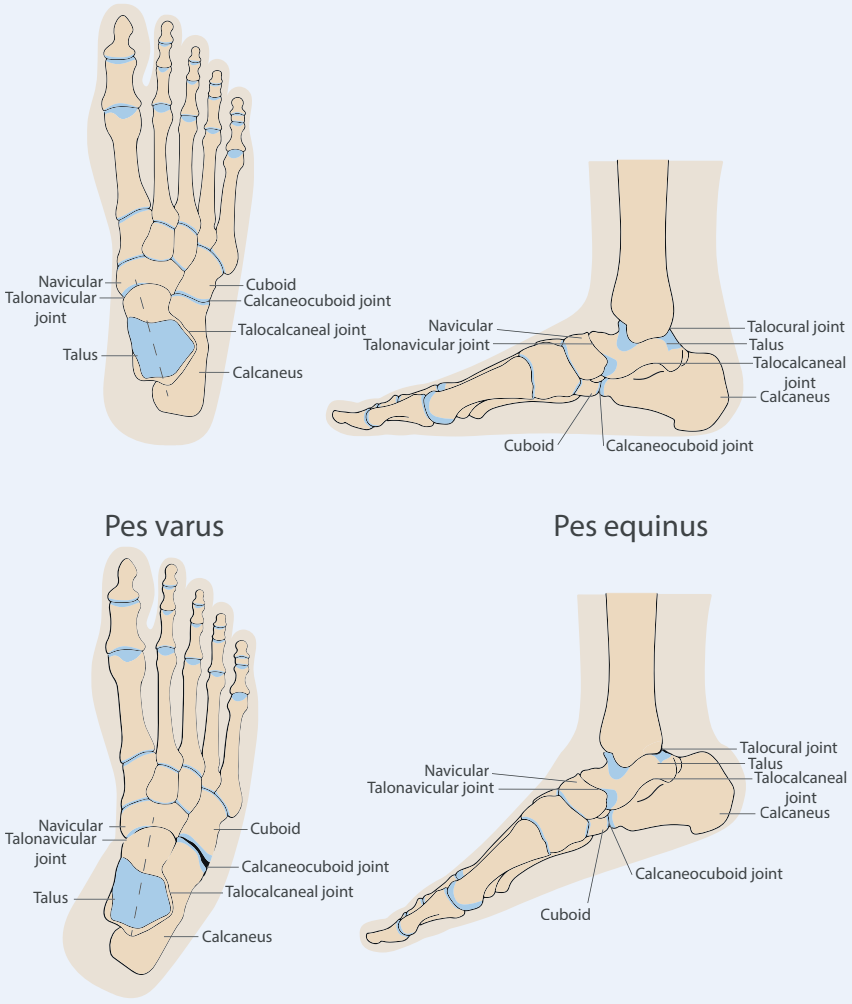


Figure 5. Pes equinovarus deformity. The top row shows the normal foot and ankle anatomy, while the bottom row illustrates pes varus and pes equinus deformity.

Spasticity of the triceps surae (gastrocnemius and soleus muscles) in combination with weakness of the ankle dorsiflexors (tibialis anterior, toe extensors and peroneus tertius muscles) commonly result in a dynamic pes equinus. In the case of prolonged spasticity of the triceps surae, muscle contracture may occur, leading to persistent (or structural) pes equinus. Likewise, spasticity of the tibialis posterior muscle, typically observed during the stance phase of gait, may result in a dynamic pes varus. In the case of contracture of the tibialis posterior muscle, a persistent (or structural) pes varus can be observed. Moreover, if the medial ankle dorsiflexors (tibialis anterior and extensor hallucis longus muscles) are stronger than the lateral ankle dorsiflexors (extensor digitorum longus and peroneus tertius muscles), a dynamic pes varus may occur during the swing phase of gait, as well as during the transition from the swing phase to the stance phase.

The primary joint responsible for plantar flexion and dorsiflexion of the foot, and consequently pes equinus, is the talocrural joint, often called the "ankle joint". Pes varus is determined by rotations and translations of the tarsal joints, which encompass all the joints between the talus, calcaneus, navicular, and cuboid bones. The most important joint here, determining the largest rotation of this complex, is the talonavicular joint [24]. Pes varus is almost fully dictated by the position of the talonavicular joint. After the talonavicular joint, most movement occurs at the talocalcaneal joint (synonymous with subtalar joint). To a lesser extent, the calcaneocuboid joint determines pes varus.

A second key motor deficit following stroke is triceps surae weakness. As the triceps surae muscles stabilize the knee joint during the midstance phase of gait, weakness can lead to excessive knee flexion, which compromises leg stability. In addition, weakness of the triceps surae can lead to stiff knee gait [19, 21]. During the push-off phase, the triceps surae muscles generate a strong ankle plantar flexion movement, propelling the leg into the swing phase. Together with a pull-off hip flexion movement, this propelling force initiates a rapid knee flexion movement, securing sufficient knee flexion during initial and midswing. Consequently, reduced push-off at the ankle joint and diminished pull-off at the hip joint may contribute to reduced knee flexion and diminished foot clearance during the swing phase of gait [25-27].

The third key motor deficit following stroke is spasticity of the quadriceps muscles, particularly of the rectus femoris muscle. This deficit may also contribute to the development of stiff knee gait by generating an increased intrinsic knee extension moment, which decelerates knee flexion during the transition from the stance to the swing phase and during the swing phase [4, 19, 28, 29]. Hence, stiff knee gait can arise from multiple underlying factors, including diminished ankle push-off and/or hip pull-off and spasticity of the quadriceps muscles. Regardless of the underlying cause, it may significantly hamper foot clearance, leading to an increased risk of falling and raised energy expenditure [30, 31]. As a result, compensatory circumduction may be required to secure foot clearance.

Assessment of post-stroke gait impairments

To evaluate post-stroke motor deficits and their resulting (compensatory) gait deviations, a comprehensive gait assessment is essential. Gait assessment should begin with thorough history taking, focusing not only on the gait impairments themselves but also on their impact on activities and participation in daily life. This should be followed by clinical examination to assess muscle deficits, such as weakness, spasticity, and contractures. Next, the gait pattern should be evaluated. If a gait laboratory is available, three-dimensional (3D) instrumented gait analysis can aid in examining gait abnormalities (Box 4). This approach allows for the evaluation and differentiation of motor deficits and their resulting (compensatory) gait deviations. For the examination of, for instance, equinovarus deformity and stiff knee gait, 3D instrumented gait analysis enhances diagnostic accuracy and supports treatment selection by offering a comprehensive evaluation of 3D body movements and forces, along with muscle activity patterns [32-34].

Box 4. Three-dimensional instrumented gait analysis

Three-dimensional (3D) instrumented gait analysis involves the use of advanced instrumentation to objectively capture and analyze human gait in three dimensions. This typically includes spatiotemporal gait parameters, kinematics, kinetics, and surface electromyography (sEMG). Measurements are conducted in a dedicated gait laboratory equipped with a wireless motion capture system, force plates, and a sEMG system (Figure 6).

The motion capture system uses reflective markers placed on the subject's skin at specific anatomical landmarks. Infrared cameras track the position of these markers in 3D space as the subject walks along a fixed track. From the 3D marker positions, spatiotemporal gait parameters can be calculated, including gait speed, step length, cadence, and step time.

Furthermore, the marker positions can be used to calculate kinematics, which describe the body's movements in space. This is accomplished using a biomechanical model that comprises multiple segments, most commonly the pelvis, upper leg, lower leg, and foot. For each segment, a coordinate system is constructed using the positions of the reflective markers. Subsequently, the rotation between the coordinate systems of two adjacent segments is computed to determine the corresponding joint angles. Typically, kinematic evaluation includes analyzing the 3D angular movements at the hip, knee, and ankle joints.

Additionally, kinetic evaluation can be performed. By combining kinematic data with force plate measurements, inverse dynamics can be applied, which yields the internal and external moments around the ankle, knee, and hip joints, as well as the concomitant joint powers, resulting from the forces produced by the muscles.

Lastly, sEMG is able to provide muscle activity patterns that can be related to the 3D kinematics and kinetics. This is done by placing electrodes on the subject's skin that measure the electrical activity of the underlying muscles. The resulting timing and relative intensity of muscle activity can be analyzed throughout the gait cycle.

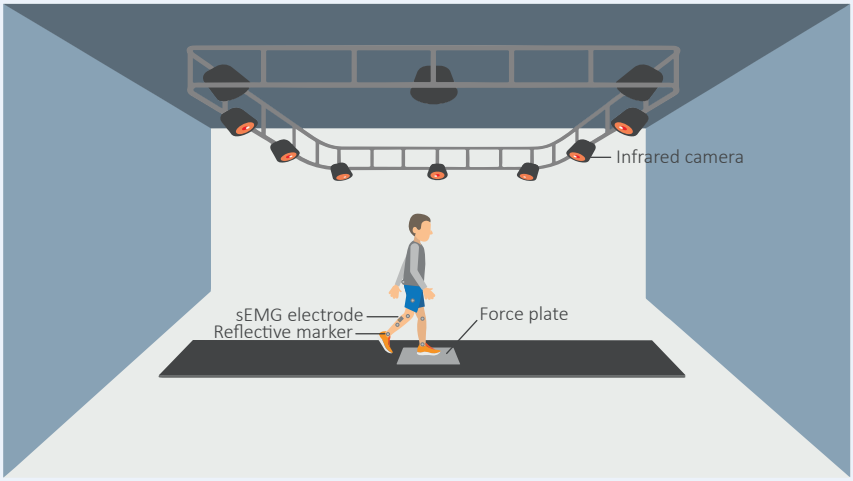


Figure 6. Three-dimensional instrumented gait analysis in a gait laboratory.

The assessment of pes equinovarus should, in addition to evaluating its underlying muscle deficits and its associated (compensatory) gait deviations, involve determination of the presence of pes equinus and/or pes varus across the phases of the gait cycle. In standard 3D instrumented gait analysis, the foot is typically modeled as a single rigid segment, which does not capture the joint movements within the foot [35]. In reality, the varus/valgus movement of the hindfoot results from the combined movements at multiple joints within the foot (Box 3). As such, conventional 3D instrumented gait analysis is insufficient for comprehensive evaluation of varus deformity. To overcome this limitation, additional methods can be employed to assess foot function during gait, including multi-segment foot models and plantar pressure measurements. Multi-segment foot models can be incorporated into 3D instrumented gait analysis. These models divide the foot into multiple segments, enabling the measurement of movements within the foot itself. Over the past decades, numerous multi-segment foot models have been developed, differing in the number of foot segments, marker locations, and the definitions of segment coordinate systems [35]. However, the discriminative capacity of these multi-segment foot models for detecting pes varus deformity in individuals with stroke remains largely unknown and underexplored. Another method for identifying pes equinovarus during gait is plantar pressure measurement, which enables the evaluation of load distribution across the plantar surface of the foot, thereby providing insight into foot function during the stance phase of gait [36]. The assessment of plantar pressures can be performed using a pressure plate or pressure insoles, either as part of 3D

instrumented gait analysis or as an independent evaluation. It can provide insight into, for instance, peak pressures, high-pressure areas, and the location of the center of pressure over the stance phase, all of which may offer valuable information about ankle-foot function during gait. Nonetheless, the application of plantar pressure measurement for assessing pes equinovarus in individuals with stroke has not yet been thoroughly investigated.

In the examination of stiff knee gait, 3D instrumented gait analysis could aid in discriminating the underlying causes, thereby distinguishing between spasticity of the quadriceps muscles and inadequate ankle push-off or hip pull-off. To assess muscle spasticity, the activity patterns of the quadriceps muscles should be evaluated during the critical phases of the gait cycle, specifically during the transition from stance to swing and during the swing phase, in relation to the deceleration of knee flexion. However, identifying spasticity is challenging, as the rectus femoris muscle is normally active around toe-off to control excessive knee flexion [19]. Furthermore, no guidelines exist for the assessment of ankle push-off and hip pull-off in relation to stiff knee gait. Hence, identifying the underlying cause of stiff knee gait remains challenging, while this is crucial for selecting the appropriate treatment. For instance, stiff knee gait caused by rectus femoris spasticity may effectively be treated with focal spasmolysis or distal rectus femoris transfer [37]. However, these interventions are not suitable if stiff knee gait is related to diminished ankle push-off and/or hip pull-off.

Treatment of pes equinovarus

Following comprehensive assessment of the gait abnormalities and their contributing motor deficits after stroke, treatment is aimed at addressing these deficits to reduce the activity limitations experienced in daily functioning. The primary goal of treatment is to achieve the personalized goals set by patients prior to treatment. These goals may include improving barefoot walking, reducing pain, or improving participation in daily activities. Furthermore, in line with the theoretical framework outlined in Figure 1, treatment focuses on improving the gait pattern and the related gait efficiency and safety, along with balance control and gait adaptability.

According to a treatment algorithm for pes equinovarus deformity previously published by our group, clinical management should follow a hierarchical approach [4]. First, any contractures should be addressed, followed by the treatment of disabling spasticity. Finally, after optimal treatment of contractures and spasticity, the management of pes equinovarus should focus on underlying muscle weakness. For the treatment of structural pes equinus caused by contracture of the triceps surae, stretching exercises are indicated first. However, the effects of muscle

stretching for equinus deformity in adults are often disappointing, which is why surgical intervention is often necessary to reduce the contracture [4]. Percutaneous Achilles tendon lengthening is generally recommended if both the gastrocnemius and soleus muscles are shortened. In cases where the contracture is limited to the gastrocnemius muscle, a gastrocnemius fasciotomy ("gastroc slide") as a selective form of Achilles tendon lengthening may be indicated. Structural pes varus resulting from muscle contracture can be surgically corrected through a tarsal arthrodesis, specifically a triple arthrodesis, which involves fusion of the talonavicular, talocalcaneal, and calcaneocuboid joints (Figure 7).

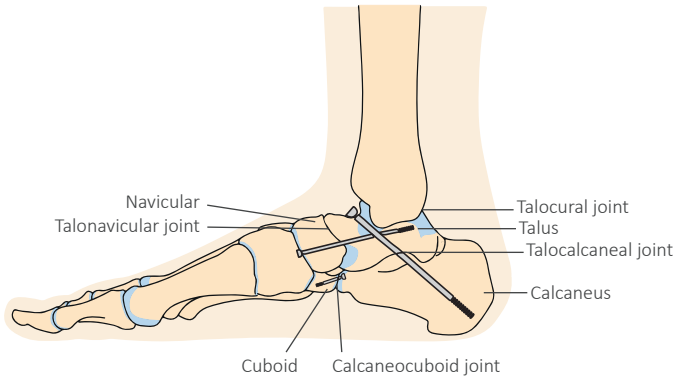


Figure 7. Triple arthrodesis: fusion of the talonavicular, talocalcaneal, and calcaneocuboid joints.

After addressing muscle contractures, or when contractures are absent, treatment of spasticity should be prioritized. Following Achilles tendon lengthening or gastrocnemius fasciotomy, triceps surae spasticity is often already reduced. However, if disabling spasticity persists, further interventions to reduce the effects of involuntary muscle tone are necessary. This can be achieved through focal spasmolysis or, for a more permanent result, selective neurotomy of the tibial nerve branches to the individual triceps surae muscles may be considered. In the case of dynamic pes varus due to tibialis posterior spasticity, surgical interventions including tarsal arthrodesis may be considered. As the talonavicular joint is the key joint in pes varus (Box 3), dynamic pes varus resulting from spasticity can most often effectively be corrected through a talonavicular arthrodesis. In the case of severe spasticity, additional fusion of the talocalcaneal joint may be considered, which could be induced by a diple arthrodesis (fusion of the talonavicular and talocalcaneal joints). Furthermore, talonavicular or diple arthrodesis could be combined with tenotomy or transfer of the tibialis posterior muscle in case of dynamic pes varus caused by tibialis posterior spasticity.

Subsequently, dynamic pes equinovarus resulting from muscle weakness should be addressed. Treatment options for dynamic pes equinus due to weakness of the ankle dorsiflexors include ankle-foot orthoses (AFO) and functional electrical stimulation (FES). A dynamic pes varus during swing and the transition from the swing phase to the stance phase can also be surgically corrected with a split anterior tibial tendon transfer (SPLATT), provided that tibialis anterior strength is sufficient, as the procedure entails some reduction in tibialis anterior strength. Alternatively, dynamic pes varus can be surgically corrected through a talonavicular arthrodesis.

Surgical interventions for correcting pes equinovarus may be more effective at improving personalized goals and gait capacity, and be more cost-efficient than nonoperative treatments [38]. Consequently, surgical interventions are recommended for managing pes equinovarus deformity [23, 32, 39]. However, to date, a limited amount of studies have been performed on the effect of reconstructive surgery for pes equinovarus, most of which are retrospective in design and include only one pre- and one post-surgical assessment without a control group, resulting in a low level of methodological quality [4, 40]. In addition, studies examining the effects of surgical correction of pes equinovarus on gait through 3D instrumented gait analysis are sparse [40, 41]. Lastly, the effects on personalized goals have not yet been investigated, even though their evaluation enables a comprehensive assessment of the specific problems an individual experiences, while accounting for differences in personalized goals between individuals [42, 43]. As personalized goals vary widely from person to person, other outcome measures are often insufficient to fully capture the individualized effects of surgery. Hence, scientific evidence supporting the effectiveness of surgical correction of pes equinovarus is still very limited and warrants further investigation, with the ultimate goal of improving management of pes equinovarus in people with stroke [4, 32, 40].

Aim and outline thesis

The aim of this thesis is to investigate the management of post-stroke gait impairments, with a specific focus on the assessment of pes equinovarus and stiff knee gait, as well as the treatment of pes equinovarus.

Part I of this thesis focuses on the assessment of gait impairments after stroke. The first two chapters specifically address the assessment of pes equinovarus deformity. In **chapter 2**, the discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus is studied in people with neurological gait impairments. In **chapter 3**, plantar pressure patterns are investigated in people with unilateral upper motor neuron syndrome, including stroke, aiming to gain insight into the plantar pressures in relation to hindfoot varus deformity. Secondly, this chapter aims to propose a quantitative, plantar pressure based, outcome measure for the evaluation of surgical interventions targeting pes varus. The last two chapters of part I focus on the assessment of stiff knee gait. In **chapter 4**, it is argued that there is a need for comprehensive guidelines for evaluating the contributors to stiff knee gait, including rectus femoris spasticity and diminished ankle push-off. To support this, a case series is presented, reporting 3D instrumented gait data of people with stroke. In **chapter 5**, a novel predictive model is introduced, designed to assess the impact of inadequate ankle push-off and hip pull-off on stiff knee gait. This model is developed using 3D instrumented gait data of people with stroke and healthy controls.

Part II of this thesis focuses on the evaluation of treatment of pes equinovarus deformity. More specifically, the effect of surgical fusion of one or more tarsal joints, often in combination with a form of Achilles tendon lengthening, is studied. In **chapter 6**, the effect of surgical correction of pes equinovarus on personalized goal attainment, balance capacity, and gait capacity is investigated in people with stroke. **Chapter 7** provides a comprehensive evaluation of gait, using 3D instrumented gait analysis and plantar pressure assessment, following surgical correction of pes equinovarus deformity in people with stroke. Finally, **chapter 8** provides a summary and discussion of the key findings of this thesis, highlighting their implications for clinical practice and future research.

References

[1] Feigin VL, Norrving B, Mensah GA. Global burden of stroke. Circ Res. 2017;120(3):439-48. https://doi.org/10.1161/CIRCRESAHA.116.308413.

[2] Grysiewicz RA, Thomas K, Pandey DK. Epidemiology of ischemic and hemorrhagic stroke: Incidence, prevalence, mortality, and risk factors. Neurol Clin. 2008;26(4):871-95. https://doi.org/10.1016/j.ncl.2008.07.003.

[3] Emos MC, Agarwal S. Neuroanatomy, upper motor neuron lesion. In: StatPearls. StatPearls Publishing; 2023.

[4] Nonnekes J, Benda N, van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[5] Mayer NH, Esquenazi A. Muscle overactivity and movement dysfunction in the upper motoneuron syndrome. Phys Med Rehabil Clin N Am. 2003;14(4):855-83. https://doi.org/10.1016/s1047-9651(03)00093-7.

[6] Miller A, Pohlig RT, Wright T, Kim HE, Reisman DS. Beyond physical capacity: Factors associated with real-world walking activity after stroke. Arch Phys Med Rehabil. 2021;102(10):1880-7.e1. https://doi.org/10.1016/j.apmr.2021.03.023.

[7] Warren M, Ganley KJ, Pohl PS. The association between social participation and lower extremity muscle strength, balance, and gait speed in US adults. Prev Med Rep. 2016;4:142-7. https://doi.org/10.1016/j.pmedr.2016.06.005.

[8] Ada L, Dean CM, Lindley R, Lloyd G. Improving community ambulation after stroke: The AMBULATE Trial. BMC Neurol. 2009;9:8. https://doi.org/10.1186/1471-2377-9-8.

[9] Algurén B, Fridlund B, Cieza A, Sunnerhagen KS, Christensson L. Factors associated with health-related quality of life after stroke: A 1-year prospective cohort study. Neurorehabil Neural Repair. 2011;26(3):266-74. https://doi.org/10.1177/1545968311414204.

[10] Natali AL, Reddy V, Bordoni B. Neuroanatomy, corticospinal cord tract. In: StatPearls. StatPearls Publishing; 2023.

[11] Jang SH, Chang CH, Lee J, Kim CS, Seo JP, Yeo SS. Functional role of the corticoreticular pathway in chronic stroke patients. Stroke. 2013;44(4):1099-104. https://doi.org/10.1161/STROKEAHA.111.000269.

[12] Li S, Chen Y-T, Francisco GE, Zhou P, Rymer WZ. A unifying pathophysiological account for post-stroke spasticity and disordered motor control. Front Neurol. 2019;10:468. https://doi.org/10.3389/fneur.2019.00468.

[13] Sheffler LR, Chae J. Hemiparetic gait. Phys Med Rehabil Clin N Am. 2015;26(4):611-23. https://doi.org/10.1016/j.pmr.2015.06.006.

[14] Li S, Francisco GE. New insights into the pathophysiology of post-stroke spasticity. Front Hum Neurosci. 2015;9:192. https://doi.org/10.3389/fnhum.2015.00192.

[15] Weerdesteyn VGM, Nonnekes JH, Fleuren JFM, Buurke JH, van Duijnhoven HJR, Geurts ACH. Herstel van loopvaardigheid na een CVA - een theoretisch framework. Nederlands Tijdschrift voor Revalidatiegeneeskunde. 2022;44(1):8-11.

[16] van Duijnhoven H. The challenges of dynamic balance and gait for people after stroke [PhD thesis]. Radboud University; 2020.

[17] Veerbeek JM, van Wegen EEH, Harmeling–van der Wel BC, Kwakkel G. Is accurate prediction of gait in nonambulatory stroke patients possible within 72 hours poststroke?: The EPOS study. Neurorehabil Neural Repair. 2011;25(3):268-74. https://doi.org/10.1177/1545968310384271.

[18] Kollen B, Kwakkel G, Lindeman E. Longitudinal robustness of variables predicting independent gait following severe middle cerebral artery stroke: A prospective cohort study. Clin Rehabil. 2006;20(3):262-8. https://doi.org/10.1191/0269215506cr910oa.

[19] Perry J. Gait analysis: Normal and pathological function. SLACK Incorporated; 1992.

[20] Weerdesteyn V, Hollands KL, Hollands MA. Gait adaptability. Handb Clin Neurol. 2018;159:135-46. https://doi.org/10.1016/B978-0-444-63916-5.00008-2.

[21] Balaban B, Tok F. Gait disturbances in patients with stroke. PM&R. 2014;6(7):635-42. https://doi.org/10.1016/j.pmrj.2013.12.017.

[22] Moon D, Esquenazi A. Instrumented gait analysis: A tool in the treatment of spastic gait dysfunction. JBJS Rev. 2016;4(6):e1. https://doi.org/10.2106/JBJS.RVW.15.00076.
[23] Keenan MA. The management of spastic equinovarus deformity following stroke and head injury. Foot Ankle Clin. 2011;16(3):499-514. https://doi.org/10.1016/j.fcl.2011.07.002.
[24] Lundberg A. Kinematics of the ankle and foot: In vivo roentgen stereophotogrammetry. Acta Orthop Scand. 1989;60:Suppl.233. https://doi.org/10.3109/17453678909154185.
[25] Goldberg SR, Anderson FC, Pandy MG, Delp SL. Muscles that influence knee flexion velocity in double support: Implications for stiff-knee gait. J Biomech. 2004;37(8):1189-96. https://doi.org/10.1016/j.jbiomech.2003.12.005.
[26] Fox MD, Delp SL. Contributions of muscles and passive dynamics to swing initiation over a range of walking speeds. J Biomech. 2010;43(8):1450-5. https://doi.org/10.1016/j.jbiomech.2010.02.009.
[27] Apti A, Akalan NE, Kuchimov S, Özdinçler AR, Temelli Y, Nene A. Plantar flexor muscle weakness may cause stiff-knee gait. Gait Posture. 2016;46:201-7. https://doi.org/10.1016/j.gaitpost.2016.03.010.
[28] Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. J Biomech. 1996;29(6):723-33. https://doi.org/10.1016/0021-9290(95)00144-1.
[29] Riley PO, Kerrigan DC. Torque action of two-joint muscles in the swing period of stiff-legged gait: A forward dynamic model analysis. J Biomech. 1998;31(9):835-40. https://doi.org/10.1016/s0021-9290(98)00107-9.
[30] Waters RL, Mulroy S. The energy expenditure of normal and pathologic gait. Gait Posture. 1999;9(3):207-31. https://doi.org/10.1016/s0966-6362(99)00009-0.
[31] Lewek MD, Osborn AJ, Wutzke CJ. The influence of mechanically and physiologically imposed stiff-knee gait patterns on the energy cost of walking. Arch Phys Med Rehabil. 2012;93(1):123-8. https://doi.org/10.1016/j.apmr.2011.08.019.
[32] Patrick JH, Keenan MAE. Gait analysis to assist walking after stroke. Lancet. 2007;369(9558):256-7. https://doi.org/10.1016/S0140-6736(07)60124-X.
[33] Fuller DA, Keenan MAE, Esquenazi A, Whyte J, Mayer NH, Fidler-Sheppard R. The impact of instrumented gait analysis on surgical planning: Treatment of spastic equinovarus deformity of the foot and ankle. Foot Ankle Int. 2002;23(8):738-43. https://doi.org/10.1177/107110070202300810.
[34] Merlo A, Campanini I. Impact of instrumental analysis of stiff knee gait on treatment appropriateness and associated costs in stroke patients. Gait Posture. 2019;72:195-201. https://doi.org/10.1016/j.gaitpost.2019.06.009.
[35] Leardini A, Caravaggi P, Theologis T, Stebbins J. Multi-segment foot models and their use in clinical populations. Gait Posture. 2019;69:50-9. https://doi.org/10.1016/j.gaitpost.2019.01.022.
[36] Orlin MN, McPoil TG. Plantar pressure assessment. Phys Ther. 2000;80(4):399-409. https://doi.org/10.1093/ptj/80.4.399.
[37] Tenniglo MJB. Stiff knee gait in stroke: Walking down the road of different treatment options [PhD thesis]. University of Twente; 2023.
[38] Reddy S, Kusuma S, Hosalkar H, Keenan MA. Surgery can reduce the nonoperative care associated with an equinovarus foot deformity. Clin Orthop Relat Res. 2008;466(7):1683-7. https://doi.org/10.1007/s11999-008-0250-3.
[39] Waters RL, Perry J, Garland D. Surgical correction of gait abnormalities following stroke. Clin Orthop Relat Res. 1978;(131):54-63. https://doi.org/10.1097/00003086-197803000-00008.
[40] Renzenbrink GJ, Buurke JH, Nene AV, Geurts ACH, Kwakkel G, Rietman JS. Improving walking capacity by surgical correction of equinovarus foot deformity in adult patients with stroke or traumatic brain injury: A systematic review. J Rehabil Med. 2012;44(8):614-23. https://doi.org/10.2340/16501977-1012.
[41] de l'Escalopier N, Voisard C, Michaud M, Moreau A, Jung S, Tervil B, et al. Evaluation methods to assess the efficacy of equinovarus foot surgery on the gait of post-stroke hemiplegic patients: A literature review. Front Neurol. 2022;13:1042667. https://doi.org/10.3389/fneur.2022.1042667.
[42] Wade DT. Goal setting in rehabilitation: An overview of what, why and how. Clin Rehabil. 2009;23(4):291-5. https://doi.org/10.1177/0269215509103551.
[43] Grenville J, Lyne P. Patient-centred evaluation and rehabilitative care. J Adv Nurs. 1995;22(5):965-72. https://doi.org/10.1111/j.1365-2648.1995.tb02649.x.



Part I

Assessment of post-stroke gait impairments



Chapter 2

Discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus in people with neurological gait impairments

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Abstract

Background

Multi-segment foot models extend standard gait analysis, typically limited to hip, knee, and ankle kinematics, by capturing hindfoot kinematics. However, their ability to discriminate between hindfoot varus and valgus remains unclear. Therefore, this study aimed to explore this discriminative capacity in people with neurological gait impairments.

Methods

In this cross-sectional study, 50 adults with various neurological gait impairments were enrolled and underwent three-dimensional instrumented gait analysis using four multi-segment foot models and a mono-segment foot model. Clinical examination was performed based on video recordings, with the hindfoot position being classified as either varus, valgus, or neutral. Mean hindfoot angles measured by the foot models, along with their 95% prediction intervals, were calculated for each clinically examined category of hindfoot position. The overlap in prediction intervals was analyzed to determine the discriminative capacity.

Results

Comparisons between foot models revealed structural and waveform differences in hindfoot kinematics. Across models, 38% to 83% of hindfoot angles fell within the overlapping region of the 95% prediction intervals for the clinically examined categories hindfoot varus and valgus, indicating substantial overlap.

Conclusion

Mono-segment and multi-segment foot models demonstrated limited ability to discriminate between hindfoot varus and valgus, indicating that absolute hindfoot angles should be interpreted with caution. Multi-segment foot models, however, seem better suited for assessing changes in hindfoot angle during the gait cycle. Hence, the clinical validity of multi-segment foot models is largely determined by the specific clinical objectives they aim to fulfill.

Introduction

In people with neurological disorders, impaired muscle control around the ankle and foot often leads to the development of ankle-foot deformities [1-3]. As a result of these deformities, gait impairments are common [1, 4]. Three-dimensional (3D) instrumented gait analysis is widely used to assess gait impairments, providing valuable insights into hip, knee, and ankle joint kinematics and kinetics [5]. However, most biomechanical models used for gait analysis represent the foot as a single rigid segment, limiting the ability to assess motions within the foot [6] and, thereby, hindering accurate evaluation of ankle-foot deformities. To address this limitation, multi-segment foot models have been developed to capture foot structure and function [6, 7]. Given that many ankle-foot deformities involve an altered hindfoot position relative to the shank in the frontal plane, as in pes equinovarus, pes cavovarus, and flatfoot deformities [1, 8, 9], multi-segment foot models must accurately capture the hindfoot varus/valgus angle to provide clinically relevant information that can support effective management of ankle-foot deformities.

To date, few studies have evaluated the validity of multi-segment foot models for measuring hindfoot kinematics in the frontal plane [6]. Nester and colleagues reported a mean difference over the stance phase of 2.3° compared to bone-mounted markers [10], while another study using biplanar fluoroscopy found a root mean squared error of 3.6° [11]. However, the discriminative capacity remains unexplored, despite the clinical need to accurately distinguish between hindfoot varus and valgus in each individual. Furthermore, the aforementioned studies were conducted in healthy participants, whereas assessment in populations with ankle-foot deformities is lacking. Additionally, previous studies focused on mean differences over the gait cycle, while investigating the hindfoot varus/valgus angle at specific gait phases during which hindfoot varus commonly manifests, i.e. initial contact and midstance [1], would be of clinical relevance.

The aim of the present study was to evaluate the discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus in people with neurological gait impairments. Four multi-segment foot models were defined using markers on palpable anatomical landmarks of the calcaneus. Hindfoot angles were calculated according to these four multi-segment foot models and a mono-segment foot model to determine which model best corresponds to clinical examination of the hindfoot position, focusing on their ability to discriminate between varus and valgus at initial contact and midstance.

Methods

Participants and design

In this cross-sectional study, we enrolled 50 adults with various neurological gait impairments who were evaluated during routine clinical care at the mobility outpatient clinic of the Gait Expertise Center of the Sint Maartenskliniek and Radboud university medical center between May 2023 and April 2024. The cohort included a variety of neurological gait impairments and corresponding ankle-foot deformities, reflecting the heterogeneity encountered in clinical practice and allowing evaluation of the potential value of multi-segment foot models for use in a clinical setting. Exclusion criteria were: 1) inability to walk barefoot and 2) inability to walk without walking aid. The mean age of the participants was 48±16 years, and 23 were female. Participants' diagnoses included: chronic stroke (n=17), hereditary motor and sensory neuropathy (n=14), incomplete spinal cord injury (n=5), traumatic brain injury (n=4), multiple sclerosis (n=2), congenital dorsiflexor paresis (n=2), pure hereditary spastic paraplegia (n=1), cerebral abscess (n=1), Marfan syndrome (n=1), hereditary neuropathy with liability to pressure palsy (n=1), compartment syndrome following a lower leg fracture (n=1), and autosomal recessive spastic ataxia of Charlevoix-Saguenay (n=1). The study adhered to the local ethical regulations. The Medical Ethical Committee determined that the Medical Research Involving Human Subjects Act (WMO) was not applicable, thereby waiving the requirement for formal ethical approval. Prior to the measurements, each participant provided written informed consent.

Examinations

A 3D instrumented gait analysis was conducted in the gait laboratory of the Sint Maartenskliniek, which is equipped with a Vicon motion capture system (Vicon Motion Systems Ltd., Oxford, UK) comprising 12 infrared cameras and four video cameras. As part of routine clinical care, reflective markers with a diameter of 14 mm were placed according to the Plug-in Gait lower body model. Furthermore, additional 9 mm markers were placed on the hindfoot and shank. An overview of all hindfoot and shank markers is provided in Table 1. Marker placement on the hindfoot and shank followed the Rizzoli Foot Model (RFM) [12], which uses markers on palpable anatomical landmarks. This approach minimizes the risk of marker placement error compared to alternative multi-segment foot models relying on less anatomically distinct locations, such as the medial and lateral aspects of the calcaneus [13-17]. Additionally, close-up video recordings of the hindfoot in the frontal plane were captured during gait to enable clinical examination of the hindfoot position. Marker data and video recordings were captured at a sample frequency of 100 Hz, with video recordings captured at a resolution of 1920 x 1080 pixels.

Table 1. Foot and shank markers.

Segment	Marker	Location
Hindfoot	Sustentaculum tali (ST)	The most medial aspect of the sustentaculum tali
	Peroneal tubercle (PT)	The most lateral aspect of the peroneal tubercle
	Posterior calcaneus proximally (PCA)#	Proximally on the midline of the posterior aspect of the calcaneus
	Posterior calcaneus distally (HEE)*	Distally on the midline of the posterior aspect of the calcaneus
Shank	Tibial tuberosity (TT)	The most anterior aspect of the tibial tuberosity
	Head of the fibula (HF)	The most proximal aspect of the head of the fibula
	Lateral malleolus (LANK)*	The most lateral aspect of the lateral malleolus
	Medial malleolus (MANK)*#	The most medial aspect of the medial malleolus
	Tibia (TIB)*	Half of the distance between the lateral epicondyle and the lateral malleolus

**=part of the Plug-in Gait lower body model. #=only placed during static calibration trial.*

The measurements commenced with a static standing trial, followed by a gait analysis while participants walked at their preferred speed. At least six walking trials were recorded for each participant.

Data analyses

To evaluate the discriminative capacity of multi-segment foot models, their outcomes were compared with clinical examination of the hindfoot position in the frontal plane. Given the limited association between static foot posture and foot kinematics during gait [18], clinical examination did not rely on static foot posture, but instead on dynamic evaluation using close-up video recordings of the posterior hindfoot during gait. Two experienced independent raters classified the hindfoot position relative to the shank as "varus", "valgus", or "neutral" at two instances: initial contact (i.e. foot strike following the swing phase) and midstance (i.e. when the swing leg crosses the stance leg). These instances were selected because hindfoot varus commonly manifests during the swing phase and loading response of gait (captured at initial contact), and/or during midstance [1]. Classification was performed using multiple gait cycles across several trials, including only the gait cycles in which the camera's optical axis was well aligned perpendicular to the hindfoot and the hindfoot was adequately visible within the camera's field of view. Any discrepancies between raters were resolved through discussion.

Gait data were processed using Vicon Nexus (Vicon Motion Systems Ltd., Oxford, UK) and MATLAB (Version R2021a, The MathWorks, Inc., Natick, MA, USA). Kinematics were calculated according to the mono-segment Plug-in Gait lower body model and four multi-segment foot models. For the Plug-in Gait lower body model, Vicon Nexus (Vicon Motion Systems Ltd., Oxford, UK) was used to determine the foot angle relative to the shank in the frontal plane. For calculating hindfoot kinematics according to multi-segment foot models, anatomical reference frames were defined, with the hindfoot angle determined by the orientation of the hindfoot anatomical reference frame relative to that of the shank. The shank's anatomical reference frame was based on the Rizzoli Foot Model [12] (see Table 2 and Figure 1), as it provides a representation of the underlying bone anatomy without requiring markers on the femoral epicondyle, unlike several alternative models [15, 16, 19]. Additionally, a technical reference frame was defined to track the shank during gait using the TIB, LANK, and TT markers (see Table 1 for marker abbreviations), since the MANK marker was removed during the walking trials and the HF marker is prone to large soft tissue artifacts [20]. For the static standing trial, the anatomical reference frame was expressed relative to the technical reference frame, enabling the reconstruction of the anatomical reference frame from the technical reference frame during gait. For the hindfoot segment, the four hindfoot markers enabled the definition of four distinct anatomical reference frames that reflect hindfoot anatomy, as detailed in Table 2 and Figure 1. These hindfoot anatomical reference frames align with those described in previously published multi-segment foot models [12, 21, 22]. Hindfoot anatomical reference frame 4 represents a modification of hindfoot anatomical reference frame 3 and requires one fewer marker when applied in combination with the Plug-in Gait lower body model, thereby increasing clinical feasibility. The combinations of hindfoot and shank anatomical reference frames are hereafter referred to as multi-segment foot model 1 to 4. The technical reference frame for the hindfoot was defined using the HEE, ST, and PT markers (see Table 1 for marker abbreviations), as the PCA marker was removed during the walking trials.

Hindfoot angles were computed following the International Society of Biomechanics (ISB) recommendations [23] and the methodology outlined by Grood and Suntay [24], consistent with previously described foot models [12, 22]. For multi-segment foot model 1, the hindfoot line segment was projected onto the shank's frontal plane, and the hindfoot angle relative to the shank was computed as the angle between this projection and the y-axis of the shank's anatomical reference frame. For multi-segment foot models 2-4, the 3D orientation of the hindfoot segment was calculated relative to the 3D orientation of the shank segment to derive hindfoot angles around the anterior-posterior axis [12, 22].

Table 2. Definitions of the anatomical reference frames for the hindfoot and shank of the right limb.

Segment	Anatomical reference frame		Description	Formula
Hindfoot	**1***	Vertical axis (y)	Line segment between HEE and PCA	y = PCA - HEE
	2**	Origin	Midpoint between HEE and PCA	o = (HEE+PCA)/2
		Anterior-posterior axis (x)	From origin to midpoint between ST and PT	x = (ST+PT)/2-o
		Vertical axis (y)	Perpendicular to the plane defined by z and x	y = cross(z,x)
		Medio-lateral axis (z)	Perpendicular to the plane defined by x and the vector from HEE to PCA	z = cross(x,PCA-HEE)
	3***	Origin	PCA	o = PCA
		Anterior-posterior axis (x)	From origin to midpoint between ST and PT	x = (ST+PT)/2-o
		Vertical axis (y)	Perpendicular to the plane defined by x and the vector from origin to ST	y = cross(x,ST-o)
		Medio-lateral axis (z)	In the plane defined by o, ST, and PT	z = cross(x,y)
	4	Origin	HEE	o = HEE
		Anterior-posterior axis (x)	From origin to midpoint between ST and PT	x = (ST+PT)/2-o
		Vertical axis (y)	Perpendicular to the plane defined by x and the vector from origin to ST	y = cross(x,ST-o)
		Medio-lateral axis (z)	In the plane defined by o, ST, and PT	z = cross(x,y)
Shank	**1******	Origin	Midpoint between LANK and MANK	o = (LANK + MANK)/2
		Anterior-posterior axis (x)	Perpendicular to the plane defined by y and the vector from origin to LANK	x = cross(y, LANK-o)

Table 2. Continued.

Segment	Anatomical reference frame		Description	Formula
Shank	**1******	Vertical axis (y)	From origin to the projection of TT on the plane defined by the origin, LANK and HF	y = TT_proj−o
		Medio-lateral axis (z)	Perpendicular to the plane defined by x and y	z = cross(x,y)

**According to Portinaro et al. [21], **According to Schallig et al. [22], ***According to Leardini et al. [12], ****According to Leardini et al. [12], and Schallig et al. [22]. Marker abbreviations are detailed in Table 1 and a graphical representation is shown in Figure 1.*

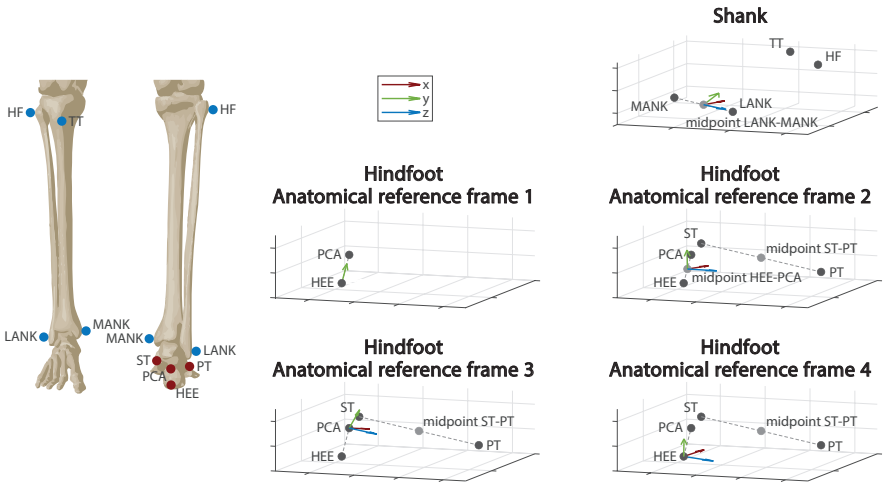


Figure 1. Graphical representation of marker positions and anatomical reference frames for the right foot. Definitions of the anatomical reference frames are provided in Table 2, while marker positions are detailed in Table 1. In the left panel, hindfoot markers are depicted in red, whereas shank markers are depicted in blue. LANK = lateral malleolus, MANK = medial malleolus, HF = head of the fibula, TT = tibial tuberosity, ST = sustentaculum tali, PT = peroneal tubercule, PCA = posterior calcaneus proximally, HEE = posterior calcaneus distally.

For each of six walking trials, gait cycles of the left and right foot were extracted by identifying initial contact events according to the methodology of Zeni [25]. The first and last initial contacts within the measurement volume were excluded. Remaining gait cycles were time-normalized to 0-100% and foot kinematics were averaged across all gait cycles per foot within a trial. Subsequently, averages across all six

walking trials were calculated per foot. Mean hindfoot angles over the gait cycle were calculated. Furthermore, hindfoot angles at initial contact and midstance (defined as the instance the swing leg's lateral malleolus passes that of the stance leg) were extracted and averaged across gait cycles and trials per foot.

Statistical analyses

Kinematics were compared between foot models to explore inter-model differences. To examine structural differences in hindfoot varus/valgus angle between models (i.e. differences present throughout the gait cycle), mean angles over the gait cycle were compared using a repeated measures Analysis of Variance (ANOVA) followed by Bonferroni-corrected post hoc paired samples t-tests. Furthermore, statistical parametric mapping (SPM) was performed using a repeated measures ANOVA and post hoc paired samples t-tests with Bonferroni correction to examine differences in hindfoot varus/valgus angle throughout the gait cycle [26]. To assess differences in waveforms independent of any structural differences in hindfoot angle, the same SPM tests were repeated after subtracting the mean hindfoot angle over the gait cycle from the angle at each time point. P-values less than 0.05 were considered as statistically significant.

For clinically applicability, a multi-segment foot model must accurately discriminate between hindfoot varus and valgus in each individual. To evaluate this, mean hindfoot angles and corresponding 95% prediction intervals were computed for each clinically defined category of hindfoot position. Prediction intervals were calculated as mean ± 1.96*standard deviation and computed at initial contact and midstance for each foot model. Discriminative capacity was quantified by the percentage of feet classified as varus or valgus falling within the overlapping area of the prediction intervals, with 0% overlap indicating perfect discrimination.

Results

Data were collected from 100 feet belonging to 50 participants. Three feet were excluded due to data quality issues: two because of a missing marker during the static trial and one due to excessive gaps in the marker data during the walking trials. As a result, 97 feet from 50 participants were included in the analysis.

Figure 2 presents the hindfoot angle for the mono-segment Plug-in Gait lower body model and the four multi-segment foot models. The mean hindfoot angle over the gait cycle was -0.3±2.2° of valgus for the Plug-in Gait lower body model, -2.1±8.9° of valgus for multi-segment foot model 1, -0.6±8.9° of valgus for multi-segment foot model 2, 8.6±5.8° of varus for multi-segment foot model 3, and 9.3±7.1° of varus for

multi-segment foot model 4. A statistically significant difference in mean hindfoot angle was found between models $F(4,384)=99.1$, $p<0.001$). Post hoc tests revealed that the Plug-in Gait lower body model and multi-segment foot models 1 and 2 differed significantly from multi-segment foot models 3 and 4 ($p<0.001$). Furthermore, the mean hindfoot angle of multi-segment foot model 1 differed significantly from the mean hindfoot angle of multi-segment foot model 2 ($p<0.001$).

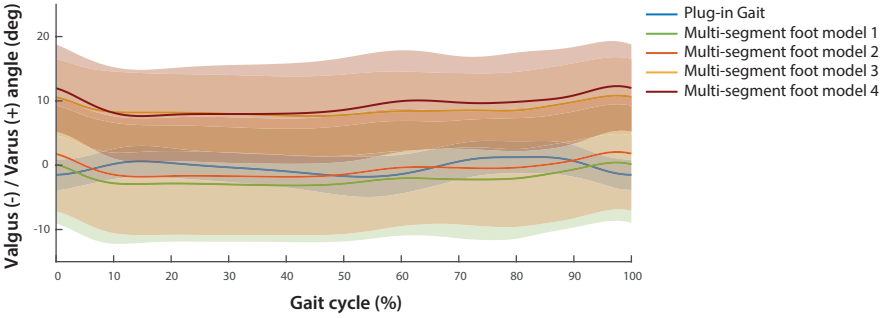


Figure 2. Hindfoot angle relative to the shank in the frontal plane throughout the gait cycle. The angle is shown for a mono-segment foot model (Plug-in Gait lower body model: foot angle relative to the shank) and four multi-segment foot models. The definitions of the anatomical reference frames for the multi-segment foot models are provided in Table 2 and Figure 1.

SPM revealed significant differences in hindfoot angle throughout the entire gait cycle ($F(4,384)=3.2$, $p<0.001$), with the Plug-in Gait lower body model and multi-segment foot models 1 and 2 differing from multi-segment foot models 3 and 4 ($p<0.001$). Furthermore, the Plug-in Gait lower body model differed from multi-segment foot model 1 between 13-17% of the gait cycle and from multi-segment foot model 2 between 97-100% of the gait cycle ($p<0.01$). In addition, multi-segment foot model 1 differed from multi-segment foot model 2 across the entire gait cycle ($p<0.001$). After correcting for structural differences, SPM showed deviations between foot models during 0-6%, 8-40%, 50-86%, and 90-100% of the gait cycle ($F(4,384)= 4.1$, $p<0.03$). The results of the post hoc tests for the hindfoot angle corrected for structural differences are presented in Figure 3. The Plug-in Gait lower body model showed differences from all multi-segment foot models across various phases of the gait cycle.

Discriminative capacity

Based on clinical examination, 22 of the 97 feet were classified as hindfoot varus at initial contact, 63 as neutral, and 12 as valgus. At midstance, 16 feet were classified as varus, 45 as neutral, and 36 as valgus.

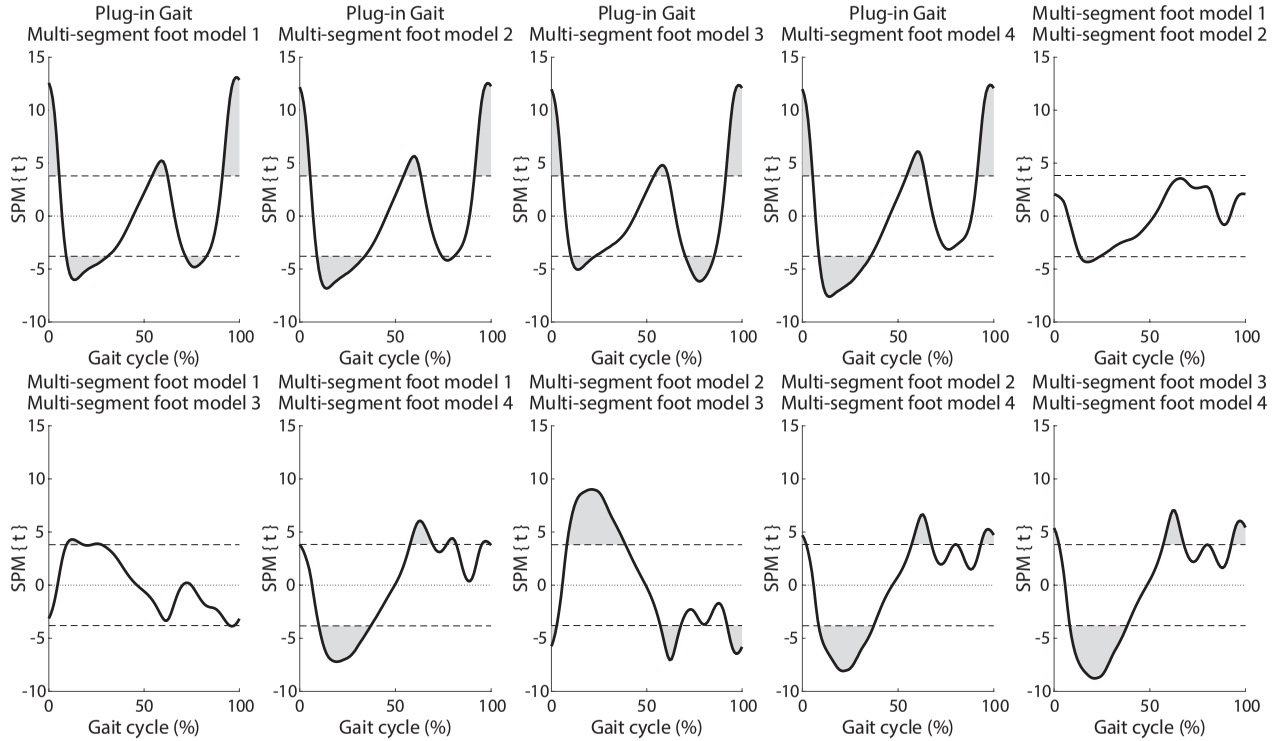


Figure 3. Statistical parametric mapping results for the hindfoot angle relative to the shank in the frontal plane, corrected for structural differences, throughout the gait cycle. The definitions of the multi-segment foot models are provided in Table 2 and Figure 1.

Table 3 and Figure 4 present the mean values, standard deviations, and 95% prediction intervals of the hindfoot angle at initial contact and midstance. At initial contact, the overlap in 95% prediction intervals of the varus and valgus groups was 71% for the Plug-in Gait lower body model, 62% for multi-segment foot model 1, 65% for multi-segment foot model 2, and 56% for multi-segment foot model 3. Multi-segment foot model 4 showed the lowest overlap, with 38% of the feet classified as varus or valgus falling within the overlapping area of their prediction intervals.

At midstance, the Plug-in Gait lower body model showed an 83% overlap in 95% prediction intervals for feet classified as varus or valgus. The corresponding overlap percentages were 62%, 73%, 81%, and 69% for multi-segment foot model 1, 2, 3, and 4 respectively.

Table 3. Mean hindfoot angles and standard deviations at initial contact and midstance for feet classified as hindfoot varus, valgus, and neutral. The reference frames of the multi-segment foot models are defined in Table 2 and Figure 1.

	Plug-in Gait	Multi-segment foot model 1	Multi-segment foot model 2	Multi-segment foot model 3	Multi-segment foot model 4
Initial contact					
Varus	-2.6±2.6°	8.5±6.8°	8.9±7.0°	13.9±6.2°	16.5±6.2°
Neutral	-1.4±2.2°	-1.9±8.4°	-0.1±8.5°	10.2±5.6°	11.6±6.4°
Valgus	0.3±1.8°	-4.4±8.0°	-1.4±7.8°	6.3±3.7°	5.8±4.0°
Midstance					
Varus	-2.5±2.8°	4.8±6.5°	5.7±7.5°	12.1±7.0°	14.8±7.8°
Neutral	-0.6±1.8°	-3.0±7.1°	-1.6±7.2°	8.6±5.5°	8.7±6.2°
Valgus	0.5±2.1°	-6.8±9.5°	-5.3±9.9°	5.0±4.8°	3.9±6.8°

Varus angles are indicated by positive values, whereas valgus angles are indicated by negative values.

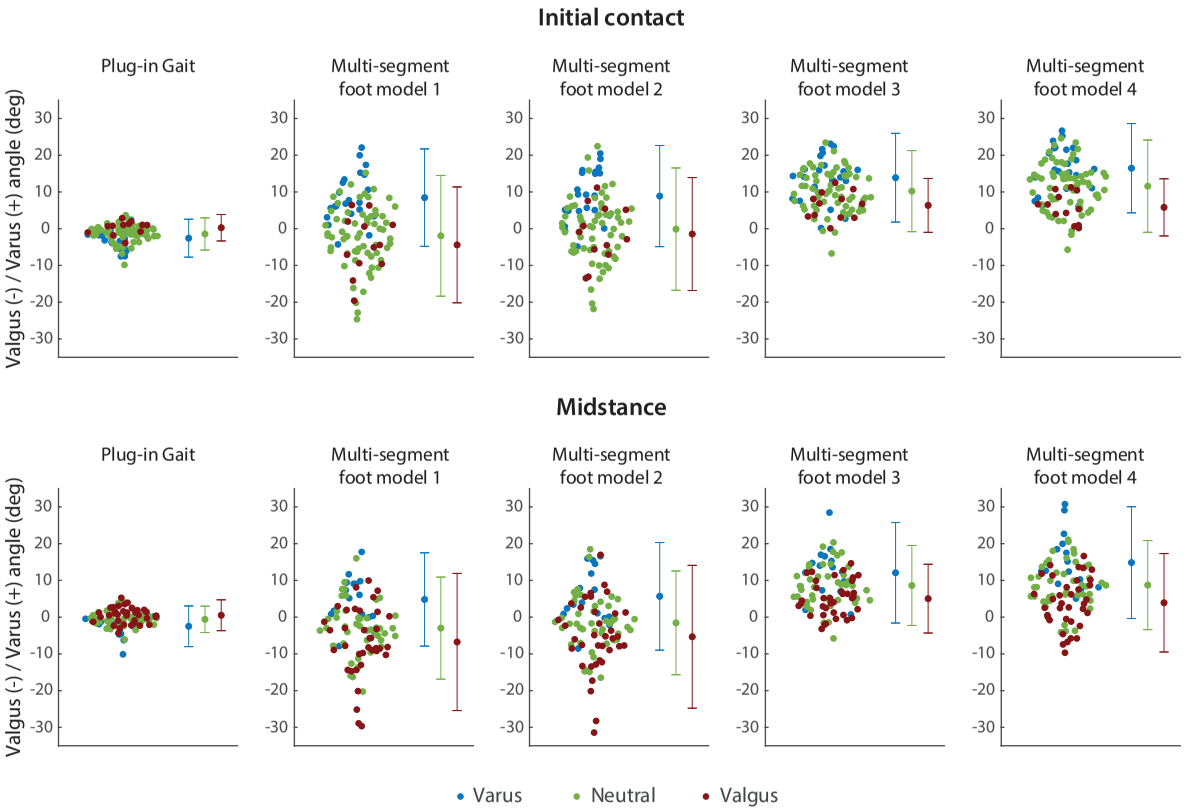


Figure 4. Hindfoot angle relative to the shank in the frontal plane at initial contact and midstance for three groups classified by clinical examination. For each group for each foot model, the mean value and 95% prediction interval is displayed. The multi-segment foot models are defined in Table 2 and Figure 1.

Discussion

This study aimed to explore the discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus in people with neurological gait impairments. Hindfoot angles were calculated according to a mono-segment foot model and four multi-segment foot models, with notable inter-model differences observed in mean hindfoot angles and waveform patterns throughout the gait cycle. The models were evaluated based on their correspondence with clinical hindfoot assessment, focusing on their ability to discriminate between varus and valgus. All foot models demonstrated limited discriminative capacity, with 38%-83% of feet classified as varus or valgus falling within the overlapping region of their 95% prediction intervals across models.

Comparisons between foot models revealed considerable differences in mean hindfoot angle, particularly for multi-segment foot models 3 and 4, which showed mean angles exceeding 8° of varus. Multi-segment foot models 3 and 4 rely primarily on markers placed on the sustentaculum tali and peroneal tubercle, which are bony landmarks with inherent asymmetry, as the sustentaculum tali lies superior to the peroneal tubercle, leading to a systematic varus bias [27, 28]. In contrast, the mono-segment foot model and multi-segment foot models 1 and 2, which rely primarily on the two posterior calcaneus markers, showed mean hindfoot angles closer to neutral (<3° of varus/valgus), suggesting they better reflect the true study sample. A distinct hindfoot movement pattern was observed across the gait cycle for the Plug-in Gait lower body model compared to the multi-segment foot models. This suggests that multi-segment foot models are more appropriate for measuring changes in hindfoot angle during gait, which aligns with prior research [29]. Additionally, the multi-segment foot models exhibited substantially greater variation in hindfoot angles between participants compared to the mono- segment foot model, suggesting that the mono-segment foot model may underestimate hindfoot angles, potentially due to intra-foot motions or misattributing frontal plane movement to other planes.

This is the first study to investigate the discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus, which is required for clinical applicability. Among all foot models, multi-segment foot model 4 showed the highest discriminative capacity. However, for all foot models, a substantial proportion of feet fell within the overlapping region of the 95% prediction intervals. Notably, neutral-positioned hindfeet were not considered in this outcome measure, indicating that the actual discriminative capacity is even lower than reflected by this analysis. Moreover, the mono-segment foot model frequently measured varus in feet classified as valgus, and vice versa, consistent with previous findings [29]. For multi-segment foot models 3 and 4, feet classified as valgus demonstrated angles within the varus

range, although these varus angles were smaller than those observed in the varus group. This highlights that, while multi-segment foot model 4 performed best, its ability to accurately quantify the absolute hindfoot angle remains limited. These findings align with a previous study that found no significant association between the bony alignment and frontal plane hindfoot angles obtained from multi-segment foot models [27]. In that study, which included only healthy participants with bony hindfoot orientations within ±5° of varus/valgus, the authors concluded that multi-segment foot models may lack the sensitivity to detect angular deviations within such a range. In healthy controls, the typical frontal-plane range of motion of the hindfoot during gait is approximately 10° [10, 30, 31]. In the current study, the average difference between the minimum and maximum mean hindfoot angle of all participants was 40° across multi-segment foot models. Thus, the lack of sensitivity within 10° constitutes a substantial portion of the typical range of motion and the variation in a population with neurological gait impairments.

A limitation of our study is that foot models were compared with clinical examination, which only allowed for categorical, rather than continuous, assessment of hindfoot position. Therefore, further work is needed to compare the absolute hindfoot angles measured by the foot models with those obtained using biplanar fluoroscopy to fully assess the accuracy of the foot models. Thereby, a comprehensive sensitivity analysis should be conducted to assess the discriminative capacity. Furthermore, as clinical examination of the hindfoot position is subjective, this may have introduced inaccuracies. However, in only one of the 194 examinations (97 feet at initial contact and midstance), one rater classified the hindfoot position as varus, while the other as valgus. Thus, despite the subjectivity of the examination, the observed overlap in 95% prediction intervals between hindfoot varus and valgus based on the multi-segment foot models far exceeds the single case of varus/valgus disagreement between raters.

In conclusion, this study indicates that current multi-segment foot models have limited discriminative capacity to identify hindfoot varus and valgus in people with neurological gait impairments. As such, absolute hindfoot angles measured by multi-segment foot models should be interpreted with caution when applied in clinical practice. Further research is therefore needed to develop a valid method for measuring absolute hindfoot angles during gait, for instance by using multi-segment foot models in combination with imaging techniques such as weight-bearing computed tomography (CT) to establish neutral reference positions. Nonetheless, multi-segment foot models seem to be more appropriate for assessing changes in hindfoot angle throughout the gait cycle. Hence, the value of multi-segment foot models for their use in clinical practice should be considered in the context of the specific clinical question being addressed.

References

[1] Nonnekes J, Benda N, Van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[2] Patrick JH, Keenan MAE. Gait analysis to assist walking after stroke. Lancet. 2007;369(9558):256-7. https://doi.org/10.1016/S0140-6736(07)60124-X.

[3] Holmes JR, Hansen Jr ST. Foot and ankle manifestations of Charcot-Marie-Tooth disease. Foot Ankle. 1993;14(8):476-86. https://doi.org./10.1177/107110079301400809.

[4] Nonnekes J, Hofstad C, de Greef-Rotteveel A, van der Wielen H, van Gelder JH, Plaats C, et al. Management of gait impairments in people with Charcot-Marie-Tooth disease: A treatment algorithm. J Rehabil Med. 2021;53(5):1-8. https://doi.org/10.2340/16501977-2831.

[5] Bonato P, Feipel V, Corniani G, Arin-Bal G, Leardini A. Position paper on how technology for human motion analysis and relevant clinical applications have evolved over the past decades: Striking a balance between accuracy and convenience. Gait Posture. 2024;113:191-203. https://doi.org/10.1016/j.gaitpost.2024.06.007.

[6] Leardini A, Caravaggi P, Theologis T, Stebbins J. Multi-segment foot models and their use in clinical populations. Gait Posture. 2019;69:50-9. https://doi.org/10.1016/j.gaitpost.2019.01.022.

[7] Deschamps K, Staes F, Roosen P, Nobels F, Desloovere K, Bruyninckx H, et al. Body of evidence supporting the clinical use of 3D multisegment foot models: A systematic review. Gait Posture. 2011;33(3):338-49. https://doi.org/10.1016/j.gaitpost.2010.12.018.

[8] Mandiroglu S, Firat M. Foot and ankle deformities in stroke: relationship with ambulation, balance, and daily living activities: A cross-sectional study. Somatosens Mot Res. 2021;39(2-4):106-10. https://doi.org/10.1080/08990220.2021.2018294.

[9] Neumann JA, Nickisch F. Neurologic disorders and cavovarus deformity. Foot Ankle Clin. 2019;24(2):195-203. https://doi.org/10.1016/j.fcl.2019.02.003.

[10] Nester C, Jones RK, Liu A, Howard D, Lundberg A, Arndt A, et al. Foot kinematics during walking measured using bone and surface mounted markers. J Biomech. 2007;40(15):3412-23. https://doi.org/10.1016/j.jbiomech.2007.05.019.

[11] Kessler SE, Rainbow MJ, Lichtwark GA, Cresswell AG, D'Andrea SE, Konow N, et al. A direct comparison of biplanar videoradiography and optical motion capture for foot and ankle kinematics. Front Bioeng Biotechnol. 2019;7:199. https://doi.org/10.3389/fbioe.2019.00199.

[12] Leardini A, Benedetti MG, Berti L, Bettinelli D, Nativo R, Giannini S. Rear-foot, mid-foot and fore-foot motion during the stance phase of gait. Gait Posture. 2007;25(3):453-62. https://doi.org/10.1016/j.gaitpost.2006.05.017.

[13] Carson MC, Harrington ME, Thompson N, O'connor JJ, Theologis TN. Kinematic analysis of a multi-segment foot model for research and clinical applications: A repeatability analysis. J Biomech. 2001;34(10):1299-307. https://doi.org/10.1016/s0021-9290(01)00101-4.

[14] Kidder SM, Abuzzahab FS, Harris GF, Johnson JE. A system for the analysis of foot and ankle kinematics during gait. IEEE Trans Rehabil Eng. 1996;4(1):25-32. https://doi.org/10.1109/86.486054.

[15] Davis RB, Jameson EG, Davids JR, Christopher LM, Rogozinski BM, Anderson JP. The design, development, and initial evaluation of a multisegment foot model for routine clinical gait analysis. CRC Press. 2007:425-44.

[16] Saraswat P, MacWilliams BA, Davis RB. A multi-segment foot model based on anatomically registered technical coordinate systems: Method repeatability in pediatric feet. Gait Posture. 2012;35(4):547-55. https://doi.org/10.1016/j.gaitpost.2011.11.022.

[17] Rao S, Saltzman C, Yack HJ. Segmental foot mobility in individuals with and without diabetes and neuropathy. Clin Biomech. 2007;22(4):464-71. https://doi.org/10.1016/j.clinbiomech.2006.11.013.

[18] Paterson KL, Clark RA, Mullins A, Bryant AL, Mentiplay BF. Predicting dynamic foot function from static foot posture: Comparison between visual assessment, motion analysis, and a commercially available depth camera. J Orthop Sports Phys Ther. 2015;45(10):789-98. https://doi.org/10.2519/jospt.2015.5616.

[19] Nester CJ, Jarvis HL, Jones RK, Bowden PD, Liu A. Movement of the human foot in 100 pain free individuals aged 18–45: Implications for understanding normal foot function. J Foot Ankle Res. 2014;7:51. https://doi.org/10.1186/s13047-014-0051-8.

[20] Tsai TY, Lu TW, Kuo MY, Lin CC. Effects of soft tissue artifacts on the calculated kinematics and kinetics of the knee during stair-ascent. J Biomech. 2011;44(6):1182-8. https://doi.org/10.1016/j.jbiomech.2011.01.009.

[21] Portinaro N, Leardini A, Panou A, Monzani V, Caravaggi P. Modifying the Rizzoli foot model to improve the diagnosis of pes-planus: Application to kinematics of feet in teenagers. J Foot Ankle Res. 2014;7:57. https://doi.org/10.1186/s13047-014-0057-2.

[22] Schallig W, van den Noort JC, Piening M, Streekstra GJ, Maas M, van der Krogt MM, et al. The Amsterdam Foot Model: A clinically informed multi-segment foot model developed to minimize measurement errors in foot kinematics. J Foot Ankle Res. 2022;15(1):46. https://doi.org/10.1186/s13047-022-00543-6.

[23] Wu G, Siegler S, Allard P, Kirtley C, Leardini A, Rosenbaum D, et al. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion—part I: Ankle, hip, and spine. J Biomech. 2002;35(4):543-8. https://doi.org/10.1016/s0021-9290(01)00222-6.

[24] Grood ES, Suntay WJ. A joint coordinate system for the clinical description of three-dimensional motions: Application to the knee. J Biomech Eng. 1983;105:136-44. https://doi.org/10.1115/1.3138397.

[25] Zeni Jr JA, Richards JG, Higginson JS. Two simple methods for determining gait events during treadmill and overground walking using kinematic data. Gait Posture. 2008;27(4):710-4. https://doi.org/10.1016/j.gaitpost.2007.07.007.

[26] Pataky TC, Vanrenterghem J, Robinson MA. Zero-vs. one-dimensional, parametric vs. non-parametric, and confidence interval vs. hypothesis testing procedures in one-dimensional biomechanical trajectory analysis. J Biomech. 2015;48(7):1277-85. https://doi.org/10.1016/j.jbiomech.2015.02.051.

[27] Hulshof CM, Schallig W, van den Noort JC, Streekstra GJ, Kleipool RP, Dobbe JGG, et al. Skin marker-based versus bone morphology-based coordinate systems of the hindfoot and forefoot. J Biomech. 2024;166:112001. https://doi.org/10.1016/j.jbiomech.2024.112001.

[28] Novak AC, Mayich DJ, Perry SD, Daniels TR, Brodsky JW. Gait analysis for foot and ankle surgeons—topical review, part 2: Approaches to multisegment modeling of the foot. Foot Ankle Int. 2013;35(2):178-91. https://doi.org/10.1177/1071100713511435.

[29] Pothrat C, Authier G, Viehweger E, Berton E, Rao G. One-and multi-segment foot models lead to opposite results on ankle joint kinematics during gait: Implications for clinical assessment. Clin Biomech. 2015;30(5):493-9. https://doi.org/10.1016/j.clinbiomech.2015.03.004.

[30] Westblad P, Hashimoto T, Winson I, Lundberg A, Arndt A. Differences in ankle-joint complex motion during the stance phase of walking as measured by superficial and bone-anchored markers. Foot Ankle Int. 2002;23(9):856-63. https://doi.org/10.1177/107110070202300914.

[31] Lundgren P, Nester C, Liu A, Arndt A, Jones R, Stacoff A, et al. Invasive in vivo measurement of rear-, mid-and forefoot motion during walking. Gait Posture. 2008;28(1):93-100. https://doi.org/10.1016/j.gaitpost.2007.10.009.



Chapter 3

Plantar pressure in relation to hindfoot varus in people with unilateral upper motor neuron syndrome

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Abstract

Background

Hindfoot varus deformity is common in people with unilateral upper motor neuron syndrome (UMNS) and can be dynamic or persistent. The aims of this study were (1) to gain insight into plantar pressure characteristics of people with chronic UMNS in relation to hindfoot varus and (2) to propose a quantitative outcome measure, based on plantar pressure, for the scientific evaluation of surgical interventions.

Methods

In this retrospective study, a cohort comprising plantar pressure data of 49 people with UMNS (22 “no hindfoot varus”, 18 “dynamic hindfoot varus”, and 9 “persistent hindfoot varus”), and 586 healthy controls was analyzed. As an indication of plantigrade foot contact, the ratio between the plantar contact area of the affected and the non-affected foot was calculated. To investigate spatial and temporal aspects of plantar pressure, normalized plantar pressure patterns and center of pressure trajectories were computed.

Results

People with UMNS had lower plantar pressure area ratios compared to healthy controls. Additionally, increased plantar pressure underneath the lateral foot was found in people with a persistent hindfoot varus. Center of pressure trajectories were more lateral during the first 26% of the stance phase in people with a dynamic hindfoot varus and during the first 82% of the stance phase in people with a persistent hindfoot varus compared to healthy controls.

Conclusion

Spatial and temporal differences in plantar pressure were found in people with dynamic or persistent hindfoot varus deformity. We propose to primarily use the medio-lateral center of pressure trajectory as outcome measure for the scientific evaluation of surgical interventions targeting hindfoot varus.

Introduction

Hindfoot varus deformities are frequently observed in people with chronic unilateral upper motor neuron syndrome (UMNS), such as stroke or traumatic brain injury [1-3]. They result from a disturbed muscle balance around the foot and ankle joint and can be dynamic or persistent [4]. A dynamic hindfoot varus, arising from muscle weakness and/or spasticity, is not continuously present during standing and walking but can severely affect positioning of the foot at initial contact when present during the terminal swing phase and loading response of walking. This is often caused by a relatively strong tibialis anterior compared to the lateral foot elevators [4]. Prolonged spastic overactivity of the lower leg muscles, most commonly the tibialis posterior, may result in muscle contractures [5], causing dynamic foot deformity evolving into persistent deformity. A persistent hindfoot varus severely impairs stability during the entire stance phase [4].

To improve standing and walking capacity, treatment of hindfoot varus in people with UMNS is essential. For appropriate treatment selection and for the scientific evaluation of surgical interventions, an accurate evaluation of the deformity is crucial [6]. Over the last decades, numerous multi-segment foot models (MSFM) have been developed to quantitatively evaluate foot function using a three-dimensional instrumented gait analysis. However, only a limited number of validation studies have been performed since a direct comparison with the underlying bone structures is challenging and can only be achieved with a sophisticated videofluoroscopy setup or by in vitro or in vivo bone pin measurements [7]. Studies examining the accuracy of MSFMs have reported moderate results for measuring hindfoot varus/valgus angles, indicated by a root mean squared error (RMSE) of 3.6° compared to videofluoroscopy [8] and an error of up to 4.8° compared to bone mounted markers [9]. Moreover, performing an instrumented gait analysis is very time consuming and requires the availability of a three-dimensional motion lab.

An alternative method for evaluating foot function is plantar pressure assessment [10-15]. Research indicates that in people with chronic UMNS, peak pressures underneath the forefoot are lower compared to those in healthy controls [10, 11, 15]. Additionally, a diminished plantar contact area has been observed for the affected foot compared to the non-affected foot in people with UMNS [13]. However, previous studies included people with chronic UMNS, irrespective of foot deformities, or did not distinguish between different types of foot deformities [10-15], which makes it difficult to draw conclusions on the clinical value of plantar pressure measurements for the evaluation of foot deformities. Furthermore, prior studies that investigated plantar pressure in people with chronic UMNS primarily focused on the spatial aspect

of plantar pressure [10-15], whereas most studies lacked evaluations of the temporal aspect. Especially for dynamic foot deformity, relying solely on spatial analyses of plantar pressure could potentially lead to underestimation of the deformity, as dynamic deformities may only be present during short periods within the stance phase. Therefore, a temporal investigation of plantar pressure may provide more profound insights here. Hence, a comprehensive evaluation examining the relationship between foot deformities and both spatial and temporal plantar pressure characteristics would be a valuable initial step to investigate the clinical value of plantar pressure measurements for the evaluation of hindfoot varus deformities in people with UMNS.

The aims of this study were (1) to gain insight into spatial and temporal plantar pressure characteristics of people with chronic UMNS in relation to dynamic and persistent hindfoot varus and (2) to propose a quantitative outcome measure based on plantar pressure for the scientific evaluation of surgical interventions. We hypothesized that the difference in plantar contact area between the affected and the non-affected foot would increase with the severity of hindfoot varus, i.e. the greatest difference in persistent hindfoot varus, followed by dynamic hindfoot varus and, ultimately, no hindfoot varus. Additionally, we hypothesized to find increased pressure underneath the lateral forefoot and decreased pressure underneath the central and medial forefoot in the case of dynamic or persistent hindfoot varus, as seen in other neurological disorders with hindfoot varus [16, 17]. Regarding the temporal aspect of plantar pressure, we hypothesized lateralized center of pressure throughout the gait phases when hindfoot varus is present, as has been observed in healthy subjects walking with an inverted foot position [18].

Methods

Participants and design

In this retrospective study, adults with chronic unilateral UMNS, including stroke and traumatic brain injury, who had visited the mobility outpatient clinic of the Gait Expertise Center of the Sint Maartenskliniek between March 2018 and October 2022 for the assessment of neurological gait impairments, were screened for inclusion. Chronic UMNS was defined as at least 6 months post onset of the brain lesion. Only subjects with at least three plantar pressure measurements of both feet and adequate photographs and video recordings of the affected foot were included. The exclusion criteria were: (1) previous foot/ankle surgery on the affected side, (2) a previous foot/ankle fracture on the affected side, and (3) comorbidities that might cause ankle-foot deformity. In addition, a group of 586 healthy controls without disabilities that might affect their gait, including the presence of foot deformities, were included as a

reference. The healthy control data has been described earlier [19] and was expanded by 174 people.

Examinations

Classification of all affected UMNS feet into foot deformity categories was performed based on photographs and video recordings. First, the hindfoot position during stance was classified as "varus" or "no varus" based on photographs of the posterior hindfoot. This was independently done by two raters who discussed their assessment in case of discrepancy to reach consensus. Subsequently, the hindfoot position during gait was classified as "varus" or "no varus" based on close-up video recordings of the posterior hindfoot during walking. One rater initially assessed these video recordings, which was subsequently discussed with a second rater. Each rater had extensive experience, with at least 2 years of expertise in assessing hindfoot varus in individuals with UMNS. Finally, all affected feet were classified into three subgroups: (1) no hindfoot varus during standing and walking ("no hindfoot varus"), (2) no hindfoot varus during standing, but a varus position of the hindfoot during the loading response phase of walking ("dynamic hindfoot varus"), and (3) a hindfoot varus during standing and the entire stance phase of walking ("persistent hindfoot varus").

Plantar pressure measurements were performed with a 0.5 m long Footscan pressure plate (RSscan) placed on top of a force plate (Kistler). The pressure plate and force plate were synchronized with an RSscan 3D-box and plantar pressure patterns were recorded at 500 Hz. Three to six barefoot walking trials were performed for both feet at preferred walking speed.

Data analyses

As indicator of plantigrade loading, the ratio between the plantar contact area of the affected and the non-affected foot was calculated. This was done by determining the contour of the mean plantar pressure pattern over the stance phase at 0.5 N/cm^2. After that, the area within the contour was calculated and averaged across three to six walking trials for each foot. Subsequently, the mean plantar contact area of the affected foot was divided by the sum of the mean plantar contact areas of the affected and non-affected foot, which will be referred to as the plantar pressure area ratio. For healthy controls, it was randomized whether the left or right foot was used as the numerator.

To be able to compare plantar pressure among subjects, plantar pressure patterns were normalized for foot size and foot progression angle according to a previously described method [20]. The focus of this study was on the plantar pressure distribution rather than absolute pressure values, as absolute values are highly influenced by

body weight and walking velocity [19]. Therefore, the pressure of each pixel was divided by the sum of the pressures of all pixels. Average normalized plantar pressure patterns over three to six measurements were calculated for each subject and multiplied by the mean total pressure of all subjects. Furthermore, center of pressure trajectories in the medio-lateral and anterior-posterior directions were calculated and normalized to foot length and width based on the pressure area. Foot length was defined as the distance between the most posterior part of the heel and the most anterior part of the toes. Foot width was defined as the distance between the most medial and the most lateral part of the forefoot. Finally, stance time was calculated and used as an indicator of walking velocity [21].

Statistical analyses

To assess the validity of the plantar pressure area ratio as outcome measure, analyses were conducted using the data from healthy controls. First, the mean absolute difference between the plantar contact area of the left and right foot was calculated to quantify differences in plantar contact area in individuals without foot deformities. Additionally, R-squared (R^2) was calculated to examine the correlation between the plantar contact areas of both feet in healthy controls.

One-way analyses of variance (ANOVA) and post hoc independent samples t-tests with Bonferroni correction were performed to compare the plantar pressure area ratios and stance times between (sub)groups. A p-value lower than 0.05 was considered statistically significant.

To compare the normalized plantar pressure patterns at pixel level, independent samples t-tests were performed. A p-value lower than 0.0125 was considered statistically significant. This p-value was calculated by dividing the initial p-value of 0.05 by the number of clusters of pixels with a similar difference from the plantar pressure pattern of healthy controls, which was determined to be four. This method to correct the p-value for the number of comparisons has previously been used in the analysis of plantar pressure patterns [22] and was initially derived from neuroscience [23].

Center of pressure trajectories were statistically compared between (sub)groups with statistical parametric mapping [24]. One-way ANOVA and post hoc independent samples t-tests with Bonferroni correction were used to compare the center of pressure trajectories. Statistical significance was defined as $p<0.05$.

Results

One hundred and four adults with UMNS were screened for eligibility. Out of these, 92 subjects had at least three plantar pressure measurements of both feet and good photographs and video recordings of the affected foot. Forty-three subjects were excluded because of previous surgery on the affected foot/ankle (n=39), a previous foot/ankle fracture on the affected side (n=3), or comorbidity that might cause ankle-foot deformity (n=1). Ultimately, 49 participants were included. Among them, 22 were classified as having "no hindfoot varus", 18 as having "dynamic hindfoot varus", and nine with "persistent hindfoot varus". Table 1 presents the subject characteristics.

Table 1. Subject characteristics

Group	n (people)	Sex (#female/#male)	Age (years, mean ± SD)	Affected side (#left/ #right)	Type of UMNS (#stroke/ #traumatic brain injury)
Healthy controls	586	294/292	49 ± 13	-	-
No hindfoot varus	22	8/14	54 ± 11	15/7	18/4
Dynamic hindfoot varus	18	9/9	51± 12	9/9	17/1
Persistent hindfoot varus	9	2/7	49 ± 15	5/4	6/3

SD: standard deviation, UMNS: unilateral upper motor neuron syndrome.

Plantar pressure areas

The mean absolute difference between the plantar contact area of the left and right foot for healthy controls was 5.1 ± 3.9 cm^2. The R-squared value between the plantar contact area of the left and right foot for healthy controls was 0.86, which implies that 86% of the variance in a plantar contact area can be explained by the plantar contact area of the contralateral foot.

Figure 1 shows the plantar pressure area ratios. Plantar pressure area ratios were significantly different between (sub)groups ($F(3,631) = 72.1$, $p<0.001$). The plantar pressure area ratio was significantly smaller in all UMNS subgroups compared to healthy controls ($p<0.001$, Figure 1). Furthermore, the plantar pressure area ratio was significantly smaller in the "persistent hindfoot varus" subgroup compared to the "no hindfoot varus" and "dynamic hindfoot varus" subgroups ($p<0.001$, Figure 1).

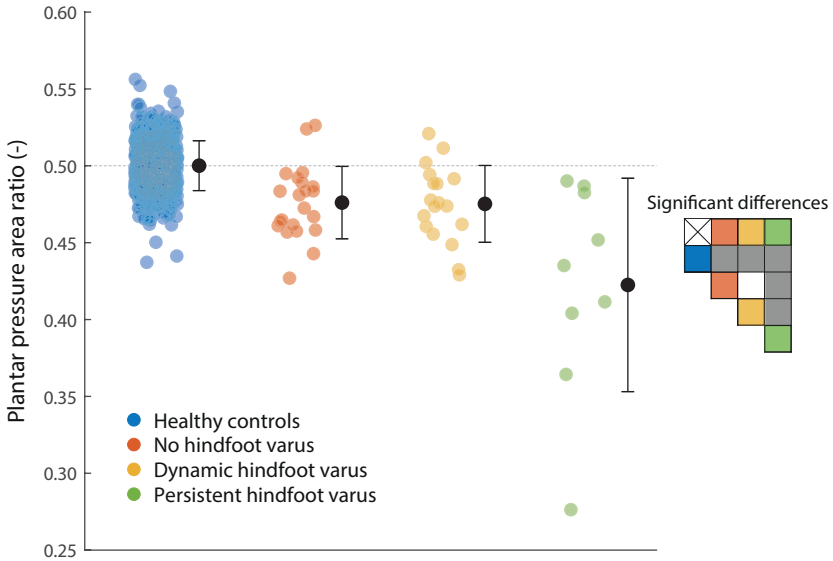


Figure 1. Plantar pressure area ratios. The plantar pressure area ratio is defined as the plantar pressure area of the affected foot divided by the sum of the plantar pressure areas of the affected and non-affected foot. Plantar pressure area ratios for all feet and the means and standard deviations for each (sub)group are presented. Significant differences between (sub) groups are shown in the table on the right side of the figure. A gray box indicates a significant difference between (sub)groups, whereas a white box indicates no significant difference between (sub)groups.

Normalized plantar pressure patterns

Seven feet could not be normalized because information about the complete foot shape was lacking due to severe equinus or varus deformity. This included one foot from the "no hindfoot varus" subgroup (plantar pressure area ratio: 0.43), two feet from the "dynamic hindfoot varus" subgroup (plantar pressure area ratios: 0.43, 0.45), and four feet from the "persistent hindfoot varus" subgroup (plantar pressure area ratios: 0.28, 0.36, 0.41, 0.44). Therefore, 21 feet classified as "no hindfoot varus", 16 feet classified as "dynamic hindfoot varus", and five feet classified as "persistent hindfoot varus" were included in all subsequent analyses.

Figure 2 presents the normalized plantar pressure patterns. A significant decrease in plantar pressure underneath the second/third metatarsal heads was found for all UMNS subgroups compared to healthy controls. A significant increase in plantar pressure underneath the midfoot was found for the "dynamic hindfoot varus" and "persistent hindfoot varus" subgroups. Furthermore, a significant increase in plantar

pressure underneath the fifth metatarsal head was found for the "persistent hindfoot varus" subgroup compared to healthy controls.

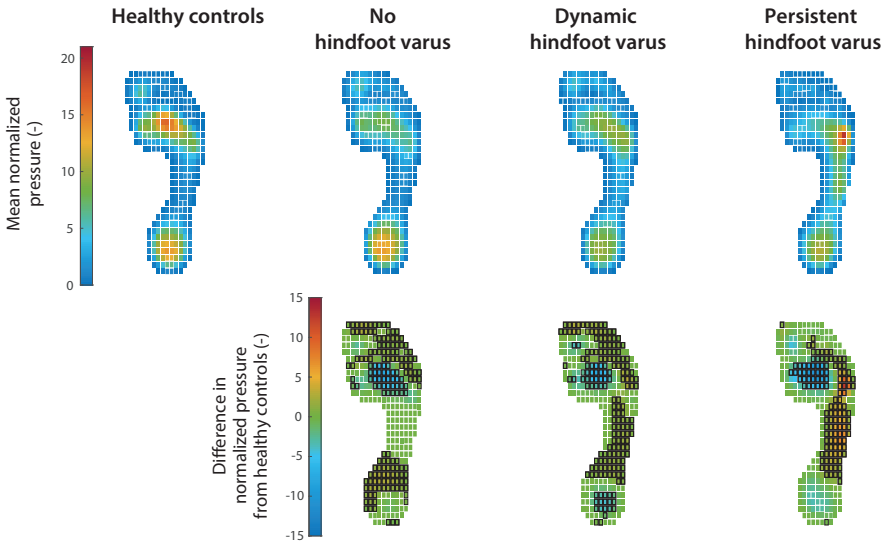


Figure 2. Plantar pressure patterns. The plantar pressure patterns of the unilateral upper motor neuron syndrome subgroups and healthy controls averaged over the stance phase are presented in the top panels. The lower panels present the differences in normalized plantar pressure from healthy controls. Positive values indicate an increase in plantar pressure, whereas negative values indicate a decrease in plantar pressure compared to healthy controls. Pixels with a black border indicate a statistically significant difference ($p<0.0125$).

Center of pressure trajectories

Center of pressure trajectories are shown in Figure 3. In the medio-lateral direction, the center of pressure trajectory was significantly different between (sub)groups during almost the entire stance phase, except between 92% and 95% of the stance phase ($F(3,624) = 4.6$, $p<0.04$). Post hoc tests revealed a lateralized center of pressure during the first 26% of the stance phase for the "dynamic hindfoot varus" subgroup compared to healthy controls ($p<0.001$). For the "persistent hindfoot varus" subgroup, the center of pressure was significantly more lateral during the first 82% of the stance phase compared to healthy controls ($p<0.001$).

In the anterior-posterior direction, the center of pressure trajectory was significantly different between (sub)groups during the first 36% and between 44% and 94% of the stance phase ($F(3,624) = 4.6$, $p<0.001$). The center of pressures of all UMNS

subgroups were more anterior during the first 30% of the stance phase compared to healthy controls ($p<0.001$). Furthermore, between 53% and 77% of the stance phase, the center of pressures of all UMNS subgroups were more posterior compared to healthy controls ($p<0.001$). All other statistical parametric mapping results are provided in Supplementary material 1.

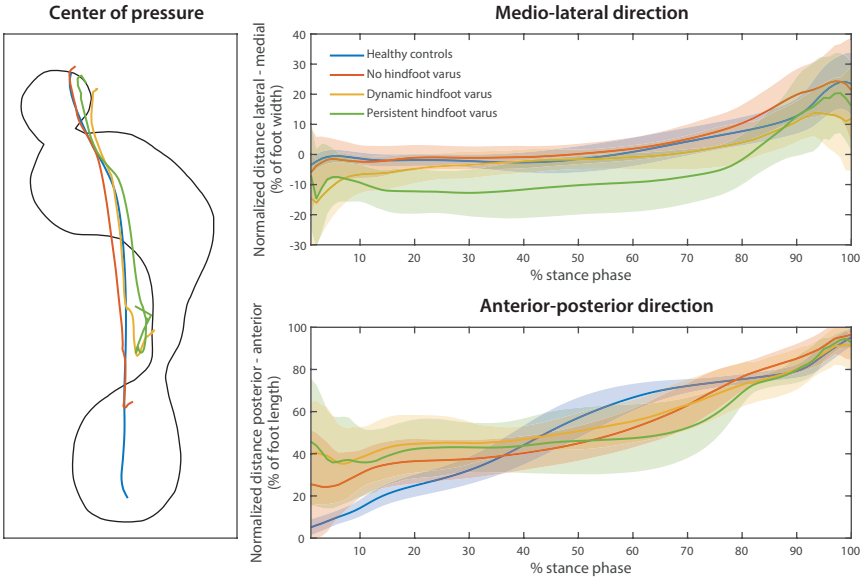


Figure 3. Center of pressure trajectories. In the left panel, the mean center of pressure trajectory is shown for all (sub)groups. In the right panels, the mean center of pressure position ± standard deviation in the medio-lateral (upper right panel) and anterior-posterior (lower right panel) directions over the stance phase are presented. In the medio-lateral direction, the y-axis indicates the normalized distance from the medio-lateral center of the foot, expressed as a percentage of foot width. In the anterior-posterior direction, the y-axis indicates the normalized distance from the most posterior part of the heel, expressed as a percentage of foot length.

Stance time was significantly different between (sub)groups ($F(3,624) = 96.1$, $p<0.001$). Stance time was significantly longer for the UMNS subgroups ("no hindfoot varus": 0.96 ± 0.27 s, "dynamic hindfoot varus": 0.99 ± 0.35 s, "persistent hindfoot varus": 0.90 ± 0.09 s) compared to healthy controls (0.67 ± 0.08 s). No significant differences in stance time were found between UMNS subgroups.

Discussion

This study revealed diminished plantar pressure area ratios among all UMNS subgroups when compared to healthy controls. In the case of persistent hindfoot varus, an increased lateral foot pressure was observed. Notably, the center of pressure was lateralized during the first 26% of the stance phase for a dynamic hindfoot varus and during the first 82% of the stance phase for a persistent hindfoot varus. Additionally, a more anterior foot landing (0%-30% of the stance phase) and a more posterior center of pressure between 53% and 77% of the stance phase was observed in all UMNS subgroups compared to healthy controls. These findings highlight spatial and temporal differences in plantar pressure in relation to hindfoot varus in people with UMNS.

The smaller plantar pressure area ratios in people with UMNS imply reduced plantigrade support on the affected side, which is in line with previous findings [13]. Although a significantly smaller plantar pressure area ratio was found for persistent hindfoot varus compared to all other (sub)groups, the between-subject variability was high. This variability can be attributed to differences in severity of hindfoot varus between subjects. Additionally, increased midfoot loading caused by hindfoot varus disturbed the plantar pressure area ratio. In some people, substantial plantar pressure was detected underneath the midfoot of their affected foot due to hindfoot varus, whereas no plantar pressure was detected underneath the midfoot of their non-affected foot. This resulted in only a small difference in plantar contact area between both feet, whereas a large difference in plantar pressure distribution existed. Therefore, the plantar pressure area ratio alone does not seem to be a valuable outcome measure to evaluate hindfoot varus deformity in people with UMNS.

The normalized plantar pressure patterns revealed decreased plantar pressure underneath the central forefoot in all UMNS subgroups, which is consistent with previous findings [10, 15]. Furthermore, spatial deviations in plantar pressure in individuals with a persistent hindfoot varus were found when compared to healthy controls. For people with a dynamic hindfoot varus, the differences from healthy controls were less pronounced. Hence, evaluation of the mean normalized plantar pressure pattern is useful for the assessment of persistent hindfoot varus but seems to be less valuable for the evaluation of dynamic hindfoot varus. Dynamic hindfoot varus manifests only briefly during the stance phase, which means that averaging the plantar pressure pattern over the entire stance phase may consequently result in underestimation of dynamic deformities. To gain further insight into foot function in people with dynamic hindfoot varus, additional analyses focusing on the temporal aspect of plantar pressure should be performed.

The temporal aspect of plantar pressure was evaluated by the center of pressure trajectory. The center of pressure was lateralized when hindfoot varus was present, i.e. during the first part of the stance phase for dynamic hindfoot varus and during almost the entire stance phase for persistent hindfoot varus. Furthermore, a more anterior foot strike and a more posterior center of pressure during 53%-77% of the stance phase were observed in all UMNS subgroups compared to healthy controls. The reduced anterior-posterior center of pressure displacement is in agreement with previous findings [25, 26]. Our results indicate that, especially for dynamic foot deformities, investigation of plantar pressure over time provides better insight into the biomechanical deviations compared to investigation of spatial aspects of plantar pressure alone.

A recent systematic review on assessment methods for the evaluation of foot surgery in people following stroke highlighted the need for an accurate and objective method to analyze foot function [6]. Based on our results, we propose to use the medio-lateral center of pressure trajectory over time as outcome measure for the evaluation of surgical interventions targeting hindfoot varus as it can identify hindfoot varus throughout the stance phase of gait for persistent as well as dynamic deformities. In addition, for the evaluation of persistent hindfoot varus, a comprehensive analysis of the complete plantar pressure pattern could be valuable to gain more insight into the exact location of plantar pressure deviations. Further research is needed to investigate the responsiveness of these outcome measures to surgical interventions. Furthermore, future studies should examine the correlation between these measures and other clinical and functional outcomes to assess their validity as evaluation tools.

A limitation of this study is that not all plantar pressure patterns could be normalized due to a lack of information on the complete foot shape, which was the case for seven people with UMNS in this study with severe equinus or varus deformity. While their exclusion is unlikely to have had a significant impact on the results, as including more severe foot deformities would probably have led to even more pronounced differences between (sub)groups, it underscores a moderate feasibility of the normalization method for plantar pressure patterns in individuals with UMNS. Consequently, future work is needed to develop a normalization method that is applicable to all plantar pressure patterns, including those with severe deviations. Such an approach could involve anthropometric measurements of the foot to overcome the limitation associated with the lack of information on the complete foot shape. Another limitation of this study is that people with UMNS had a longer stance time compared to healthy controls. Since decreased walking velocity is associated with spatial and temporal differences in plantar pressure [21, 27, 28], the results reported in this study could partially be explained by a lower walking speed in people with UMNS as

represented by the longer stance time. Nonetheless, stance time was not different between UMNS subgroups, whereas the spatial and temporal plantar pressure characteristics were different between these subgroups. In addition, stance time may not be a valid indicator of walking speed in individuals with UMNS since stance time of the affected leg may be significantly reduced compared to the non-affected leg [29]. However, stance time was not used to accurately estimate walking speed in this study but only to provide an impression on the potential differences in walking speed between (sub)groups and consequently the influence of these differences on plantar pressure. Lastly, this study related plantar pressure to classification of hindfoot varus deformities, which makes it impossible to study hindfoot varus deformities on a continuous scale. To address this limitation, further research is recommended to validate plantar pressure measurements for studying foot function on a continuous scale, for example by using biplanar videofluoroscopy. This will allow to compare the plantar pressure patterns with the hindfoot angle during gait and can, therefore, provide further insight into the value of plantar pressure measurements for the evaluation of foot deformities.

Conclusion

This study revealed spatial and temporal differences in plantar pressure among individuals with UMNS, in relation to dynamic and persistent hindfoot varus deformity. We propose to use the medio-lateral center of pressure trajectory as key outcome measure for the scientific evaluation of surgical interventions that aim to correct the hindfoot position. In the case of a persistent hindfoot varus, a comprehensive analysis of the complete plantar pressure pattern could offer additional valuable insights. Future work should now focus on investigating the validity and responsiveness of the proposed outcome measures.

References

[1] Mandiroglu S, Firat M. Foot and ankle deformities in stroke: relationship with ambulation, balance, and daily living activities: A cross-sectional study. Somatosens Mot Res. 2021;39(2:4):106-10. https://doi.org/10.1080/08990220.2021.2018294.

[2] Forghany S, Tyson S, Nester C, Preece S, Jones R. Foot posture after stroke: Frequency, nature and clinical significance. Clin Rehabil. 2011;25(11):1050-5. https://doi.org/10.1177/0269215511410581.

[3] Li S. Ankle and foot spasticity patterns in chronic stroke survivors with abnormal gait. Toxins. 2020;12(10):646. https://doi.org/10.3390/toxins12100646.

[4] Nonnekes J, Benda N, van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[5] Gracies JM. Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. Muscle Nerve. 2005;31(5):535-51. https://doi.org/10.1002/mus.20284.

[6] de l'Escalopier N, Voisard C, Michaud M, Moreau A, Jung S, Tervil B, et al. Evaluation methods to assess the efficacy of equinovarus foot surgery on the gait of post-stroke hemiplegic patients: A literature review. Front Neurol. 2022;13(1042667):1-18. https://doi.org/10.3389/fneur.2022.1042667.

[7] Leardini A, Caravaggi P, Theologis T, Stebbins J. Multi-segment foot models and their use in clinical populations. Gait Posture. 2019;69:50-9. https://doi.org/10.1016/j.gaitpost.2019.01.022.

[8] Kessler SE, Rainbow MJ, Lichtwark GA, Cresswell AG, D'Andrea SE, Konow N, et al. A direct comparison of biplanar videoradiography and optical motion capture for foot and ankle kinematics. Front Bioeng Biotechnol. 2019;7:199. https://doi.org/10.3389/fbioe.2019.00199.

[9] Nester C, Jones RK, Liu A, Howard D, Lundberg A, Arndt A, et al. Foot kinematics during walking measured using bone and surface mounted markers. J Biomech. 2007;40(15):3412-23. https://doi.org/10.1016/j.jbiomech.2007.05.019.

[10] Meyring S, Diehl RR, Milani TL, Hennig EM, Berlit P. Dynamic plantar pressure distribution measurements in hemiparetic patients. Clin Biomech. 1997;12(1):60-5. https://doi.org/10.1016/S0268-0033(96)00050-2.

[11] Forghany S, Nester C, Tyson S, Preece S, Jones R. Plantar pressure distribution in people with stroke and association with functional mobility. J Rehabil Sci Res. 2019;6(2):80-5. https://doi.org/10.30476/JRSR.2019.81232.

[12] Kim TH, Yoon JS, Lee JH. The effect of ankle joint muscle strengthening training and static muscle stretching training on stroke patients' C.O.P sway amplitude. J Phys Ther Sci. 2013;25(12):1613-6. https://doi.org/10.1589/jpts.25.1613.

[13] Kawate N, Matsumoto M, Iijima S. Comparison of plantar pressure and plantar contact area before and after botulinum toxin type A (BoNT-A) therapy in stroke patients with lower limb spasticity. Showa Med Univ J. 2017;29(2):193-200. https://doi.org/10.15369/sujms.29.193.

[14] Lee NK, Kwon JW, Son SM, Nam SH, Choi YW, Kim CS. Changes of plantar pressure distributions following open and closed kinetic chain exercise in patients with stroke. NeuroRehabilitation. 2013;32(2):385-90. https://doi.org/10.3233/NRE-130859.

[15] Chen C, Hong PW, Chen C, Chou SW, Wu C, Cheng P, et al. Ground reaction force patterns in stroke patients with various degrees of motor recovery determined by plantar dynamic analysis. Chang Gung Med J. 2007;30(1):62-71.

[16] Stebbins J, Giacomozzi C, Theologis T. Correlation between plantar pressure and Oxford Foot Model kinematics. J Foot Ankle Res. 2008;1(Suppl 1):1-2. https://doi.org/10.1186/1757-1146-1-S1-O22.

[17] Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Quantitative assessment of plantar pressure patterns in relation to foot deformities in people with hereditary motor and sensory neuropathies. J Neuroeng Rehabil. 2023;20:65. https://doi.org/10.1186/s12984-023-01172-1.

[18] Lugade V, Kaufman K. Center of pressure trajectory during gait: a comparison of four foot positions. Gait Posture. 2014;40(4):719-22. https://doi.org/10.1016/j.gaitpost.2014.07.001.

[19] Keijsers NLW, Goonetilleke RS. Plantar pressure analysis. The Science of Footwear. 2012:377-408. https://doi.org/10.1201/b13021.

[20] Keijsers NLW, Stolwijk NM, Nienhuis B, Duysens J. A new method to normalize plantar pressure measurements for foot size and foot progression angle. J Biomech. 2009;42(1):87-90. https://doi.org/10.1016/j.jbiomech.2008.09.038.
[21] Rosenbaum D, Hautmann S, Gold M, Claes L. Effects of walking speed on plantar pressure patterns and hindfoot angular motion. Gait Posture. 1994;2(3):191-7. https://doi.org/10.1016/0966-6362(94)90007-8.
[22] Keijsers NLW, Stolwijk NM, Louwerens JWK, Duysens J. Classification of forefoot pain based on plantar pressure measurements. Clin Biomech. 2013;28(3):350-6. https://doi.org/10.1016/j.clinbiomech.2013.01.012.
[23] Maris E, Oostenveld R. Nonparametric statistical testing of EEG-and MEG-data. J Neurosci Methods. 2007;164(1):177-90. https://doi.org/10.1016/j.jneumeth.2007.03.024.
[24] Pataky TC, Vanrenterghem J, Robinson MA. Zero-vs. one-dimensional, parametric vs. non-parametric, and confidence interval vs. hypothesis testing procedures in one-dimensional biomechanical trajectory analysis. J Biomech. 2015;48(7):1277-85. https://doi.org/10.1016/j.jbiomech.2015.02.051.
[25] Valentini FA, Granger B, Hennebelle DS, Eythrib N, Robain G. Repeatability and variability of baro-podometric and spatio-temporal gait parameters–results in healthy subjects and in stroke patients. Neurophysiol Clin. 2011;41(4):181-9. https://doi.org/10.1016/j.neucli.2011.08.004.
[26] Khalil N, Chauvière C, Le Chapelain L, Guesdon H, Speyer E, Bouaziz H, et al. Plantar pressure displacement after anesthetic motor block and tibial nerve neurotomy in spastic equinovarus foot. J Rehabil Res Dev. 2016;53(2):219-28. https://doi.org/10.1682/jrrd.2014.11.0298.
[27] Burnfield JM, Few CD, Mohamed OS, Perry J. The influence of walking speed and footwear on plantar pressures in older adults. Clin Biomech. 2004;19(1):78-84. https://doi.org/10.1016/j.clinbiomech.2003.09.007.
[28] Pataky TC, Robinson MA, Vanrenterghem J, Savage R, Bates KT, Crompton RH. Vector field statistics for objective center-of-pressure trajectory analysis during gait, with evidence of scalar sensitivity to small coordinate system rotations. Gait Posture. 2014;40(1):255-8. https://doi.org/10.1016/j.gaitpost.2014.01.023.
[29] Patterson KK, Gage WH, Brooks D, Black SE, McIlroy WE. Evaluation of gait symmetry after stroke: A comparison of current methods and recommendations for standardization. Gait Posture. 2010;31(2):241-6. https://doi.org/10.1016/j.gaitpost.2009.10.014.

Supplementary material 1: statistical parametric mapping results

Figures S1 and S2 show the statistical parametric mapping post hoc test results performed on the center of pressure trajectories. In the medio-lateral direction, the center of pressure trajectory of the "no hindfoot varus" subgroup was significantly more medial between 83% and 92% of the stance phase compared to healthy controls ($p < 0.02$). For the "dynamic hindfoot varus" subgroup, the center of pressure trajectory was significantly more lateral compared to healthy controls in the first 26% of the stance phase and from 95% till the end of the stance phase ($p < 0.04$). For the "persistent hindfoot varus" subgroup, the center of pressure position was significantly more lateral during the first 82% of the stance phase compared to healthy controls ($p < 0.001$). Furthermore, the center of pressure trajectory was significantly more lateral for the "persistent hindfoot varus" subgroup compared to the "no hindfoot varus" subgroup between 16% and 79% of the stance phase ($p < 0.001$).

In the anterior-posterior direction, the center of pressure trajectories of all UMNS subgroups were more anterior during the first part of the stance phase compared to healthy controls ("no hindfoot varus": between 1-30% of the stance phase, "dynamic hindfoot varus": between 1-36% of the stance phase, "persistent hindfoot varus": between 1-30% of the stance phase, $p < 0.001$). Furthermore, later in the stance phase, the center of pressure trajectories of all UMNS subgroups were more posterior compared to healthy controls ("no hindfoot varus": between 44-77% of the stance phase, "dynamic hindfoot varus": between 53-80% of the stance phase, "persistent hindfoot varus": between 53-82% of the stance phase, $p < 0.001$). For the "no hindfoot varus" subgroup, the center of pressure trajectory was more anterior between 81% and 95% of the stance phase compared to healthy controls ($p < 0.003$).

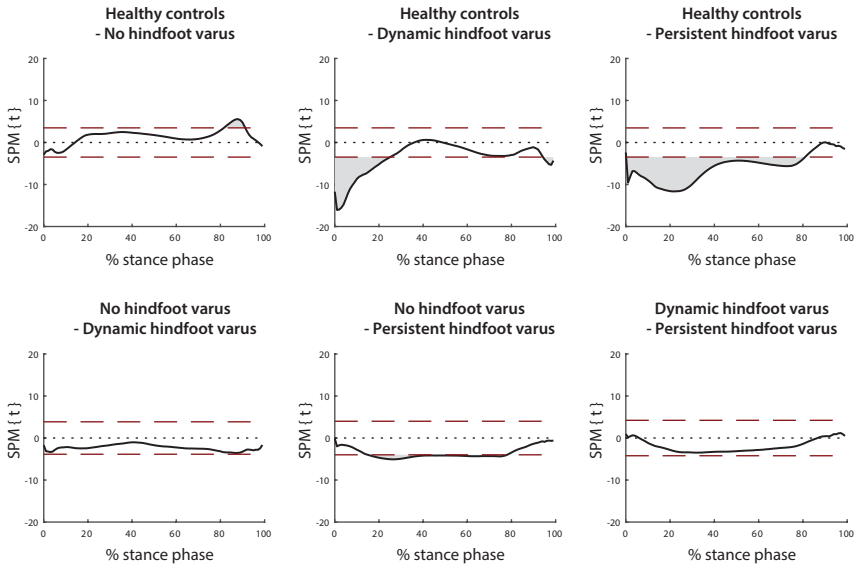


Figure S1. Statistical parametric mapping post hoc test results on the center of pressure trajectory in the medio-lateral direction. Each panel presents the test statistics for a comparison between (sub)groups. In case the black line exceeds the critical t threshold (shown in red), the difference in center of pressure trajectory is statistically significant.

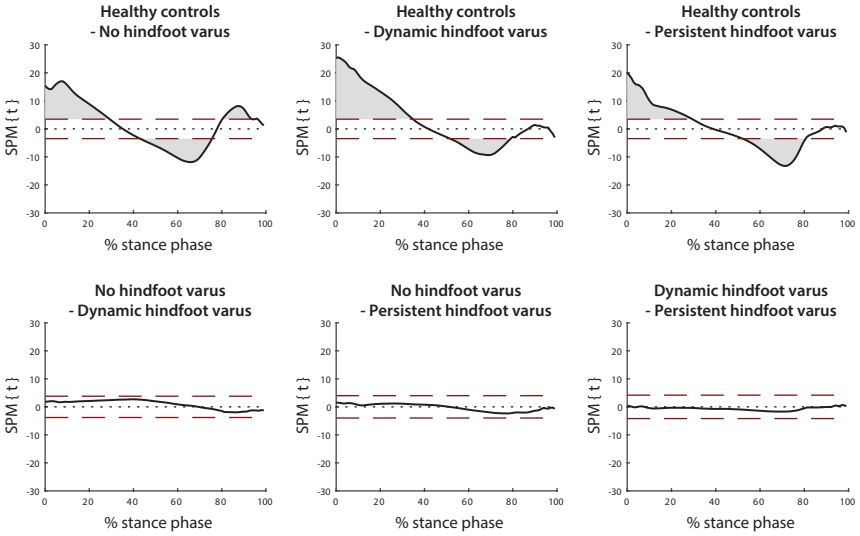


Figure S2. Statistical parametric mapping post hoc test results on the center of pressure trajectory in the anterior-posterior direction. Each panel presents the test statistics for a comparison between (sub)groups. In case the black line exceeds the critical t threshold (shown in red), the difference in center of pressure trajectory is statistically significant.

Chapter 4

Stiff knee gait after stroke: The potential compensatory role of mid-swing rectus femoris activity

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Abstract

Reduced knee flexion during the swing phase of gait, commonly referred to as "stiff knee gait", is frequently encountered in patients with upper motor neuron syndrome, e.g. due to stroke. Rectus femoris spasticity is one of the main causes of stiff knee gait and can be treated with botulinum toxin (BoNT-A) injections. However, previous literature shows large response variations after BoNT-A treatment between individual participants. These variations could be due to the overestimation of rectus femoris spasticity during gait analyses based on the current main indicator: mid-swing rectus femoris activity. The objective of this video-illustrated case series, including gait data of four stroke patients with stiff knee gait, is to propose an alternative explanation for this mid-swing rectus femoris activity. The presented patients all show mid-swing rectus femoris activity, which could be considered a sign of spasticity, but was interpreted as compensatory activity to improve foot clearance during the swing phase instead. Misinterpretation of compensatory rectus femoris activity as spasticity may lead to inadequate treatment with BoNT-A injections in some patients, possibly explaining the response variations found in previous literature. Additional biomechanical markers should be explored to better determine the contribution of rectus femoris spasticity in stiff knee in individual patients.

Introduction

People with upper motor neuron syndrome (e.g. due to stroke) often show reduced knee flexion during the swing phase of gait, commonly referred to as "stiff knee gait" [1-4]. Stiff knee gait impairs foot clearance of the affected leg, thereby increasing fall risk and energy costs of walking. In unaffected gait, knee flexion during the swing phase is not initiated by the knee flexors. Instead, during pre-swing, push-off from the calf muscles in combination with pull-off from the large hip flexors is responsible for an extrinsic knee flexion moment, forcing the knee into rapid flexion. The rectus femoris muscle, a biarticular muscle that crosses the hip and knee joints, regulates the degree of knee flexion during the swing phase through eccentric contraction, causing a counterbalancing intrinsic knee extension moment [5-7]. Without rectus femoris activity, excessive knee flexion would occur, whereas rectus femoris overactivity results in reduced knee flexion.

There are roughly three factors that contribute to stiff knee gait in people with upper motor neuron syndrome, either individually or in combination. First, decreased push-off at the ankle joint may lead to a lower extrinsic knee flexion moment during pre-swing, leading to a lower swing-phase knee flexion angle [1-4, 6-13]. Second, decreased pull-off at the hip joint may also cause a lower extrinsic knee flexion moment during pre-swing, resulting in less swing-phase knee flexion [4, 8-10, 12]. Third, inappropriate activity of the quadriceps femoris, including the rectus femoris, potentially causes a premature deceleration of the knee flexion by generating an intrinsic knee extension moment [1-10, 12].

The above-mentioned causal triad of stiff knee gait is not easily disentangled in individual patients, as it requires a three-dimensional (3D) instrumented gait analysis [14]. Clinical literature seems to focus on rectus femoris spasticity as the main cause of stiff knee gait. Such spasticity can be reduced by chemodenervation of the muscle, for example with botulinum toxin (BoNT-A) injections [1, 4, 8]. However, previous studies on the effect of BoNT-A injections on stiff knee gait have generated inconsistent results, with large variations between individual study participants and often rather small improvements in swing phase knee flexion angle after treatment [2-4, 8, 13, 15].

The indication for treatment with BoNT-A is typically based on the presence of reduced knee flexion during the swing phase of gait – frequently characterized by a distinctive interruption of the knee flexion kinematic curve (i.e., a "double bump shape") [13, 15] – combined with inappropriately timed rectus femoris activity measured with surface electromyography (sEMG) [13]. Notably, interpreting rectus femoris

activity during the gait cycle is not straightforward. Theoretically, rectus femoris spasticity would be triggered during pre-swing, when knee flexion angular velocity peaks. Yet, it can be difficult to label pre-swing sEMG activity as inappropriate, as in unaffected walking the rectus femoris should become active during pre-swing to decelerate knee flexion [5, 12]. Therefore, the presence of mid-swing rectus femoris activity is often used as a criterion for rectus femoris spasticity during gait [2-4, 8, 16].

The objective of this video-illustrated case series is to question the use of mid-swing rectus femoris activity as a criterion of rectus femoris spasticity, and therefore also as an indication for treatment with BoNT-A injections. This notion is substantiated based on four patients with stiff knee gait after stroke who were referred to our hospital for a clinical 3D instrumented gait analysis.

Case presentation

This video-illustrated case series presents four patients (ages 41 to 56), who were in the chronic phase after stroke (16 to 64 months) and referred to our hospital for a clinical 3D gait analysis. Figure 1 shows the knee flexion angle, rectus femoris sEMG, ankle power, hip flexion angle, and vastus lateralis sEMG for all patients separately. Gait speed varied between 0.36 m/s and 0.64 m/s.

Diagnostic assessment

All four patients (see Figure 1 and Videos (Supplementary material 1-4)) displayed a reduced peak knee flexion angle in the swing phase (13.3 – 27.5°), with the presence of a distinct interruption of knee flexion in their knee kinematic curve, the so called “double bump”. They all suffered from severe weakness of the calf muscles on the affected side (Medical Research Council (MRC) scale scores 0-2) upon clinical examination and strongly diminished or even absent push-off power at the ankle joint during walking, as assessed with force plates. Furthermore, they all had moderate weakness of the hip flexors (MRC scores 3-4). Finally, all showed mid-swing rectus femoris activity, which could be interpreted as rectus femoris spasticity, and all showed spasticity of the rectus femoris during clinical examination (Tardieu scale (V2) scores 1-3). Relevant individual results of the strength and spasticity tests are presented in Table 1.

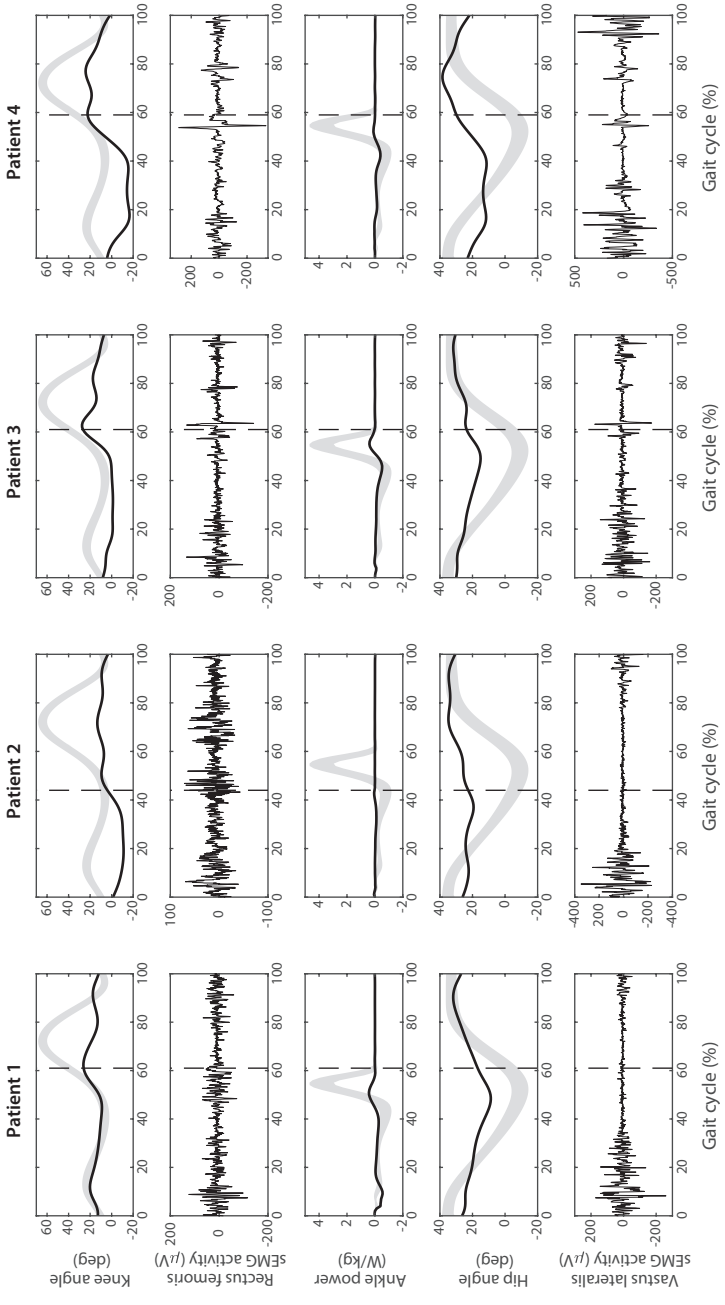


Figure 1. Gait characteristics of a full gait cycle of four cases with chronic stroke and stiff knee gait. The figure includes knee kinematics, rectus femoris sEMG activity, ankle power, hip kinematics, and vastus lateralis sEMG activity during the gait cycle (initial contact - initial contact). Norm data for kinematics and ankle power are shown in grey. The vertical dotted line indicates the transition from the stance phase to the swing phase. Please be aware of the individually scaled sEMG graphs and of possible motion artifacts in the sEMG data at pre-swing and initial swing in patients 3 and 4.

Table 1. Strength and spasticity measures from clinical physical examination.

	Patient 1	Patient 2	Patient 3	Patient 4
Muscle strength affected side (MRC 0-5)				
Hip flexors	4	4	4	3
Knee extensors	4	4	5	4
Ankle dorsiflexors	0	1	3	1
Ankle plantar flexors	1	0	2	0
Spasticity affected side (MAS 0-4)				
Gastrocnemius	0	1	1	2
Soleus	0	1	1	1
Rectus femoris	0	3	1	1
Spasticity affected side (Tardieu(V2) scale 0-4)				
Gastrocnemius	0	1	2	2
Soleus	0	2	1	4
Rectus femoris	1	3	2	1

MRC: Medical Research Council, MAS: Modified Ashworth Scale.

Clinical reasoning

Based on the presence of stiff knee gait, the clinical examination, and mid-swing rectus femoris activity, one would usually conclude that rectus femoris spasticity played a role in the stiff knee gait in these patients, and therefore proceed with spasticity treatment. However, closer examination of the rectus femoris sEMG data, aligning the timing of the mid-swing rectus femoris activity with the knee and hip kinematics, suggested an alternative explanation of the mid-swing rectus femoris activity for two reasons. First, rectus femoris activity occurred simultaneously with or prior to an acceleration in knee flexion, whereas in the case of spasticity one would expect such activity to coincide with a subsequent deceleration of knee flexion. Second, the rectus femoris activity occurred simultaneously with a flexion motion in the hip. Therefore, mid-swing rectus femoris activity may not just be inappropriately timed activity due to spasticity but could also reflect compensation through hip flexion in an effort to improve foot clearance, threatened by a severely decreased push-off.

This possible compensatory nature of rectus femoris activity is supported by the differences between barefoot walking (see Video, Supplementary material 4) versus walking with orthopedic footwear and a dorsal ankle-foot orthosis (see Video, Supplementary material 5) in patient 4. Figure 2 presents the individual's knee kinematics, rectus femoris sEMG activity, and push-off power in both conditions. When walking barefoot, the patient showed a diminished and interrupted swing-phase knee flexion, decreased push-off power, and mid-swing rectus femoris activity. When walking with orthopedic footwear and a dorsal ankle-foot orthosis, ankle push-off power improved leading to an increase in walking speed from 0.50 m/s to 0.78 m/s. Furthermore, maximum knee flexion in the swing phase improved from 24° to 36°, pre-swing knee flexion angular velocity from 239°/s to 332°/s, and the interruption in the knee flexion kinematic curve disappeared. Remarkably, the rectus femoris overactivity during mid-swing completely disappeared when walking with orthopedic footwear. In the case of rectus femoris spasticity, one would expect a higher walking speed and knee flexion angular velocity to elicit a stronger velocity-dependent stretch reflex of the rectus femoris during pre-swing. Given that the opposite occurred while walking with orthopedic footwear, the swing phase

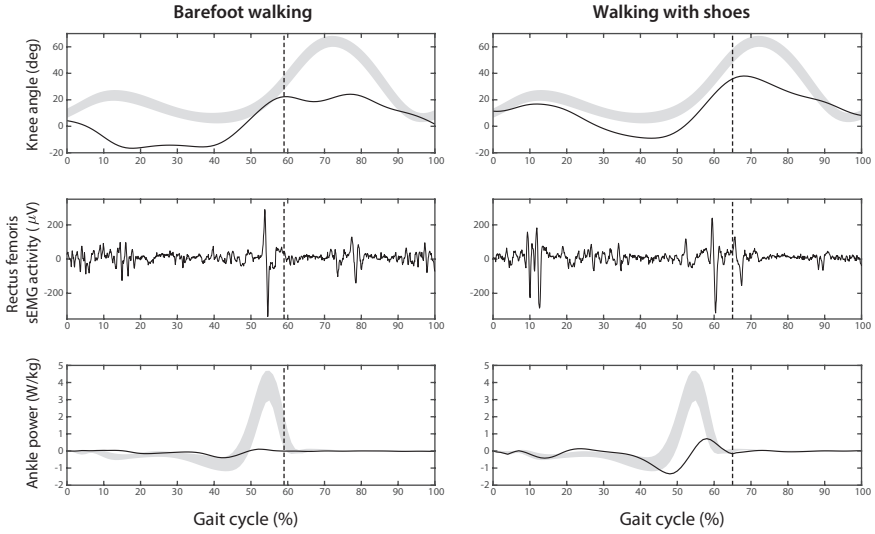


Figure 2. Knee kinematics, rectus femoris sEMG activity, and ankle power of patient 4 during the gait cycle (initial contact - initial contact) for two gait conditions: barefoot walking and walking with orthopedic footwear and a dorsal ankle-foot orthosis. Norm data for kinematics and ankle power are shown in grey. The vertical dotted line indicates the transition from the stance phase to the swing phase. Please be aware of possible motion artifacts in the sEMG data at pre-swing in both conditions.

knee flexion likely improved due to the higher push-off power. This was probably due to prior improvement of the second ankle rocker in mid-stance with the AFO-footwear combination. The mid-swing rectus femoris activity in the barefoot condition was therefore probably a compensation for the lack of ankle push-off and not an expression of spasticity. This conclusion is in line with a recent study in healthy individuals that showed that rectus femoris activity during the swing phase increased following a series of fatiguing, repetitive concentric hip flexion exercises performed until task failure [16].

Discussion

It is fair to conclude that determining the contribution of rectus femoris spasticity to stiff knee gait in people with upper motor neuron syndrome is complex and that interpretation of rectus femoris sEMG activity is difficult [17]. The observations described in this case series indicate that the timing of rectus femoris activity in relation to knee and hip kinematics, and thus the potential compensatory role of the rectus femoris, should be considered during clinical interpretation of 3D gait analysis data. This is especially relevant for those with a strongly diminished push off, e.g. due to calf weakness, abnormal second rocker, pain, abnormal structure of the ankle/foot complex, lack of selective motor control, or a combination of these factors. Furthermore, when interpreting rectus femoris activity, walking speed should be taken into account. When walking at low velocities, the need for regulation of knee flexion by the rectus femoris is much lower [18]. Misinterpretation of compensatory activity of the rectus femoris as spasticity could lead to a suboptimal treatment of stiff knee gait. Furthermore, our observations may at least partly explain the large response variations in several studies examining the effect of BoNT-A on stiff knee gait, when mid-swing rectus femoris activity is used as an inclusion criterion in these studies [2, 3, 15]. For example, one of these studies by Tenniglo et al. presented a knee kinematic curve of a patient with little treatment effect and, although ankle kinetics were not shown, this patient exhibited knee hyperextension during the stance phase, probably hampering effective push-off [3]. This patient may have had a substantially decreased push-off that could have been the primary cause of the stiff knee gait instead of rectus femoris spasticity.

Hence, there is a need for a better marker to determine the contribution of rectus femoris spasticity to stiff knee gait. Interestingly, a study by Campanini and colleagues described a promising method to determine the underlying cause of stiff knee gait based on the linear relationship between maximum knee flexion angular velocity and the vertical acceleration of the lateral malleolus during pre-swing [9]. This relationship

was assessed in healthy individuals walking at several speeds, also comparing this data with that obtained from participants with stiff knee gait in the chronic phase of stroke. Of the 52 included participants with stiff knee gait, 85% fell within the 95% confidence interval of the regression model, suggesting that decreased push-off was the primary cause of their stiff knee gait. Yet, three participants with a lower knee flexion velocity than expected based on the regression model were suspected of rectus femoris spasticity and treated with a diagnostic femoral nerve block (motor branch of rectus femoris) using a local anesthetic (lidocaine). This block resulted in varying increments in peak knee flexion of 2°, 10° and 30°, corresponding with the magnitude of their deviation from the model. A recent study by our team presented a more comprehensive model that included both ankle push-off and hip pull-off and explained a significant amount of peak knee flexion in patients after stroke [19]. Biomechanical measures of push-off and pull-off in relation to maximum knee flexion angular velocity could be useful clinical markers to better estimate the contribution of quadriceps femoris spasticity to stiff knee gait [20]. In conclusion, misinterpretation of compensatory mid-swing rectus femoris activity as spasticity could possibly lead to inadequate treatment with BoNT-A injections in some patients, and may explain response variations to BoNT-A treatment found in previous literature. Further research in this field is required to improve the diagnostic process and to better tailor treatment strategies for individuals with stiff knee gait.

References

[1] Nonnekes J, Benda N, van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[2] Tenniglo MJ, Nederhand MJ, Prinsen EC, Nene AV, Rietman JS, Buurke JH. Effect of chemodenervation of the rectus femoris muscle in adults with a stiff knee gait due to spastic paresis: A systematic review with a meta-analysis in patients with stroke. Arch Phys Med Rehabil. 2014;95(3):576-87. https://doi.org/10.1016/j.apmr.2013.11.008.

[3] Tenniglo MJ, Nene AV, Rietman JS, Buurke JH, Prinsen EC. The effect of botulinum toxin type A injection in the rectus femoris in stroke patients walking with a stiff knee gait: A randomized controlled trial. Neurorehabil Neural Repair. 2023;37(9):640-51. https://doi.org/10.1177/15459683231189712.

[4] Stoquart GG, Detrembleur C, Palumbo S, Deltombe T, Lejeune TM. Effect of botulinum toxin injection in the rectus femoris on stiff-knee gait in people with stroke: A prospective observational study. Arch Phys Med Rehabil. 2008;89(1):56-61. https://doi.org/10.1016/j.apmr.2007.08.131.

[5] Perry J. Gait analysis: Normal and pathological function. SLACK Incorporated; 1992.

[6] Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. J Biomech. 1996;29(6):723-33. https://doi.org/10.1016/0021-9290(95)00144-1.

[7] Goldberg SR, Anderson FC, Pandy MG, Delp SL. Muscles that influence knee flexion velocity in double support: Implications for stiff-knee gait. J Biomech. 2004;37(8):1189-96. https://doi.org/10.1016/j.jbiomech.2003.12.005.

[8] Goldberg SR, Õunpuu S, Delp SL. The importance of swing-phase initial conditions in stiff-knee gait. J Biomech. 2003;36(8):1111-6. https://doi.org/10.1016/s0021-9290(03)00106-4.

[9] Campanini I, Merlo A, Damiono B. A method to differentiate the causes of stiff-knee gait in stroke patients. Gait Posture. 2013;38(2):165-9. https://doi.org/10.1016/j.gaitpost.2013.05.003.

[10] Roche N, Boudarham J, Hardy A, Bonnyaud C, Bensmail B. Use of gait parameters to predict the effectiveness of botulinum toxin injection in the spastic rectus femoris muscle of stroke patients with stiff knee gait. Eur J of Phys Rehabil Med. 2015;51(4):361-70.

[11] Reinbolt JA, Fox MD, Arnold AS, Õunpuu S, Delp SL. Importance of preswing rectus femoris activity in stiff-knee gait. J Biomech. 2008;41(11):2362-9. https://doi.org/10.1016/j.jbiomech.2008.05.030.

[12] Rybar MM, Walker ER, Kuhnen HR, Ouellette DR, Berrios R, Hunter SK, et al. The stroke-related effects of hip flexion fatigue on over ground walking. Gait Posture. 2014;39(4):1103-8. https://doi.org/10.1016/j.gaitpost.2014.01.012.

[13] Caty GD, Detrembleur C, Bleyenheuft C, Deltombe T, Lejeune TM. Effect of simultaneous botulinum toxin injections into several muscles on impairment, activity, participation and quality of life among stroke patients presenting with a stiff knee gait. Stroke. 2008;39(10):2803-8. https://doi.org/10.1161/STROKEAHA.108.516153.

[14] Tenniglo MJB, Nederhand MJ, Fleuren JF, Rietman JS, Buurke JH, Prinsen EC. Does the Duncan-Ely test predict abnormal activity of the rectus femoris in stroke survivors with a stiff knee gait? J Rehabil Med. 2022;54:jrm00247. https://doi.org/10.2340/jrm.v53.637.

[15] Chantraine F, Schreiber C, Pereira JAC, Kaps J, Dierick F. Classification of stiff-knee gait kinematic severity after stroke using retrospective k-means clustering algorithm. J Clin Med. 2022;11(21):6270. https://doi.org/10.3390/jcm11216270.

[16] Knuppe AE, Bishop NA, Clark AJ, Alderink GJ, Barr KM, Miller AL. Prolonged swing phase rectus femoris activity is not associated with stiff-knee gait in children with cerebral palsy: A retrospective study of 407 limbs. Gait Posture. 2013;37(3):345-8. https://doi.org/10.1016/j.gaitpost.2012.07.034.

[17] Krajewski KT, Correa JS, Siu R, Cunningham D, Sulzer JS. Mechanisms of post-stroke stiff knee gait: A narrative review. Am J Phys Med Rehabil. 2025;104(7):e101–e108. https://doi.org/10.1097/PHM.0000000000002678.

[18] Nene A, Byrne C, Hermens H. Is rectus femoris really a part of quadriceps? Assessment of rectus femoris function during gait in able-bodied adults. Gait Posture. 2004;20(1):1-13. https://doi.org/10.1016/S0966-6362(03)00074-2.

[19] Bloks BE, Keijsers NLW, van Oorschot W, Geurts AC, Nonnekes J. Towards a precision rehabilitation approach for post-stroke stiff knee gait. Clin Biomech. 2025;127:106587. https://doi.org/10.1016/j.clinbiomech.2025.106587.
[20] Lee J, Seamon BA, Lee RK, Kautz SA, Neptune RR, Sulzer JS. Post-stroke stiff-knee gait: are there different types or different severity levels? J NeuroEngineering Rehabil. 2025;22:36. https://doi.org/10.1186/s12984-025-01582-3.

Supplementary materials

Supplementary material 1:
A 46-year-old patient with a right-sided hemiparesis after an ischemic stroke (2 years and 3 months ago). This video corresponds with the graphs presented for patient 1 in Figure 1.



Supplementary material 2:
A 51-year-old patient with a right-sided hemiparesis after an ischemic stroke (2 years ago). The patient was not able to walk barefoot without support and is therefore shown while walking with store-bought footwear. This video corresponds with the graphs presented for patient 2 in Figure 1.



Supplementary material 3:
A 56-year-old patient with a left-sided hemiparesis after a hemorrhagic stroke (1 year and 5 months ago). This video corresponds with the graphs presented for patient 3 in Figure 1.



Supplementary material 4:
A 41-year-old patient with a right-sided hemiparesis after an ischemic stroke (6 years ago). This video corresponds with the graphs presented for patient 4 in Figure 1, and the graphs presented for barefoot walking in Figure 2.



Supplementary material 5:
Patient 4, the same patient as Supplementary material 4 (41-year-old patient with a right-sided hemiparesis after an ischemic stroke (6 years ago)), walking with orthopedic shoes and a dorsal ankle-foot-orthosis. This video corresponds with the graphs presented for shoe-walking in Figure 2.



Chapter 5

Towards a precision rehabilitation approach for post-stroke stiff knee gait

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Abstract

Background

Stiff knee gait, characterized by reduced knee flexion during swing, may arise from rectus femoris spasticity or inadequate pre-swing biomechanics. Difficulty in identifying each factor's contribution complicates clinical management. This study aimed to develop a predictive model for determining the contribution of inadequate pre-swing biomechanics to stiff knee gait in people with stroke.

Methods

This historic cohort study analyzed gait data of 122 people with stroke and 20 healthy controls walking at four speeds. Linear regression analyses examined the relationship between pre-swing biomechanics and peak knee flexion in healthy controls. The pre-swing biomechanical measure explaining most of the variance in peak knee flexion was used in the predictive model, which was then applied to stroke data.

Results

Peak knee flexion angles of people with stroke were lower compared to healthy controls (stroke: 41±16°; healthy controls: 61±5°, 52±8°, 57±6°, and 61±4° for self-selected walking speed, 0.4 m/s, 0.8 m/s, and 1.2 m/s, respectively). For healthy controls, peak knee flexion variance was best explained by combined pre-swing ankle and hip work (R^2 = 0.58). For 65% of people with stroke, peak knee flexion fell above the lower bound of the regression model's prediction interval, suggesting stiff knee gait may primarily be caused by inadequate ankle push-off and hip pull-off.

Conclusion

Our predictive model holds the potential to improve treatment selection by determining the impact of inadequate pre-swing biomechanics on stiff knee gait. In many participants, peak knee flexion was explained by pre-swing biomechanics, highlighting their key role in stiff knee gait.

Introduction

Stiff knee gait is a common gait abnormality in people with stroke and is characterized by reduced peak knee flexion during the swing phase of gait [1, 2]. Stiff knee gait significantly impairs functional mobility as it hampers foot clearance, leading to an increased risk of falling and raised energy expenditure [3, 4].

Three factors are identified as potential contributors to the manifestation of stiff knee gait. First, stiff knee gait is frequently attributed to spasticity of the quadriceps muscles, in particular spasticity of the rectus femoris muscle [5-7]. Spasticity of the quadriceps muscles, elicited by rapid muscle lengthening during knee flexion in pre-swing and early swing, results in an increased intrinsic knee extension moment, decelerating knee flexion and limiting peak knee flexion. Second, decreased pre-swing push-off power at the ankle joint is recognized as a potential contributor to stiff knee gait [8, 9]. Ankle push-off power enables a rapid knee flexion movement during pre-swing. Accordingly, reduced ankle push-off power results in diminished knee flexion velocity at toe-off, contributing to reduced peak knee flexion. Third, decreased pull-off power at the hip joint may also play a role in the occurrence of stiff knee gait [10, 11]. Hip pull-off power facilitates knee flexion during (pre-)swing. Consequently, decreased hip pull-off power may impede knee flexion velocity at toe-off and peak knee flexion during swing.

In daily clinical practice, the importance of adequate pre-swing ankle push-off and hip pull-off power for achieving sufficient knee flexion during swing is often undervalued, as stiff knee gait is commonly intuitively linked to spasticity of the rectus femoris muscle [6, 12]. Management of rectus femoris spasticity typically involves chemodenervation, such as intramuscular botulinum toxin injections [12-16]. However, botulinum toxin treatment effects exhibit substantial between-subjects variability [13, 17], potentially due to the intricate interpretation of surface electromyography (sEMG) data [18], the primary technique for assessing spasticity during gait. Rectus femoris spasticity is typically triggered by a velocity-dependent change in muscle length during pre-swing [2]. However, in unaffected gait, the rectus femoris muscle also becomes active during pre-swing to decelerate knee flexion [2, 19], complicating spasticity identification with sEMG and increasing the risk of misdiagnosis. This may partly explain the large variation in treatment responses [18]. In individuals with very low walking speeds, a rapid change in muscle length during pre-swing is absent, making it unlikely that rectus femoris spasticity contributes significantly to stiff knee gait. Consequently, in this low performing stroke population, the focus should shift from quadriceps spasticity to inadequate pre-swing biomechanics when examining stiff knee gait. In contrast, in people with stroke who

exhibit relatively high walking speeds, distinguishing between inadequate pre-swing biomechanics and quadriceps spasticity as the primary cause of stiff knee gait remains challenging. To enhance treatment selection, the field of post-stroke gait rehabilitation needs a precision rehabilitation approach to examine the underlying factors causing stiff knee gait [2].

Campanini and colleagues previously introduced a predictive model to assess the influence of inadequate ankle push-off on the occurrence of stiff knee gait [9]. In this model, peak vertical acceleration of the malleolus during pre-swing is used as a predictor of knee flexion velocity at toe-off. However, although knee flexion velocity at toe-off influences peak knee flexion during swing, the direct relationship between the vertical acceleration of the malleolus during pre-swing and peak knee flexion during swing is still unknown. Moreover, hip pull-off is not taken into account in this model, while people post-stroke often compensate for reduced ankle push-off by generating more hip pull-off [20], which contributes to knee flexion during swing [10, 11].

The aim of this study was to introduce a predictive model for determining the contribution of inadequate pre-swing biomechanics to post-stroke stiff knee gait on an individual level. To this aim, gait data of healthy subjects walking at four different gait speeds and a large cohort of people with stroke were analyzed. Based on the common occurrence of both quadriceps spasticity and muscle weakness around the ankle and hip joints in individuals post stroke [21], we hypothesized that people with stroke would exhibit reduced average peak knee flexion during swing compared to healthy controls, when taking into account gait speed. Additionally, we hypothesized that ankle push-off would serve as a predictor of peak knee flexion during swing in healthy subjects, and that this predictive relationship could be enhanced by integrating hip pull-off. Finally, based on the prevalence of spasticity in people with stroke [22], we hypothesized that in approximately one third of the individuals post stroke, reduced peak knee flexion could not be sufficiently explained by diminished ankle push-off and/or hip pull-off, resulting in a peak knee flexion angle falling significantly below the 95% prediction interval of the regression model.

Methods

Participants and design

In this historic cohort study, three-dimensional gait data of adults with unilateral stroke and of healthy controls were analyzed. All people with stroke who underwent a three-dimensional instrumented gait analysis during their outpatient clinic visit of

the Gait Expertise Center at the Sint Maartenskliniek between January 2019 and July 2023 were eligible. Exclusion criteria for people with stroke were: 1) comorbidities that might cause gait impairments, 2) inability to accurately assess gait kinetics, e.g. due to the use of a walking aid or orthosis, and 3) a walking speed below 0.4 m/s, as spasticity resulting from a velocity-dependent change in muscle length was deemed unlikely in such cases. Gait data of 20 healthy controls were previously collected with the purpose of serving as reference data for our gait laboratory studies. Exclusion criteria for healthy controls were: 1) any gait impairment and 2) previous lower body surgery. This study was conducted in accordance with the local ethical guidelines.

Experiments

All data were collected at the gait laboratory of the Sint Maartenskliniek. This lab is equipped with a ten-camera motion capture system (Vicon Motion Systems Ltd., Oxford, UK) and two force plates (Kistler, Winterthur, Switzerland) embedded in the middle the walkway. Reflective markers were placed according to the Plug-in Gait lower body model (Vicon Motion Systems Ltd., Oxford, UK) and recorded at a sample frequency of 100 Hz. Force plate data were recorded at a sample frequency of 1000 Hz.

Twenty healthy controls walked at their preferred walking speed. Subsequently, subjects were instructed to walk at three predefined walking speeds in random order: 0.4 m/s, 0.8 m/s, and 1.2 m/s. Walking speed was evaluated real-time based on the position of a marker placed on the 7th cervical vertebra (C7). Feedback on walking speed was provided to the subject after each walking trial, with a correct walking speed defined as one that did not deviate more than 0.05 m/s from the predefined walking speed. The analysis incorporated right-limb data from four correct walking trials (i.e. with proper speed and force plate contact) for each walking speed condition.

People with stroke underwent an instrumented gait analysis while walking at their preferred walking speed. Only subjects with at least two successful walking trials (i.e. with proper force plate contact) were included in the analysis, using data of their paretic limb.

Outcomes

Kinematics and kinetics were calculated according to the Plug-in Gait lower body model (Vicon Motion Systems Ltd., Oxford, UK) with the use of Vicon Nexus (Vicon Motion Systems Ltd., Oxford, UK) and further processed using MATLAB (Version R2021a, The MathWorks, Inc., Natick, MA, USA). Gait events, specifically toe-off and initial contact, were manually entered into Vicon Nexus by experienced Vicon Nexus users, using force plate data as a reference. The swing phase was defined as the phase between ipsilateral toe-off and subsequent ipsilateral initial contact. Peak knee

flexion angles during the swing phase were derived from the kinematics. Walking speed was calculated by dividing the distance traveled by the heel marker by the elapsed time. Pre-swing was defined as the phase between contralateral initial contact and ipsilateral toe-off. Seven pre-swing biomechanical measures were calculated to serve as predictors of the peak knee flexion angle during swing: peak vertical acceleration of the lateral malleolus during pre-swing, peak ankle angular velocity during pre-swing, propulsive impulse during pre-swing (area under the curve of the anteriorly oriented ground reaction force), peak of the anteriorly oriented ground reaction force during pre-swing, peak ankle power during pre-swing, ankle work during pre-swing, and hip work during pre-swing. The selection of these pre-swing biomechanical measures was based on their potential for showing a relationship with peak knee flexion during swing, as supported by literature demonstrating associations between peak knee flexion and the anterior-posterior ground reaction force [23], ankle push-off [9], and hip pull-off [10, 11]. Furthermore, clinical applicability was considered by also opting for measures solely based on kinematics (including peak malleolus vertical acceleration and peak ankle angular velocity), thereby eliminating the requirement of a force plate. All kinematic and kinetic outcome parameters were averaged across walking trials.

Data analyses

A four-pronged approach was followed to develop the predictive model. First, data exploration was conducted by examining the relationship between peak knee flexion angles during the swing phase and walking speed in people with stroke and in healthy controls.

Second, single linear regression analyses were conducted on the healthy control data to determine the relationship between each of the seven pre-swing biomechanical measures and swing phase peak knee flexion. Subsequently, a multiple linear regression analysis was performed to investigate the correlation between peak knee flexion during swing and the combined effects of ankle push-off and hip pull-off. To ensure clinical applicability and mitigate multicollinearity, it was decided to include only two independent variables in the multiple regression analysis. The selection of the independent variable related to ankle push-off was based on the outcomes of the single linear regression analyses, specifically opting for the most predictive measure. For the multiple regression analysis, the independent variables pertaining to ankle push-off and hip pull-off were normalized using min-max scaling, according to: $Xnorm = (X - Xmin)/(Xmax - Xmin)$. For all single and multiple linear regression models, the coefficients of determination (R^2) were calculated as measures of goodness of fit. In addition, 95% prediction intervals of the regression models were computed, representing the range within which future observations of healthy gait

are expected to fall. Additionally, to evaluate whether more complex models provide greater explanatory power than the single and multiple linear regression models described above, forward stepwise regression was performed using all seven pre-swing biomechanical measures as used in the single and multiple linear regression analyses. Pre-swing biomechanical measures were included as predictors in the model if their p-value was below 0.05. The explanatory power of the resulting model was evaluated using the R^2 value.

Third, following the outcomes of the linear regression analyses, the pre-swing biomechanical measure or combination of measures that explained the highest proportion of variance in peak knee flexion during swing (indicated by the highest R^2 value) was considered as the most predictive. Data of healthy controls and of people with stroke were then compared regarding this most predictive biomechanical measure or combination of measures. Individuals with stroke who had a lower value for this most predictive biomechanical measure or combination of measures than the average value of healthy controls minus two times the standard deviation were excluded from comparison with the final regression model and its corresponding 95% prediction interval. This decision was made to avoid extrapolation of the regression model, as the regression equation may not be valid outside the scope of the model.

Fourth, data of people with stroke who had a pre-swing biomechanical measure or combination of measures falling within the reference range of healthy controls were compared to the regression model and the corresponding 95% prediction interval.

Results

A database consisting of gait data of 225 adults with unilateral stroke was screened for eligibility. People were excluded because of a lack of accurate kinetic measurements (e.g. due to the use of a walking aid or orthosis, n = 79), a walking speed below 0.4 m/s (n = 23), or comorbidities that might cause gait impairments (n = 1). This resulted in 122 people with stroke that were included. Table 1 presents the subject characteristics of people with stroke and healthy controls.

Peak knee flexion during swing and walking speed

Figure 1 shows the peak knee flexion angle during swing versus walking speed of individuals with stroke and of healthy controls. The average peak knee flexion angle during swing of people with stroke was 41° with a standard deviation of 16°. For healthy controls walking at their self-selected walking speed, the average peak knee

flexion angle during swing was 61±5°, whereas the imposed walking speeds showed an average peak knee flexion angle during swing of 52±8°, 57±6°, and 61±4° for the conditions 0.4 m/s, 0.8m/s, and 1.2 m/s, respectively.

Table 1. Subject characteristics.

	Stroke	Healthy controls
n (people)	122	20
Sex (#female/#male)	60/62	12/8
Age (years, mean±SD)	53.1±13.0	62.2±10.4
Mass (kg, mean±SD)	79.1±14.0	77.2±8.5
Height (cm, mean±SD)	173.5±8.5	173.3±7.7
Paretic side (#left/#right)	60/62	-

SD: standard deviation.

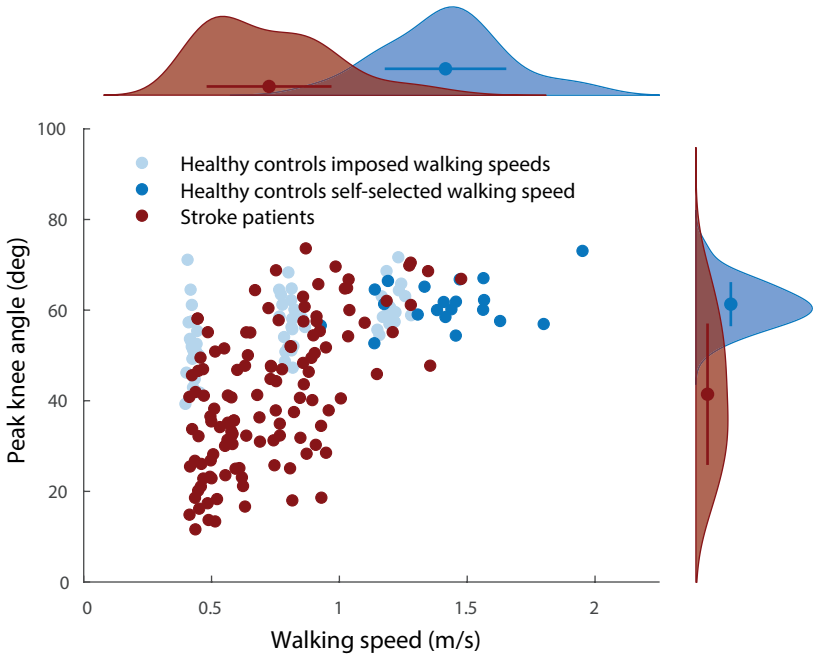


Figure 1. Walking speed versus peak knee angle during swing of healthy controls and individuals with stroke. On top and on the right side of the figure, the distributions and means and standard deviations are presented for people with stroke and healthy controls walking at their self-selected walking speed.

The average preferred walking speed of people with stroke was 0.72±0.24 m/s. For healthy controls walking at their self-selected walking speed, the average speed was 1.42±0.24 m/s. The average walking speeds of healthy controls walking at the imposed speeds were 0.43±0.02 m/s, 0.81±0.03 m/s, and 1.20±0.03 m/s for the conditions 0.4 m/s, 0.8 m/s, and 1.2 m/s, respectively.

Regression analyses pre-swing biomechanics and peak knee flexion during swing

The linear relationships between the pre-swing biomechanical measures and the peak knee flexion angle during swing, along with their goodness of fit, as indicated by the R^2 values, are provided in Figure 2. Among the ankle-related biomechanical measures, ankle work emerged as the most predictive factor for peak knee flexion during swing (R^2 = 0.50). The combination of the work generated around the ankle and hip joint explained most of the variance in peak knee flexion angle during swing, as indicated by an R^2 value of 0.58. Forward stepwise regression analysis showed that more complex models did not improve exploratory power, as this analysis identified a final model that included ankle work ($p<0.001$) and hip work ($p<0.001$), with an R^2 value of 0.57. Therefore, the combination of work generated around the ankle and hip joints was used to generate the final predictive model. The correlation between peak knee flexion during swing and ankle and hip work during pre-swing is described by the following formula:

$$PKFA = 25.1 * A + 15.5 * H - 15.9 * AH + 42.8$$

Where *PKFA = peak knee flexion angle during swing, A= normalized ankle work, H = normalized hip work,* and *AH=normalized ankle work*normalized hip work*. Normalization of ankle and hip work was carried out using min-max scaling: *Xnorm = (X - Xmin)/(Xmax - Xmin)*, with *Xmin ankle work* = 0.07 Nm/kg, *Xmax ankle work* = 0.44 Nm/kg, *Xmin hip work* = 0.01 Nm/kg, and *Xmax hip work* = 0.16 Nm/kg, based on healthy control data.

In Figure 3, the work generated around the ankle and hip joint during pre-swing for healthy controls and individuals with stroke is shown. During pre-swing in individuals with stroke, the average work generated around the ankle and hip joints was 0.10±0.07 Nm/kg and 0.05±0.04 Nm/kg, respectively. In comparison, for healthy controls walking at their self-selected speed, the average ankle and hip work during pre-swing was 0.28±0.08 Nm/kg and 0.12±0.02 Nm/kg, respectively.

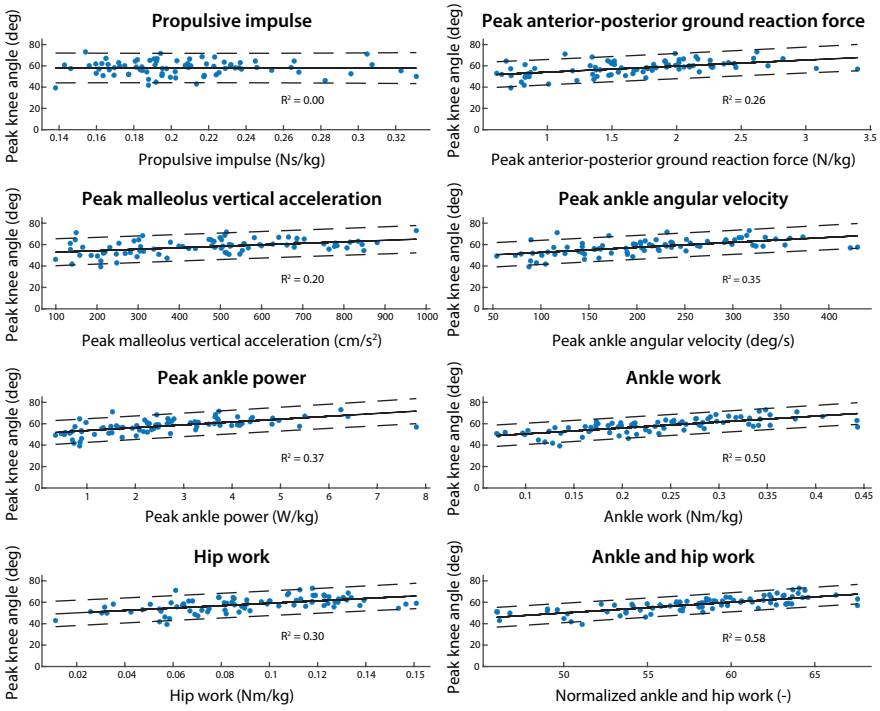


Figure 2. Relationships between pre-swing biomechanical measures and peak knee flexion angle during swing for healthy controls. Goodness of fit of the regression models are indicated by the R-squared (R^2) values, while the dotted lines represent the 95% prediction intervals. All panels depict single linear regression analyses, except the bottom-right panel, which illustrates a two-dimensional representation of a multiple linear regression analysis. In this panel, the predicted value for peak knee flexion, calculated using combined normalized ankle and hip work based on the regression model (25.1*A + 15.5*H – 15.9*AH + 42.8) is placed on the x-axis.

For 60 out of 122 people with stroke, the combined normalized ankle and hip work, calculated according to the regression model (25.1*A + 15.5*H – 15.9*AH + 42.8), was lower than the average combined normalized ankle and hip work of healthy controls minus two times the standard deviation (47.5, see Figure 3: right panel). Therefore, the remaining 62 individuals with stroke with a combined normalized ankle and hip work falling within the reference range of healthy controls were compared to the final predictive model and its 95% prediction interval.

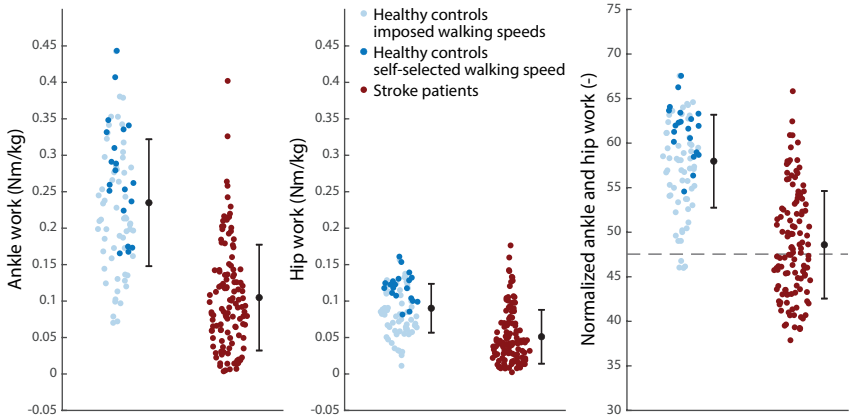


Figure 3. Ankle and hip work during pre-swing for healthy controls and individuals with stroke. The work generated around the ankle (left panel) and hip (middle panel) joints during pre-swing is depicted for individuals with stroke (red) and healthy controls (blue), along with the average values and standard deviations. In the right panel, the combined normalized ankle and hip work during pre-swing is depicted, according to the regression model (25.1*A + 15.5*H – 15.9*AH + 42.8, where A = normalized ankle work, H = normalized hip work, and AH = normalized ankle work * normalized hip work). Average values and standard deviations are also displayed. Additionally, the dotted line represents the average value of healthy controls minus two times the standard deviation, which is used as a threshold for excluding participants with stroke from further analysis. Individual data points are jittered along the x-axis to prevent overlap.

A two-dimensional representation of the final multiple regression model is shown in Figure 4, including data of 62 individuals with stroke. For 22 out of 62 people, the peak knee flexion angle during swing fell below the lower bound of the 95% prediction interval of the regression model. For these 22 people, the average difference in peak knee flexion angle from the regression line of the predictive model was 17±5°.

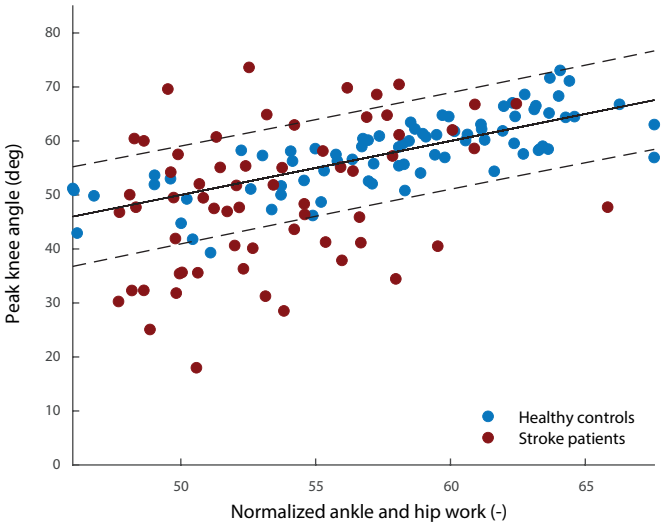


Figure 4. Final predictive model. Two-dimensional representation of the relationship between peak knee flexion angle during swing and normalized ankle and hip work during pre-swing for healthy controls and people with stroke. The x-axis represents the predicted value for peak knee flexion, calculated using combined normalized ankle and hip work based on the regression model: 25.1*A + 15.5*H – 15.9*AH + 42.8. The dotted lines indicate the upper and lower bounds of the 95% prediction interval.

Discussion

This study presents a predictive model for determining the contribution of ankle push-off and hip pull-off to the occurrence of post-stroke stiff knee gait. The model uses combined ankle and hip work during pre-swing as predictor of peak knee flexion during swing. Our study demonstrates that this combined ankle and hip work during pre-swing accounts for a substantial proportion (58%) of the variance in peak knee flexion in healthy controls. When applying our predictive model to individuals with stroke, the actual measured peak knee flexion angle during swing fell above the lower bound of the 95% prediction interval in 40 out of 62 cases (65%), indicating agreement with the predicted peak knee flexion angle based on ankle push-off and hip pull-off. This suggests that, in these people, stiff knee gait may primarily be attributed to insufficient pre-swing biomechanics. Hence, this study offers a comprehensive framework for individualized clinical assessment of post-stroke stiff knee gait, providing indications for tailored treatment strategies.

The observed peak knee flexion angles in healthy controls in this study ranged from 39 to 73° across all walking speeds, which is in line with previous research [24]. Individuals with stroke had reduced swing phase peak knee flexion angles compared to healthy controls. This underscores the high prevalence of diminished knee flexion in individuals with stroke [1], which may contribute to foot clearance problems, thereby increasing the risk of falling and fall-related injuries. Hence, once validated, the predictive model presented in this study may have substantial impact on healthcare through improved treatment of post-stroke stiff knee gait.

In line with our hypothesis and with previous research [10, 11], the combined ankle push-off and hip pull-off work emerged as the most predictive factor for the variability in swing phase peak knee flexion in healthy controls, accounting for 58% of the variance. Forward stepwise regression analysis revealed that adding more independent variables did not improve the explanatory power of the model. The final regression model demonstrates a considerable influence of ankle and hip work during pre-swing on peak knee flexion during swing, which is reflected by the regression line that depicts peak knee flexion angles ranging notably from 46 to 68° in response to alterations in ankle and hip work. Hence, this suggests that the combined ankle push-off and hip pull-off work may serve as a valuable predictor of peak knee flexion during swing, particularly when using the 95% prediction interval to detect abnormalities.

When comparing individuals with stroke to our predictive model, 22 out of 122 people (18%) that were included in the study showed a peak knee flexion angle during swing that fell below the lower bound of the 95% prediction interval of the regression model. This suggests that, in these people, stiff knee gait may not be sufficiently explained by inadequate pre-swing biomechanics, implying that additional contributing factors are likely involved. In such cases, quadriceps spasticity or stiffness is most probably an important contributing factor. Accordingly, spasticity may be present in approximately 18% of participants in this study. This is less than hypothesized, which can potentially be attributed to the challenge of formulating an accurate hypothesis due to the limited prevalence data on quadriceps spasticity in individuals with stroke [22]. Nevertheless, as the model is not yet validated, this would be the required next step before definitive conclusions can be drawn regarding the contribution of quadriceps spasticity to stiff knee gait. Likewise, although this does not reflect stiff knee gait, future research should investigate the underlying cause of the excessive knee flexion as observed in individuals whose peak knee flexion fell above the upper bound of the 95% prediction interval.

In cases where spasticity of the rectus femoris muscle contributes to stiff knee gait, treatment focused on the reduction of spasticity, e.g. chemodenervation, would be beneficial. We argued that the complex interpretation of sEMG could be a reason for the large variations in treatment effects of chemodenervation as described in the literature [18]. For instance, a systematic review on chemodenervation of the rectus femoris muscle reported an average increase in peak knee flexion angle during swing varying between 1.9 and 15.4° across studies [17]. Moreover, the peak knee flexion angle after intervention remained on average lower than 36° [17], whereas normative values of peak knee flexion for healthy controls typically vary between 50 and 65° at walking velocities ranging from 0.40 to 1.60 m/s [24]. Hence, the large variation in treatment effects observed after interventions targeting quadriceps spasticity may indicate an overvaluation of the contribution of rectus femoris spasticity to stiff knee gait. Furthermore, the current study demonstrates that ankle push-off and hip pull-off can explain peak knee flexion angles in nearly two-thirds of individuals with stroke. We therefore recommend to primarily consider inadequate pre-swing biomechanics as cause of limited peak knee flexion during swing, rather than focusing on quadriceps spasticity.

Campanini and colleagues previously introduced a model in which the peak vertical acceleration of the malleolus during pre-swing is used as a predictor of knee flexion velocity at toe-off [9]. However, the current study reveals that the direct relationship between the peak vertical acceleration of the malleolus during pre-swing and peak knee flexion during swing is modest ($R^2 = 0.20$). Moreover, Campanini's model did not account for hip pull-off, while the current study shows an enhanced relationship between pre-swing biomechanics and peak knee flexion during swing when also including hip pull-off work in the regression model ($R^2 = 0.58$ for ankle and hip work, compared to $R^2 = 0.50$ for ankle work).

A limitation of the current study is that our model has not yet been validated. An important next step, essential for the integration of our model into clinical practice, involves assessing its validity through examination of its positive and negative predictive value in a new, prospective sample of people with stroke. The validation of our model could be accomplished by predicting treatment effects of interventions focusing on reducing quadriceps spasticity. In cases where muscle spasticity is identified as potential contributor to stiff knee gait, our model holds potential for predicting treatment effects. The extent to which muscle spasticity contributes to stiff knee gait varies among individuals with stroke and can be estimated based on the deviation in peak knee flexion from the regression line of the predictive model. Larger deviations suggest a higher potential for improvement through interventions targeting spasticity reduction. The predicted treatment

effects could subsequently be validated by clinical trials in which participants receive both intramuscular botulinum toxin and placebo injections in a randomized order. Before and after the injections, an instrumented gait analysis, examining kinematic and kinetic outcomes, should be performed. The performance of the predictive model can then be evaluated by comparing the observed change in swing phase knee flexion (in response to botulinum toxin and placebo) with the predicted treatment effects from our model.

Another limitation of this study pertains to the focus of the predictive model solely on the pre-swing phase, rather than encompassing the entire push-off phase, including the terminal stance phase. By excluding the terminal stance phase from our analyses, the positive work generated during this phase was not considered, while previous research has highlighted the importance of ankle power and work during the terminal stance and pre-swing phases for initiating knee flexion [25]. However, expanding our analysis to include the entire push-off phase resulted in a comparable relationship of combined ankle and hip work with peak knee flexion during the swing phase in healthy controls (R^2 = 0.59 for ankle and hip work during the push-off phase, compared to R^2 = 0.58 for ankle and hip work during the pre-swing phase alone). Moreover, incorporating the entire push-off phase presents challenges in the clinical applicability of the predictive model, since detecting the start of this phase (i.e. the moment of heel rise) in individuals with stroke can be difficult, as some people may lack heel contact with the ground surface during midstance.

A third possible limitation of our study is that individuals with stroke with either a very low walking speed (<0.4 m/s) or low combined ankle and hip work during pre-swing were not compared to our predictive model. However, we consider it to be unlikely that quadriceps spasticity would be a significant cause of stiff knee gait in this low performing stroke population, as the rapid knee flexion movement that typically triggers spasticity is absent in these individuals. This notion should also be validated by future clinical trials. Additionally, people who used a walking aid or orthosis were excluded from our study due to the inability to accurately assess gait kinetics in this group. It is recommended for future research to focus on expanding the proposed predictive model to enable its application to individuals using walking aids, thereby enhancing clinical applicability.

Conclusion

We introduced a predictive model aimed at assessing the impact of ankle push-off and hip pull-off on the occurrence of stiff knee gait in people with unilateral stroke. The findings of this study indicate that in many participants with stroke, peak knee flexion could be explained by ankle push-off and hip pull-off. This underscores that

inadequate pre-swing biomechanics is an important factor to consider in the evaluation stiff knee gait, and that treatment strategies should be tailored accordingly. In order to use our model in clinical practice, further research is needed to study its predictive validity in a new, prospective sample of individuals with stroke.

References

[1] Balaban B, Tok F. Gait disturbances in patients with stroke. PM&R. 2014;6(7):635-42. https://doi.org/10.1016/j.pmrj.2013.12.017.

[2] Krajewski KT, Correa JS, Siu R, Cunningham D, Sulzer JS. Mechanisms of post-stroke stiff knee gait: A narrative review. Am J Phys Med Rehabil. 2025;104(7):e101-e8. https://doi.org/10.1097/PHM.0000000000002678.

[3] Lewek MD, Osborn AJ, Wutzke CJ. The influence of mechanically and physiologically imposed stiff-knee gait patterns on the energy cost of walking. Arch Phys Med Rehabil. 2012;93(1):123-8. https://doi.org/10.1016/j.apmr.2011.08.019.

[4] Waters RL, Mulroy S. The energy expenditure of normal and pathologic gait. Gait Posture. 1999;9(3):207-31. https://doi.org/10.1016/s0966-6362(99)00009-0.

[5] Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. J Biomech. 1996;29(6):723-33. https://doi.org/10.1016/0021-9290(95)00144-1.

[6] Perry J. Distal rectus femoris transfer. Dev Med Child Neurol. 1987;29(2):153-8. https://doi.org/10.1111/j.1469-8749.1987.tb02130.x.

[7] Riley PO, Kerrigan DC. Torque action of two-joint muscles in the swing period of stiff-legged gait: a forward dynamic model analysis. J Biomech. 1998;31(9):835-40. https://doi/org/10.1016/s0021-9290(98)00107-9.

[8] Apti A, Akalan NE, Kuchimov S, Özdinçler AR, Temelli Y, Nene A. Plantar flexor muscle weakness may cause stiff-knee gait. Gait Posture. 2016;46:201-7. https://doi.org/10.1016/j.gaitpost.2016.03.010.

[9] Campanini I, Merlo A, Damiano B. A method to differentiate the causes of stiff-knee gait in stroke patients. Gait Posture. 2013;38(2):165-9. https://doi.org/10.1016/j.gaitpost.2013.05.003.

[10] Goldberg SR, Anderson FC, Pandy MG, Delp SL. Muscles that influence knee flexion velocity in double support: Implications for stiff-knee gait. J Biomech. 2004;37(8):1189-96. https://doi.org/10.1016/j.jbiomech.2003.12.005.

[11] Fox MD, Delp SL. Contributions of muscles and passive dynamics to swing initiation over a range of walking speeds. J Biomech. 2010;43(8):1450-5. https://doi.org/10.1016/j.jbiomech.2010.02.009.

[12] Robertson JV, Pradon D, Bensmail D, Fermanian C, Bussel B, Roche N. Relevance of botulinum toxin injection and nerve block of rectus femoris to kinematic and functional parameters of stiff knee gait in hemiplegic adults. Gait Posture. 2009;29(1):108-12. https://doi.org/10.1016/j.gaitpost.2008.07.005.

[13] Tenniglo MJB, Nene AV, Rietman JS, Buurke JH, Prinsen EC. The effect of botulinum toxin type A injection in the rectus femoris in stroke patients walking with a stiff knee gait: A randomized controlled trial. Neurorehabil Neural Repair. 2023;37(9):640-51. https://doi.org/10.1177/15459683231189712.

[14] Stoquart GG, Detrembleur C, Palumbo S, Deltombe T, Lejeune TM. Effect of botulinum toxin injection in the rectus femoris on stiff-knee gait in people with stroke: A prospective observational study. Arch Phys Med Rehabil. 2008;89(1):56-61. https://doi.org/10.1016/j.apmr.2007.08.131.

[15] Caty GD, Detrembleur C, Bleyenheuft C, Deltombe T, Lejeune TM. Effect of simultaneous botulinum toxin injections into several muscles on impairment, activity, participation, and quality of life among stroke patients presenting with a stiff knee gait. Stroke. 2008;39(10):2803-8. https://doi.org/10.1161/STROKEAHA.108.516153.

[16] Tok F, Balaban B, Yasar E, Alaca R, Tan AK. The effects of onabotulinum toxin A injection into rectus femoris muscle in hemiplegic stroke patients with stiff-knee gait: A placebo-controlled, nonrandomized trial. Am J Phys Med Rehabil. 2012;91(4):321-6. https://doi.org/10.1097/PHM.0b013e3182465feb.

[17] Tenniglo MJ, Nederhand MJ, Prinsen EC, Nene AV, Rietman JS, Buurke JH. Effect of chemodenervation of the rectus femoris muscle in adults with a stiff knee gait due to spastic paresis: A systematic review with a meta-analysis in patients with stroke. Arch Phys Med Rehabil. 2014;95(3):576-87. https://doi.org/10.1016/j.apmr.2013.11.008.

[18] van Oorschot W, Bloks B, Kamphuis J, Pieterse A, Geurts A, Keijsers N, et al. Stiff knee gait after stroke: The potential compensatory role of mid-swing rectus femoris activity. Case Rep Neurol Med. 2025;2025(1):7703081. https://doi.org/10.1155/crnm/7703081.

[19] Perry J. Gait analysis: normal and pathological function. SLACK Incorporated; 1992.

[20] Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clin Biomech. 1999;14(2):125-35. https://doi.org/10.1016/s0268-0033(98)00062-x.
[21] Nonnekes J, Benda N, Van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.
[22] Martin A, Abogunrin S, Kurth H, Dinet J. Epidemiological, humanistic, and economic burden of illness of lower limb spasticity in adults: A systematic review. Neuropsychiatr Dis Treat. 2014;10:111-22. https://doi.org/10.2147/NDT.S53913.
[23] Brough LG, Kautz SA, Neptune RR. Muscle contributions to pre-swing biomechanical tasks influence swing leg mechanics in individuals post-stroke during walking. J Neuroeng Rehabil. 2022;19(1):55. https://doi.org/10.1186/s12984-022-01029-z.
[24] Mentiplay BF, Banky M, Clark RA, Kahn MB, Williams G. Lower limb angular velocity during walking at various speeds. Gait Posture. 2018;65:190-6. https://doi.org/10.1016/j.gaitpost.2018.06.162.
[25] Kerrigan DC, Gronley J, Perry J. Stiff-legged gait in spastic paresis. A study of quadriceps and hamstrings muscle activity. Am J Phys Med Rehabil. 1991;70(6):294-300.



Part II

Treatment of pes equinovarus



Chapter 6

Surgical correction of pes equinovarus in people with stroke: Effects on personalized goal attainment and balance and gait capacity

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Abstract

Background

The aim of this study was to investigate the effect of surgical correction of pes equinovarus on personalized goal attainment and balance and gait capacity.

Methods

In this prospective observational cohort study, outcome measures were assessed before and 12 months after surgery. For a subgroup of participants, a 4-month baseline period with repeated assessments was used. Adults with pes equinovarus deformity following chronic stroke were recruited between 2019 and 2023 at a tertiary referral center. All participants underwent reconstructive surgery for pes equinovarus, with tarsal arthrodesis as the principal procedure, often combined with Achilles tendon lengthening. Personalized goal attainment, balance capacity, and gait capacity were assessed with the Canadian Occupational Performance Measure (COPM), the Mini-Balance Evaluation Systems Test (Mini-BESTest), and barefoot three-dimensional instrumented gait analysis, respectively.

Results

Forty-six participants were included. No changes in COPM, Mini-BESTest, and gait speed were observed during the 4-month baseline period. After surgery, significant improvements were found in personalized goal attainment, with COPM-performance increasing by 3.5±1.7 points ($p<0.001$) and COPM-satisfaction increasing by 3.9±1.8 points ($p<0.001$). Balance capacity also showed significant improvement after surgery (Mini-BESTest: +5.1±5.4, $p<0.001$). The vast majority of participants who were unable to walk barefoot before surgery were able to do so post surgery (19/24 participants). Furthermore, improvements in barefoot gait speed after surgery were found in participants with low pre-surgical gait speeds.

Conclusion

Improvements in personalized goal attainment and balance and gait capacity after reconstructive surgery for pes equinovarus were clinically relevant. Therefore, reconstructive surgery for pes equinovarus, with tarsal arthrodesis as the principal procedure, can be considered as a valuable treatment in people with chronic stroke. Our findings provide guidance for optimal pre-operative counseling.

Introduction

Pes equinovarus is a common ankle-foot deformity in individuals with stroke [1-3]. It arises from an imbalance in muscle strength and activity around the hindfoot and ankle, typically caused by overactivity of the plantar flexors and inverters, along with weakness of the dorsiflexors and evertors [4]. As a result of pes equinovarus, people often experience impaired gait and balance capacity, with the severity and specific symptoms varying widely among individuals. Common symptoms include pain, instability during loading, and reliance on assistive devices, which are often especially pronounced during barefoot walking [4, 5].

Treatment of pes equinovarus remains challenging, but surgical interventions can restore a plantigrade ankle-foot position [4, 6, 7]. Specifically, tarsal arthrodesis, when necessary combined with a form of Achilles tendon lengthening, can correct pes equinovarus [4, 8]. However, despite the clinical experience that surgical correction often yields positive results, scientific evidence supporting its effectiveness remains limited [4, 5]. Previous studies revealed a clinically relevant increase in gait speed after reconstructive surgery for pes equinovarus in people with stroke [9-11]. However, further validation is necessary because of the relatively small sample size of previous research and the potential influence of nonspecific time effects. Moreover, the effect of the surgical intervention on personalized goals has yet to be examined [5, 6, 9], which is crucial for optimal counseling in clinical practice [12]. Furthermore, in contrast to gait improvement, the impact of surgical correction on balance capacity has not yet been investigated [13], despite the clinical experience indicating that pes equinovarus frequently causes reduced postural control.

The aim of this study was to investigate the effect of surgical correction of pes equinovarus in individuals with stroke, focusing primarily on personalized goal attainment and secondarily on balance and gait capacity. We hypothesized that surgery would improve the attainment of personalized goals and enhance balance and gait capacity.

Methods

Participants and design

A prospective observational study was conducted in adults with chronic stroke (≥6 months post-onset). Exclusion criteria were as follows: (1) age <18 years at the onset of stroke; (2) any other disorder that affects gait; and (3) previous ankle/foot surgery in the past two years. Between 2019 and 2023, all 62 individuals scheduled

for surgery were invited to participate in the study by their rehabilitation physician during visits to the Gait Expertise Center of the Sint Maartenskliniek and Radboud university medical center. Initially, the study comprised single baseline and post-surgery assessments ("single baseline subgroup"). In a later stage, a second baseline assessment ("double baseline subgroup") was introduced to examine possible time-based changes in outcomes unrelated to the intervention. This assessment was conducted approximately four months after the first baseline assessment, coinciding with the waiting period for surgery. The original target was to include 22 participants in the double baseline subgroup. However, recruitment progressed more slowly than anticipated, as many people were unwilling to participate in the double baseline subgroup because of the burden of the additional assessment. After a preliminary analysis, participant recruitment was discontinued in 2023, as the combined sample size of the single and double baseline subgroups was deemed sufficient to meet the study objectives. The final study protocol was approved by the Medical Ethical Committee Oost-Nederland (NL77992.091.21) and registered in Overview of Medical Research in the Netherlands (OMON; https://onderzoekmetmensen.nl/en/trial/54441). All participants provided written informed consent.

The indication for surgery was established during a joint consultation with the orthopedic surgeon and rehabilitation physician, using the principles outlined in a previously published treatment algorithm [4]. All participants underwent fusion of one or more tarsal joints to ensure permanent stabilization of the foot. This was often combined with additional procedures, such as a form of Achilles tendon lengthening and/or claw toe correction. Postoperatively, participants were treated with a non-weight-bearing cast for four weeks. This was followed by a below-the-knee walking cast for another four weeks, after which it was replaced by a removable walker. From that moment, rehabilitation was personalized, involving home-based physiotherapy focused on gait and balance training.

Outcome assessments

Personalized goal attainment, balance capacity, and gait capacity were assessed approximately four months before surgery ("Baseline 1"), shortly before surgery ("Baseline 2": only in the double baseline subgroup), and approximately 12 months after surgery ("Post surgery"). Personalized goals were evaluated using the Canadian Occupational Performance Measure (COPM) [14]. At Baseline 1, participants identified three to six goals and rated their importance on a scale from 1 to 10. Perceived performance of and satisfaction with each goal were assessed at all three time points, also on a 10-point scale. Balance capacity was examined using the Mini-Balance Evaluation Systems Test (Mini-BESTest) [15], which was performed barefoot without walking aid. To examine gait capacity, a three-dimensional instrumented gait

analysis was performed in our gait laboratory that features a Vicon motion capture system with ten infrared cameras (Vicon Motion Systems Ltd., Oxford, UK). Reflective markers were applied according to the Plug-in Gait lower body model (Vicon Motion Systems Ltd., Oxford, UK) and recorded at 100 Hz. Participants completed three to six barefoot walking trials at their preferred walking speed, with the option to use a personal walking aid if necessary.

Data analyses

To assess the overall change in personalized goal attainment after surgery, weighted mean COPM scores were calculated by multiplying the scores per individual goal by its importance score. Subsequently, the resulting weighted scores were summed and divided by the sum of the importance scores for all goals per individual and time point. The proportion of participants who showed an increase of at least two points in weighted mean COPM scores for both performance and satisfaction from Baseline 1 to Post surgery was calculated, as this is considered clinically meaningful [16, 17]. In addition, a detailed analysis was performed to examine the changes in personalized goals for different goal categories. Fifteen personalized goal categories were created using the nominal group technique [18, 19], as detailed in Supplementary material 1. Two raters independently classified all participant-identified goals into these categories. For the goals on which the raters disagreed, a third rater was included to reach a final decision based on consensus. COPM-performance and -satisfaction scores were calculated for each goal category, averaging scores when participants had multiple goals within the same category. The proportion of participants showing an increase of at least two points was calculated for the three most mentioned goal categories.

To assess balance capacity, Mini-BESTest scores before and after surgery were calculated. Furthermore, the proportion of participants showing an increase of at least four points, which is considered clinically meaningful [20], was calculated.

As a measure of gait capacity, gait speed was computed from the instrumented gait analysis. Data were processed using Vicon Nexus (Vicon Motion Systems Ltd., Oxford, UK) and MATLAB (Version R2021a, The MathWorks, Inc., Natick, MA, USA). Gait speed was calculated by dividing the distance covered by the heel marker by the time elapsed between two consecutive initial contacts, identified based on Zeni's methodology [21]. Subsequently, gait speed was averaged across walking trials. To evaluate post-surgical changes in gait speed, participants were grouped based on their barefoot pre-surgical gait speed: non-ambulators (<0.1 m/s), household ambulators (0.1-0.4 m/s), limited community ambulators (0.4-0.8 m/s), and unlimited community ambulators (>0.8 m/s) [22]. The proportion of participants who transitioned to a different gait speed group after surgery was calculated.

Statistical analyses

To study the change in COPM, Mini-BESTest, and gait speed during the baseline period, mean change scores between Baseline 1 and Baseline 2, and corresponding 95% confidence intervals (CIs), were calculated for participants in the double baseline subgroup. Furthermore, participants in the double baseline subgroup were compared with those in the single baseline subgroup by examining mean values and 95% CIs at Baseline 1 and Post surgery. For non-normally distributed data, median values were computed, and a percentile bootstrap method was used to calculate the 95% CIs [23].

Weighted mean COPM and Mini-BESTest scores were compared between Baseline 1 and Post surgery using paired samples t-tests or Wilcoxon signed-rank tests for non-normally distributed data. To compare changes in barefoot gait speed between the four groups classified by pre-surgical gait speed, a one-way Analysis of Variance (ANOVA) was used. If the data were not normally distributed, a Kruskal-Wallis test was used instead. Statistical significance was set at $p<0.05$.

Results

Of the 62 adults eligible for inclusion, 51 were willing to participate. Four participants were lost to follow-up, and one participant passed away because of an unrelated cause, resulting in a final sample of 46 participants (Table 1). Of these, 13 participants were included in the double baseline subgroup.

Table 1. Subject characteristics.

Characteristic	
Sex (# female)	27
Age (years: mean [range])	51 [20-74]
Time post stroke (years: mean [range])	7 [1-47]
Paretic side (# left)	19

A detailed overview of all surgical procedures is provided in Supplementary material 2. Postoperative complications arose in seven participants, including nonunion (n=3), neurogenic pain (n=2), wound infection (n=1), and a displaced screw (n=1). All participants with nonunion underwent restorative surgery, which in two cases involved a triple arthrodesis after an initial talonavicular arthrodesis.

Personalized goal attainment

No significant differences were observed in COPM-performance and COPM-satisfaction scores during the baseline period among participants in the double baseline subgroup. The mean change score between Baseline 1 and Baseline 2 for performance was -0.06 (95% CI: -0.55 to 0.43) and for satisfaction 0.14 (95% CI: -0.29 to 0.56). Furthermore, COPM scores of the double baseline subgroup were comparable to those of the single baseline subgroup, evidenced by the average weighted mean COPM scores of the double baseline subgroup falling within the 95% CIs of the weighted mean COPM scores of the single baseline subgroup at Baseline 1 and Post surgery (Supplementary material 3).

Weighted mean COPM scores improved significantly from Baseline 1 to Post surgery (COPM-performance: +3.5±1.7, $t(45)$=-13.6, $p<0.001$; COPM-satisfaction: +3.9±1.8, $t(45)$=-14.7, $p<0.001$, Figure 1). A clinically meaningful improvement in personalized goals (≥2 points) was observed in 78% of participants.

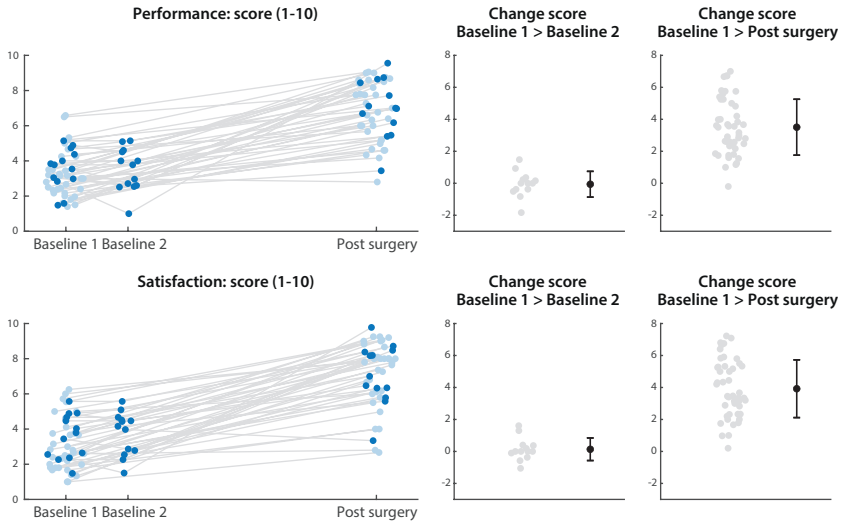


Figure 1. Personalized goal attainment before and after reconstructive surgery for pes equinovarus. In the top and bottom left panels, individual weighted mean COPM scores on performance and satisfaction at Baseline 1, Baseline 2, and Post surgery are presented. Participants in the single baseline subgroup are depicted in light blue, whereas participants in the double baseline subgroup are depicted in dark blue. In the top and bottom middle and right panels, the change scores between Baseline 1 and Baseline 2, and between Baseline 1 and Post surgery are presented, with positive values indicating an improvement at Baseline 2 or Post surgery with respect to Baseline 1, respectively. COPM: Canadian Occupational Performance Measure.

Figure 2 shows the COPM-performance and COPM-satisfaction scores across the 15 different goal categories. The most frequently mentioned category was "Improving standing or walking barefoot", which was identified by 24 out of 46 participants, followed by the categories "Increasing walking duration" (n=23) and "Improving walking on uneven surfaces" (n=20). Clinically meaningful improvements in COPM-performance and COPM-satisfaction scores (≥2 points) were observed in 21 of 24 participants (88%) for the category "Improving standing or walking barefoot", 17 of 23 participants (74%) for "Increasing walking duration", and 16 out of 20 participants (80%) for "Improving walking on uneven surfaces".

Balance capacity

No significant change in Mini-BESTest score was observed during the baseline period for the double baseline subgroup. The mean change score between Baseline 1 and Baseline 2 was 0.38 (95% CI: -1.61 to 2.38). Furthermore, Mini-BESTest scores of the double baseline subgroup showed no significant difference from the single baseline subgroup, as denoted by the mean values of the double baseline subgroup falling within the 95% confidence intervals of the single baseline subgroup at Baseline 1 and Post surgery (Supplementary material 3).

Twenty-nine participants performed the Mini-BESTest before and after surgery, as many were unable to complete the assessment. Mini-BESTest scores showed a statistically significant improvement after surgery of 5.1±5.4 ($t(28)=-5.0$, $p<0.001$, Figure 3). In 15 out of 29 participants (52%), a clinically meaningful enhancement in balance capacity (≥4 points) was observed.

Gait capacity

Regarding gait capacity, three out of the 46 participants were excluded because of missing gait data, and two participants were excluded as they no longer required a walking aid after surgery, rendering conditions incomparable. Of the remaining 41 participants, 24 were unable to walk barefoot before surgery, making barefoot instrumented gait analysis impossible. After surgery, 19 of these 24 participants (79%) were able to perform the instrumented gait analysis barefoot.

No significant difference in gait speed was observed between Baseline 1 and Baseline 2 (mean change: -0.04 m/s, 95% CI: -0.15 to 0.07). Gait speed was not different in the double compared to the single baseline subgroup, indicated by the mean values of the double baseline subgroup falling within the 95% CIs of the single baseline subgroup at Baseline 1 and Post surgery (Supplementary material 3).

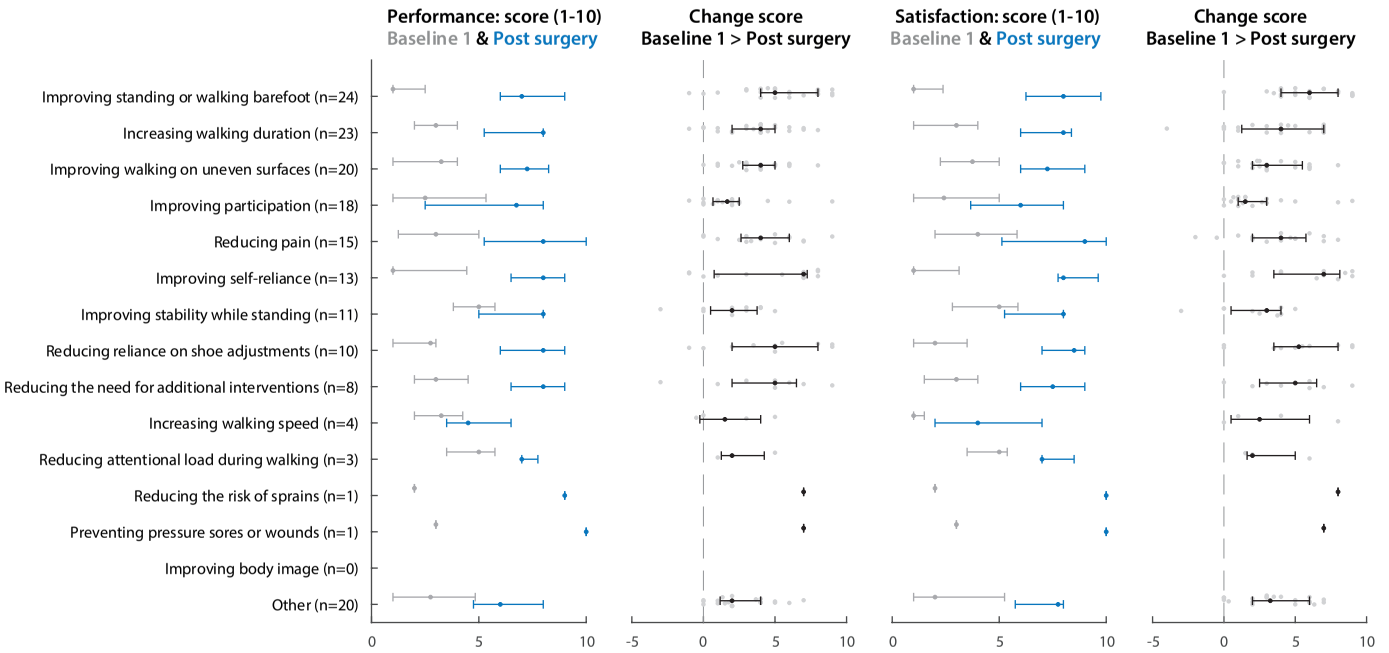


Figure 2. Personalized goal attainment for different goal categories before and after reconstructive surgery for pes equinovarus. In the first and third panels, median COPM scores and corresponding interquartile ranges on performance and satisfaction are depicted for Baseline 1 and Post surgery. In the second and fourth panels, change scores between Baseline 1 and Post surgery are depicted, with positive values indicating an improvement at Post surgery with respect to Baseline 1. COPM: Canadian Occupational Performance Measure.

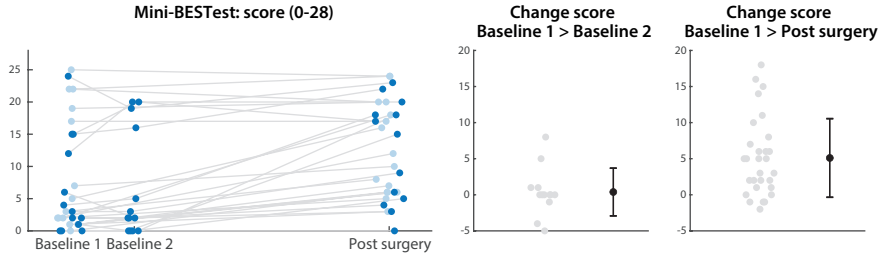


Figure 3. Balance capacity before and after reconstructive surgery for pes equinovarus. Mini-BESTest scores at Baseline 1, Baseline 2, and Post surgery are presented in the panel at the left. Participants from the single baseline subgroup are depicted in light blue, whereas those from the double baseline subgroup are depicted in dark blue. The change scores between Baseline 1 and Baseline 2, and between Baseline 1 and Post surgery are presented in the middle and right panels. Positive values indicate an improvement at Baseline 2 or Post surgery with respect to Baseline 1, respectively. Mini-BESTest: Mini-Balance Evaluation Systems Test.

Changes in barefoot gait speed after surgery differed among the four groups classified by pre-surgical gait speed ($\chi 2(3)=13.9$, $p=0.003$, Figure 4). Increases in barefoot gait speed were observed in the pre-surgical non-ambulators (median change: +0.21 m/s; interquartile range (IQR): 0.10-0.34 m/s) and the pre-surgical household ambulators (median change: +0.18 m/s; IQR: 0.06-0.34 m/s). No differences in barefoot gait speed were observed in the limited community ambulators (median change: +0.09 m/s; IQR: 0.00-0.11 m/s) and the unlimited community ambulators (median change: -0.08 m/s; IQR: -0.15-0.04 m/s). Among the 24 pre-surgical non-ambulators, 16 (67%) achieved barefoot household ambulatory status, whereas three (13%) attained limited community ambulatory status. Of the six pre-surgical household ambulators, four (67%) transitioned to post-surgery limited community ambulatory status. In addition, among the six pre-surgical unlimited community ambulators, one (17%) transitioned to limited community ambulator status. All other participants retained their pre-surgical ambulatory status after surgery.

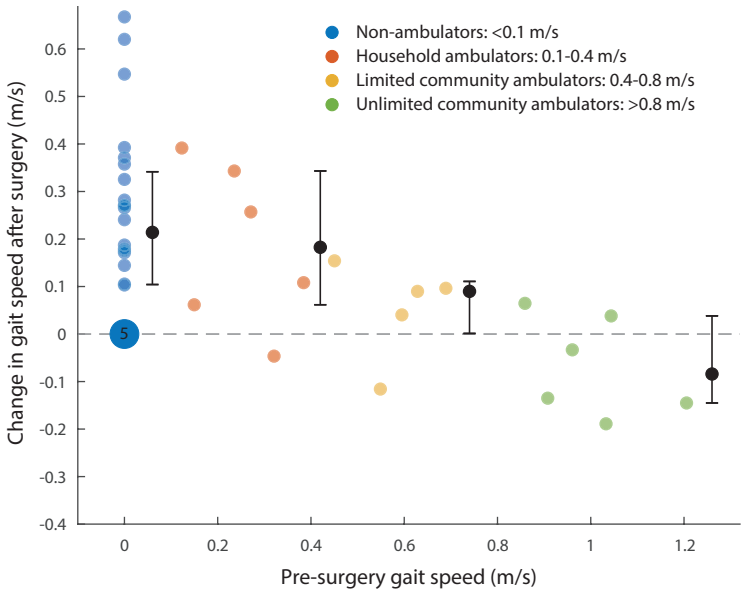


Figure 4. Barefoot gait capacity in relation to reconstructive surgery for pes equinovarus. Individual data points, median values, and interquartile ranges of post-surgical changes in barefoot gait speed are presented for four groups, classified by barefoot pre-surgical gait speed: non-ambulators (<0.1 m/s), household ambulators (0.1-0.4 m/s), limited community ambulators (0.4-0.8 m/s), unlimited community ambulators (>0.8 m/s).

Discussion

This study evaluated the effect of surgical equinovarus correction on personalized goal attainment and balance and gait capacity in individuals with stroke. A prospective observational study was conducted, with no differences observed during the baseline period. Twelve months after surgery, significant improvements were found in personalized goals and balance capacity. Gait capacity also improved, with many participants who were unable to walk barefoot before surgery able to do so afterward. Improvements in barefoot gait speed were found to be related to pre-surgical gait speed, with individuals walking slower before surgery showing greater improvement. These findings support the effectiveness of tarsal arthrodesis, a conclusion further strengthened by the low incidence of post-surgical complications.

This study is the first to examine the effect of corrective surgery for pes equinovarus through tarsal arthrodesis on personalized goals [13]. A previous study examined the effect of selective tibial neurotomy on personalized goal attainment assessed with

the Goal Attainment Scale [24]. Although after a different surgical intervention, this earlier study also demonstrated improvements in personalized goals in 72% of participants, compared to 78% in the current study. However, in this previous study, not all outcome measures demonstrated improvements [24], including the Functional Ambulation Category and the Short-Form Health Survey, suggesting that individuals' personalized goals do not always align with the aspects assessed by standardized tests or questionnaires. This highlights that personalized goal assessment is highly suitable for evaluating surgery, as it accounts for the goals most important to each individual, thereby ensuring a comprehensive and individualized assessment of surgical effectiveness. In the current study, the most frequently mentioned goal category was "Improving standing or walking barefoot", for which a substantial improvement was seen. By providing insights into which personalized goals are likely to improve after surgery, this study offers valuable guidance for optimizing pre-operative counseling.

As for balance capacity, we found a clinically meaningful improvement in most participants after surgery. Given that impaired balance capacity elevates the risk of falls, the observed enhancement in balance capacity may have a significant positive impact on health care, considering the severe physical and psychosocial consequences of falls [25]. However, future research is needed to determine whether the surgical intervention actually reduces the incidence of falls.

A clinically meaningful improvement in barefoot gait capacity was observed in most participants. Previous research revealed a 25% increase in gait speed after surgery [9, 10]. Considering that in these prior studies mainly individuals with low pre-surgical gait speeds were included (0.38±0.20 m/s [9] and 0.32±0.19 m/s [10]), the findings align with this study's results. However, this study expands on this by including participants with a larger variation of pre-surgical gait speeds, revealing that improvements are not the same across all individuals. This insight is valuable for pre-operative counseling, helping to set realistic expectations. Future research should further explore the biomechanical mechanisms underlying changes in gait speed to enhance our understanding of the effect of surgery. In particular, non-paretic push-off power may warrant further investigation, given evidence of increased push-off power after equinovarus correction [10].

Notably, four out of six participants whose gait speed did not improve still reported clinically meaningful gains in personalized goals. Their personalized goals were most commonly related to improving walking on uneven surfaces, improving walking duration, and reducing pain. Therefore, future research should explore the effect of surgery on other aspects of gait, including gait adaptability and daily life gait

performance [26-28], aligning with the frequently mentioned goal categories "Improving walking on uneven surfaces" and "Increasing walking duration". This may be particularly relevant for participants with high pre-surgical gait speeds, helping to explain the improvements in personalized goals despite no corresponding gait speed improvement.

Study Limitations

While a randomized clinical trial would provide stronger scientific evidence, it was unfeasible since many people would not accept randomization to a control group. By introducing a second baseline assessment, this study was able to examine whether potential time effects influenced the outcomes. As all participants were in the chronic phase following stroke, it was expected that nonspecific time effects would be minimal, which was confirmed by the absence of outcome differences during the baseline period. In addition, selection bias may have been introduced, as the second baseline assessment was not conducted for all participants. This was because we started collecting these second baseline assessments at a later stage, and not all participants were willing to participate. However, no significant differences were observed between the double and the single baseline subgroups, suggesting that the results from the double baseline subgroup are generalizable. Furthermore, selection bias may have arisen from missing data. As missing data primarily occurred in participants with more severe impairments who were unable to complete the assessments, the effects of surgery may have been underestimated. Another limitation of this study is the absence of investigation into potential confounding factors, such as cognitive impairments and sensory loss, whose impact on surgical outcomes should be addressed in future research. Likewise, potential causes of post-surgical complications were not examined and warrant further investigation. Lastly, the variety of surgical procedures introduced heterogeneity, and the limited sample size precluded subgroup analyses. Accordingly, this study evaluated the effect of a normalized ankle-foot position achieved through various surgical procedures, rather than examining any single procedure. Moreover, this study focused on tarsal arthrodesis, whereas soft tissue procedures represent an alternative approach. Future studies comparing these approaches are needed to guide evidence-based surgical management of pes equinovarus.

Conclusion

This study demonstrated clinically relevant improvements in personalized goals, balance, and gait following reconstructive surgery for pes equinovarus in people with stroke. The findings provide guidance for optimal pre-operative counseling. Further studies are recommended to determine whether reconstructive surgery is more cost-effective compared with nonoperative treatment, as suggested by previous research [29].

References

[1] Verdié C, Daviet JC, Borie MJ, Popielarz S, Munoz M, Salle JY, et al. Epidemiology of pes varus and/or equinus one year after a first cerebral hemisphere stroke: Apropos of a cohort of 86 patients. Ann Readapt Med Phys. 2004;47(2):81-6. https://doi.org/10.1016/j.annrmp.2003.10.005.

[2] Patrick JH, Keenan MAE. Gait analysis to assist walking after stroke. Lancet. 2007;369(9558):256-7. https://doi.org/10.1016/S0140-6736(07)60124-X.

[3] Mandiroglu S, Firat M. Foot and ankle deformities in stroke: relationship with ambulation, balance, and daily living activities: A cross-sectional study. Somatosens Mot Res. 2022;39(2-4):106-10. https://doi.org/10.1080/08990220.2021.2018294.

[4] Nonnekes J, Benda N, Van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[5] Renzenbrink GJ, Buurke JH, Nene AV, Geurts ACH, Kwakkel G, Rietman JS. Improving walking capacity by surgical correction of equinovarus foot deformity in adult patients with stroke or traumatic brain injury: A systematic review. J Rehabil Med. 2012;44(8):614-23. https://doi.org/10.2340/16501977-1012.

[6] Allart É, Sturbois-Nachef N, Salga M, Rosselin C, Gatin L, Genêt F. Neuro-orthopedic surgery for equinovarus foot deformity in adults: A narrative review. J Foot Ankle Surg. 2022;61(3):648-56. https://doi.org/10.1053/j.jfas.2021.11.012.

[7] Genêt F, Denormandie P, Keenan M. Orthopaedic surgery for patients with central nervous system lesions: Concepts and techniques. Ann Phys Rehabil Med. 2019;62(4):225-33. https://doi.org/10.1016/j.rehab.2018.09.004.

[8] Louwerens JWK. Triple arthrodesis. Tech Foot Ankle Surg. 2012;11(4):181-8. https://doi.org/10.1097/BTF.0b013e3182743f68.

[9] Nonnekes J, Kamps M, den Boer J, van Duijnhoven H, Lem F, Louwerens JWK, et al. Tarsal fusion for pes equinovarus deformity improves gait capacity in chronic stroke patients. J Neuroeng Rehabil. 2019;16:102. https://doi.org/10.1186/s12984-019-0572-2.

[10] Carda S, Bertoni M, Zerbinati P, Rossini M, Magoni L, Molteni F. Gait changes after tendon functional surgery for equinovarus foot in patients with stroke: Assessment of temporo-spatial, kinetic, and kinematic parameters in 177 patients. Am J Phys Med Rehabil. 2009;88(4):292-301. https://doi.org/10.1097/PHM.0b013e318198b593.

[11] Giannotti E, Merlo A, Zerbinati P, Prati P, Masiero S, Mazzoli D. Safety and long-term effects on gait of hemiplegic patients in equinovarus foot deformity surgical correction followed by immediate rehabilitation: A prospective observational study. Eur J Phys Rehabil Med. 2018;55(2):169-75. https://doi.org/10.23736/S1973-9087.18.05290-5.

[12] Salga M, Gatin L, Deltombe T, Gustin T, Carda S, Marque P, et al. International recommendations to manage poststroke equinovarus foot deformity validated by a panel of experts using delphi. Arch Phys Med Rehabil. 2023;104(3):372-9. https://doi.org/10.1016/j.apmr.2022.07.020.

[13] de l'Escalopier N, Voisard C, Michaud M, Moreau A, Jung S, Tervil B, et al. Evaluation methods to assess the efficacy of equinovarus foot surgery on the gait of post-stroke hemiplegic patients: A literature review. Front Neurol. 2022;13:1042667. https://doi.org/10.3389/fneur.2022.1042667.

[14] Law M, Baptiste S, McColl M, Opzoomer A, Polatajko H, Pollock N. The Canadian occupational performance measure: An outcome measure for occupational therapy. Can J Occup Ther. 1990;57(2):82-7. https://doi.org/10.1177/000841749005700207.

[15] Franchignoni F, Horak F, Godi M, Nardone A, Giordano A. Using psychometric techniques to improve the Balance Evaluation System's Test: The mini-BESTest. J Rehabil Med. 2010;42(4):323-31. https://doi.org/10.2340/16501977-0537.

[16] Eyssen ICJM, Steultjens MPM, Oud TAM, Bolt EM, Maasdam A, Dekker J. Responsiveness of the Canadian occupational performance measure. J Rehabil Res Dev. 2011;48(5):517-28. https://doi.org/10.1682/jrrd.2010.06.0110.

[17] Law M, Baptiste S, Carswell A, McColl M, Polatajko H, Pollock N. Canadian Occupational Performance Measure. 4th ed. Toronto (Canada): Canadian Association of Occupational Therapists. 2005;
[18] Gallagher M, Hares T, Spencer J, Bradshaw C, Webb I. The nominal group technique: a research tool for general practice? Fam Pract. 1993;10(1):76-81. https://doi.org/10.1093/fampra/10.1.76.
[19] McMillan SS, King M, Tully MP. How to use the nominal group and Delphi techniques. Int J Clin Pharm. 2016;38(3):655-62. https://doi.org/10.1007/s11096-016-0257-x.
[20] Beauchamp MK, Niebuhr R, Roche P, Kirkwood R, Sibley KM. A prospective study to establish the minimal clinically important difference of the Mini-BESTest in individuals with stroke. Clin Rehabil. 2021;35(8):1207-15. https://doi.org/10.1177/02692155211025131.
[21] Zeni Jr JA, Richards JG, Higginson JS. Two simple methods for determining gait events during treadmill and overground walking using kinematic data. Gait Posture. 2008;27(4):710-4. https://doi.org/10.1016/j.gaitpost.2007.07.007.
[22] Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. Stroke. 1995;26(6):982-9. https://doi.org/10.1161/01.str.26.6.982.
[23] DiCiccio TJ, Efron B. Bootstrap confidence intervals. Statist. Sci. 1996;11(3):189-228. https://doi.org/10.1214/ss/1032280214.
[24] Deltombe T, Gilliaux M, Peret F, Leeuwerck M, Wautier D, Hanson P, et al. Effect of the neuro-orthopedic surgery for spastic equinovarus foot after stroke: A prospective longitudinal study based on a goal-centered approach. Eur J Phys Rehabil Med. 2018;54(6):853-9. https://doi.org/10.23736/S1973-9087.18.04993-6.
[25] Weerdesteyn V, de Niet M, van Duijnhoven HJR, Geurts ACH. Falls in individuals with stroke. J Rehabil Res Dev. 2008;45(8):1195-213. https://doi.org/10.1682/JRRD.2007.09.0145.
[26] Balasubramanian CK, Clark DJ, Fox EJ. Walking adaptability after a stroke and its assessment in clinical settings. Stroke Res Treat. 2014;2014:591013. https://doi.org/10.1155/2014/591013.
[27] Braakhuis HEM, Berger MAM, Regterschot RGRH, van Wegen EEH, Selles RW, Ribbers GM, et al. Physical activity dimensions after stroke: Patterns and relation with lower limb motor function. J Neuroeng Rehabil. 2021;18(1):171. https://doi.org/10.1186/s12984-021-00960-x.
[28] Hendriks MMS, Vos-van der Hulst M, Weijs RWJ, van Lotringen JH, Geurts ACH, Keijsers NLW. Using sensor technology to measure gait capacity and gait performance in rehabilitation inpatients with neurological disorders. Sensors. 2022;22(21):8387. https://doi.org/10.3390/s22218387.
[29] Reddy S, Kusuma S, Hosalkar H, Keenan MA. Surgery can reduce the nonoperative care associated with an equinovarus foot deformity. Clin Orthop Relat Res. 2008;466(7):1683-7. https://doi.org/10.1007/s11999-008-0250-3.

Supplementary material 1: Devising of personalized goal categories

Goal categories were formulated using the nominal group technique (NGT) with a panel of clinical experts specialized in ankle-foot surgery in people with stroke. The clinical expert group comprised three orthopedic surgeons, three rehabilitation physicians, and one physician assistant in rehabilitation medicine. The goal of the NGT meeting was to formulate categories of personalized goals of individuals with stroke who undergo a surgical correction of pes equinovarus.

Prior to the live meeting, all experts were asked to conduct an individual brainstorming session aimed at devising as many categories as possible. The devised categories of all clinical experts were then shared with the group before the live meeting. The live meeting consisted of four rounds. During the first round, the clinical experts could introduce additional categories to those initially devised during the individual brainstorming sessions. The second round consisted of a group discussion in which all devised categories were discussed. During this round, it was allowed to add new categories, to combine categories, and to remove categories as long as everybody agreed. In the third round, the outcomes of the second round were presented, after which the group could decide whether further adjustments were desired. In the fourth and final round, all clinical experts voted either for or against each devised category, determining whether it should be included in the ultimate list of categories. If a 100% consensus on the list of categories was not reached, a new group discussion was initiated to address the categories where consensus had not yet been reached. Finally, a new round of voting was conducted. If five or more clinical experts (71%) voted to include the respective category, the category was added to the final list of categories.

Following the NGT meeting, 14 categories of personalized goals were formulated, with a 100% consensus reached for each category. The list of categories consisted of: 1) Increasing walking duration, 2) Increasing walking speed, 3) Reducing attentional load during walking, 4) Improving stability while standing, 5) Improving standing or walking barefoot, 6) Reducing the risk of sprains, 7) Preventing pressure sores or wounds, 8) Reducing pain, 9) Improving participation in work, hobbies, or social activities, 10) Improving self-reliance, 11) Improving body image, 12) Reducing reliance on shoe adjustments, 13) Reducing the need for additional interventions such as orthoses or focal spasmolysis, and 14) Other. Subsequently, based on pilot classification of the participant-identified goals into the defined categories, an additional category was introduced: 15) Improving walking on uneven surfaces. Furthermore, the thirteenth category was revised to: 13) Reducing the need for additional interventions such as orthoses, walking aids, or focal spasmolysis.

Supplementary material 2: Surgical interventions

Surgical interventions were tailored to address the specific needs of each individual. All participants underwent fusion of one or more tarsal joints to correct pes varus deformity. This included one of the following procedures: talonavicular arthrodesis (fusion of the talonavicular joint), diple arthrodesis (fusion of the talonavicular and talocalcaneal joints), triple arthrodesis (fusion of the talonavicular, talocalcaneal and calcaneocuboid joints), or triple arthrodesis with Lambrinudi component (a triple arthrodesis combined with a wedge-shaped resection of bone from the talus and calcaneus to correct pes equinus). Pes equinus was more commonly treated with Achilles tendon lengthening or gastrocnemius fasciotomy.

Table S1. Overview of performed surgical procedures.

Surgical procedure		Number of participants who underwent this procedure
Tarsal arthrodesis		46 (100%)
	Talonavicular arthrodesis	27* (59%)
	Diple arthrodesis	8 (17%)
	Triple arthrodesis	5 (11%)
	Triple arthrodesis with Lambrinudi component	6 (13%)
Achilles tendon lengthening		23 (50%)
Gastrocnemius fasciotomy		9 (20%)
Lesser toes correction**		40 (87%)
Hallux correction***		25 (54%)
Tibialis posterior tenotomy		6 (13%)
Tibialis posterior transfer		4 (9%)
Dorsiflexion osteotomy first metatarsal bone		8 (17%)

**In two participants, a talonavicular arthrodesis was initially performed, followed by reconstructive triple arthrodesis due to nonunion. **Correction of the lesser toes involved tenotomy of the toe flexor(s) and/or proximal interphalangeal joint resection of the toes. ***Correction of the hallux included interphalangeal joint arthrodesis and/or tenotomy, transfer, or lengthening of the extensor hallucis longus, flexor hallucis longus, and/or flexor hallucis brevis.*

Supplementary material 3: Personalized goal attainment, balance capacity, and gait capacity of participants in the single and double baseline subgroups

Table S2. Mean values and 95% confidence intervals of personalized goal attainment (COPM), balance capacity (Mini-BESTest), and gait capacity (gait speed).

	Baseline 1	Baseline 2	Post surgery
COPM (score)			
Performance			
Total group	3.3 [2.9, 3.7]	-	6.8 [6.3, 7.3]
Single baseline subgroup	3.2 [2.7, 3.7]	-	6.7 [6.1, 7.3]
Double baseline subgroup	3.6 [2.9, 4.3]	3.5 [2.8, 4.2]	7.0 [6.0, 8.0]
Satisfaction			
Total group	3.2 [2.8, 3.6]	-	7.1 [6.6, 7.7]
Single baseline subgroup	3.1 [2.5, 3.6]	-	7.1 [6.5, 7.8]
Double baseline subgroup	3.6 [2.9, 4.4]	3.8 [3.0, 4.5]	7.1 [6.1, 8.2]
Mini-BESTest (score)			
Total group	7.4 [4.2, 10.6]	-	12.5 [9.7, 15.3]
Single baseline subgroup	8.1 [3.2, 13.1]	-	12.6 [8.8, 16.4]
Double baseline subgroup	6.4 [1.9, 11.1]	6.8 [1.7, 11.9]	12.3 [7.5, 17.2]
Gait speed (m/s)			
Total group*	0.64 [0.47, 0.82]	-	0.68 [0.54, 0.82]
Single baseline subgroup	0.64 [0.39, 0.88]	-	0.68 [0.48, 0.88]
Double baseline subgroup	0.66 [0.33, 0.99]	0.62 [0.37, 0.88]	0.69 [0.48, 0.89]

*COPM: Canadian Occupational Performance Measure, Mini-BESTest: Mini-Balance Evaluation Systems Test. *Due to missing gait data at Baseline 2 for one participant in the double baseline subgroup, the values presented in this table include data from five participants from the double baseline subgroup, 11 from the single baseline subgroup, and 16 participants in the total group. Mean gait speed values and 95% confidence intervals for the total group of 17 participants were: 0.61 [0.44, 0.79] for Baseline 1, and 0.67 [0.54, 0.80] for Post surgery.*



Chapter 7

Improved gait biomechanics following surgical correction of pes equinovarus in individuals with stroke

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Submitted

Abstract

Background

In people with stroke, pes equinovarus often leads to gait impairments. This study aimed 1) to investigate the effects of reconstructive surgery for pes equinovarus on spatiotemporal gait parameters and lower limb kinematics and kinetics, 2) to identify biomechanical determinants of changes in gait speed following surgery, and 3) to assess plantar pressure after surgical correction.

Methods

Forty-six adults were included in this repeated measures study. Each participant underwent surgical equinovarus correction, which included tarsal arthrodesis, frequently combined with Achilles tendon lengthening. Three-dimensional instrumented gait analysis was performed before and twelve months post surgery. Plantar pressure measurements were conducted twelve months post surgery and compared to a database of healthy controls.

Results

Among the 17 participants who were able to walk barefoot before and after surgery, increased ankle dorsiflexion and a reduced ankle moment on the paretic side were observed after surgery. Post-surgical changes in gait speed were associated with changes in stride length, cadence, step length on both sides, and peak ankle power on both sides. Compared to healthy controls (n=586), people with stroke post surgery (n=16) demonstrated minor differences in medio-lateral center of pressure trajectory.

Conclusion

Surgical correction of pes equinus led to increased ankle dorsiflexion, whereas correction of pes varus was reflected by a normalized medio-lateral center of pressure trajectory. Improvements in gait speed after surgery were associated with increased push-off power at the ankle joint on the non-paretic side, while a decrease in gait speed was associated with a decrease in peak ankle power on the paretic side.

Introduction

Pes equinovarus, characterized by a plantar flexed foot position (pes equinus) and varus alignment of the hindfoot (pes varus), is common after stroke and often results in reduced functional mobility [1-3]. In a recent large prospective observational study, we demonstrated that surgical correction of pes equinovarus, involving fusion of the talonavicular or more tarsal joints and often combined with Achilles tendon lengthening, is an effective treatment for improving personalized goals and enhancing balance capacity in people with stroke [4]. Additionally, our study revealed that individuals with a relatively low pre-surgical gait speed showed meaningful improvements in gait speed after surgery.

Despite these promising findings, two questions remain. First, the effects of surgical equinovarus correction on gait biomechanics have yet to be explored. Prior research in cohorts with relatively low pre-surgical gait speeds (0.32±0.19 m/s [5] and 0.38±0.20 m/s [6]) reported improvements of cadence, stride length, and of step length on both the paretic and non-paretic sides, as well as increased peak ankle power on the non-paretic side following surgery [5, 6]. However, our recent study revealed considerable variability in gait speed improvements associated with pre-surgical gait speed in a cohort with a mean pre-surgical gait speed of 0.61±0.34 m/s [4]. Therefore, examining biomechanical changes after surgery and their association with post-surgical changes in gait speed across this broader range of pre-surgical gait speeds is crucial. Second, the effect of surgical correction of pes equinovarus on foot loading during gait has not yet been established [7]. Altered foot loading during gait in people with pes equinovarus has been documented, particularly in the medio-lateral center of pressure trajectory [8]. As altered foot loading may compromise stability during stance, assessing whether surgical equinovarus correction restores more plantigrade loading is important.

The aims of this study were: 1) to investigate the effect of reconstructive surgery for pes equinovarus on barefoot gait in individuals with stroke, including spatiotemporal parameters and lower limb kinematics and kinetics, 2) to identify biomechanical determinants associated with changes in gait speed following surgery, and 3) to assess plantar pressure after surgical equinovarus correction. Since tarsal arthrodesis was often combined with a form of Achilles tendon lengthening to correct pes equinus, we expected increased ankle dorsiflexion throughout the gait cycle [5, 9]. Furthermore, we hypothesized that improved gait speed would be associated with enhanced ankle push-off power on the non-paretic side [5]. Lastly, we hypothesized that reconstructive surgery involving arthrodesis of the talonavicular or more tarsal joints, aimed at correcting pes varus deformity, would normalize the medio-lateral center of pressure trajectory to resemble that of healthy controls [8].

Methods

Participants and design

This study was part of a larger study involving additional measurements, with a previous publication detailing the effects of reconstructive surgery for pes equinovarus in people with stroke on personalized goal attainment, balance capacity, and gait speed (registration Overview of Medical-Scientific Research in the Netherlands: https://onderzoekmetmensen.nl/en/trial/54441). All adults with chronic stroke who underwent surgical equinovarus correction at the Gait Expertise Center of the Sint Maartenskliniek and Radboud university medical center were eligible. Exclusion criteria were: 1) presence of any other disorder significantly impacting gait, 2) previous ankle/foot surgery within the preceding two years, and 3) onset of stroke before the age of 18. The surgical intervention adhered to a previously published treatment algorithm for addressing pes equinovarus in people with stroke [3] and involved fusion of the talonavicular joint or more tarsal joints, often in combination with a form of Achilles tendon lengthening and/or claw toe correction. Ethical approval for the study was granted by the Medical Ethical Committee (NL77992.091.21) and written informed consent was obtained from all participants.

Examinations

Measurements were performed approximately four months before and twelve months after surgery. At both time points, comprehensive three-dimensional instrumented gait analysis was conducted in the gait laboratory of the Sint Maartenskliniek. This gait laboratory is equipped with a ten-camera motion capture system (Vicon Motion Systems Ltd., Oxford, UK) and two force plates (Kistler, Winterthur, Switzerland). Reflective markers were attached to the participants according to the Plug-in Gait lower body model (Vicon Motion Systems Ltd., Oxford, UK). Marker data were recorded at a sample frequency of 100 Hz, while force plate data were captured at a sample frequency of 1000 Hz. Participants walked barefoot at a self-selected walking speed, completing two to four measurements for each foot. If participants required a walking aid, ground reaction forces were not measured, as the walking aid interferes with force measurement.

Because plantar pressure analysis is not feasible in severe equinovarus deformity [8], plantar pressure measurements were obtained only twelve months after surgery. Plantar pressure measurements were conducted using a Footscan pressure plate of 0.5 m long (RSscan, Olen, Belgium), positioned on and synchronized with a force plate (Kistler, Winterthur, Switzerland). Pressure data were sampled at 500 Hz. Participants completed three to six barefoot trials with the paretic foot at self-selected walking speed, without using a walking aid. A historical cohort of plantar pressure data from

586 healthy controls with normal gait was used as reference [8, 10]. Although initially included in the trial registration, GRAIL (Gait Real-time Analysis Interactive Lab, Motek Medical BV, the Netherlands) measurements and daily life gait performance assessments were deemed unfeasible, as participants' gait capacity was too limited to complete these measurements and obtain clinically meaningful data.

Data analyses

Gait data processing was carried out using Vicon Nexus (Vicon Motion Systems Ltd., Oxford, UK) following the Plug-in Gait lower body model (Vicon Motion Systems Ltd., Oxford, UK). Subsequent analyses were conducted in MATLAB (Version R2021a, The MathWorks, Inc., Natick, MA, USA). Gait events for the paretic and non-paretic leg were identified using the methodology described by Zeni and colleagues [11]. Spatiotemporal gait parameters, kinematics, and kinetics were averaged across walking trials for each participant for the paretic and non-paretic leg. The following spatiotemporal gait parameters were calculated: gait speed, stride length, step length (paretic and non-paretic side), cadence, single support time (paretic and non-paretic side), and double support time. Additionally, hip, knee, and ankle angles, moments, and powers across the gait cycle were derived. Finally, post-surgical changes in gait speed, cadence, stride length, step length on both the paretic and non-paretic sides, and peak ankle power on both the paretic and non-paretic sides were calculated.

Following the recommendations outlined in previous research [8], mean normalized plantar pressure patterns and medio-lateral center of pressure trajectories were computed from the plantar pressure measurements for people with stroke post surgery and for healthy controls. A previously established method was used to normalize the plantar pressure patterns for foot length, width, and progression angle [12]. After this normalization, each pixel's pressure was divided by the total pressure of all pixels to account for variations in body weight and walking speed [10]. For each participant, the average normalized plantar pressure pattern was calculated over all trials. Subsequently, the pressure of each pixel was multiplied by the average total pressure of all pixels across all subjects. Additionally, the center of pressure (COP) trajectory in the medio-lateral direction was calculated and normalized to foot width, which was based on the pressure contact area [8].

Statistical analyses

Paired samples t-tests were used to statistically compare gait speed, stride length, cadence, and double support time before and after surgery. Instead, for non-normally distributed data, Wilcoxon signed-rank tests were used. Furthermore, two-way repeated measures Analysis of Variance (ANOVA) tests were conducted with surgery

(pre vs. post) and side (paretic vs. non-paretic) as independent variables to assess the effects of surgery on step length, single support time, and peak ankle power. In the case of a statistically significant interaction effect, post hoc tests were performed to study the effects of surgery for the paretic and non-paretic side. In addition, paired samples t-tests were used to compare hip, knee, and ankle kinematics and kinetics before and after surgery using statistical parametric mapping. This method enables the identification of significant differences in the kinematic and kinetic curves across the gait cycle, while accounting for multiple comparisons by using random field theory [13]. Statistical significance was defined as $p<0.05$.

To explore the determinants associated with changes in gait speed after surgery, single linear regression analyses were conducted to examine the relationships between post-surgical changes in gait speed and changes in cadence, stride length, step length on both the paretic and non-paretic sides, and peak ankle power on both the paretic and non-paretic sides. For all six linear regression models, the coefficients of determination (R^2) were calculated as measures of goodness of fit.

Post-surgical normalized plantar pressure patterns of people with stroke were compared to healthy controls using independent samples t-tests for each pixel within the plantar pressure pattern. A p-value below 0.017 was deemed statistically significant, based on a Bonferroni-type correction (0.05/3), corresponding to the three clusters of pixels showing similar deviation from the healthy control pattern. This approach for correcting the p-value regarding the number of deviations has been previously advocated in the analysis of plantar pressure patterns [8, 14, 15]. Post-surgical medio-lateral center of pressure trajectories of people with stroke were statistically compared to those of healthy controls using statistical parametric mapping [13]. Independent samples t-tests were used for the comparisons and statistical significance was determined at $p<0.05$.

Results

The study sample comprised 46 participants with a mean age of 51 years (range: 20-74 years) and a mean time post stroke of 7 years (range: 1-47 years). Twenty-seven participants were female and 19 participants had left-sided hemiparesis. A detailed overview of the performed surgical interventions is provided in our previous publication [4].

Three-dimensional instrumented gait analysis

For instrumented gait analysis, five participants were excluded due to missing data or cessation of walking aid use after surgery, as previously described [4], resulting in a final sample of 41 participants for whom barefoot gait was assessed. As reported in our previous publication [4], 24 of these participants (59%) were unable to walk barefoot before surgery, but 19 of them (79%) were able to do so afterwards. Spatiotemporal gait parameters and kinematics were analyzed in the 17 participants who completed barefoot instrumented gait analysis both before and after surgery. Since four participants used a walking aid at both time points, preventing accurate force plate measurements, kinetics were analyzed in 13 participants.

T-tests revealed no statistically significant differences in gait speed, stride length, cadence, or double support time following surgery (Table 1). Two-way repeated measures ANOVA revealed a statistically significant main effect of side (paretic vs. non-paretic) for step length ($F(1,16) = 11.3$, $p=0.004$). Furthermore, although there was a trend towards significance, the main effect of surgery on step length did not reach statistical significance ($F(1,16) = 4.0$, $p=0.06$), nor did the interaction between side and surgery ($F(1,16) = 0.2$, $p=0.7$) (Figure 1A). Similarly, for single support time, there was a statistically significant main effect of side ($F(1,16) = 31.6$, $p<0.001$), whereas no statistically significant main effect of surgery was found ($F(1,16) = 2.7$,

Table 1. Spatiotemporal gait parameters before and after surgical correction of pes equinovarus (n=17).

Spatiotemporal parameter	Pre surgery (mean±SD)	Post surgery (mean±SD)	Change (mean±SD)	p-value
Gait speed (m/s)	0.61±0.34	0.67±0.25	+0.06±0.17	p=0.17
Stride length (m)	0.85±0.30	0.94±0.21	+0.09±0.18	p=0.06
Cadence (steps/min)	81.2±24.6	84.1±20.5	+2.9±12.4	p=0.35
Double support time (%)	36.2±12.7	31.9±6.1	-4.3±10.8	p=0.12

SD: standard deviation.

p=0.1), and no statistically significant interaction effect (F(1,16) = 0.2, p=0.6) (Figure 1B). For peak ankle power, a statistically significant interaction effect (F(1,12) = 14.4, p=0.003) and main effect of side (paretic vs. non-paretic: F(1,12) = 75.6, p<0.001) were found, whereas no statistically significant main effect of surgery was found (F(1,12) = 0.0, p=0.8) (Figure 1C). Post hoc testing revealed non-statistically significant trends towards a decrease in peak ankle power on the paretic side (p=0.09) and an increase in peak ankle power on the non-paretic side following surgery (p=0.08).

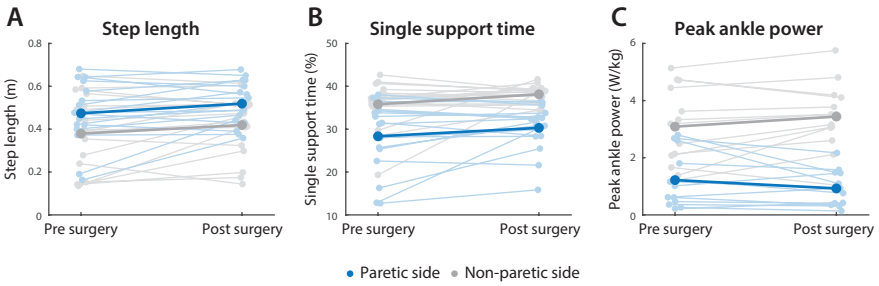


Figure 1. Step length, single support time, and peak ankle power before and after surgery. Panel A shows step length (n=17), panel B single support time (n=17), and panel C peak ankle power (n=13). Individual data points are depicted in light blue for the paretic side, and in light grey for the non-paretic side. Mean values are depicted in dark blue and dark grey for the paretic and non-paretic side, respectively.

Statistically significant differences in kinematics and kinetics were found only at the ankle joint on the paretic side (Figure 2). A statistically significant increase in ankle dorsiflexion on the paretic side was observed after surgery between 0-4% of the gait cycle (p=0.046, Figure 2) and between 66-100% of the gait cycle (p<0.001, Figure 2). Furthermore, internal ankle plantar flexion moment on the paretic side significantly decreased after surgery between 10-12% (p=0.02, Figure 2) and 18-39% of the gait cycle (p<0.001, Figure 2).

Figure 3 illustrates the relationships between post-surgical changes in gait speed and corresponding changes in stride length, cadence, step length, and peak ankle power. Significant correlations were observed between all determinants and post-surgical changes in gait speed, with R^2 values of 0.91 for stride length, 0.53 for cadence, 0.85 and 0.82 for step length on the paretic and non-paretic sides, respectively, and 0.59 and 0.48 for peak ankle power on the paretic and non-paretic sides, respectively.

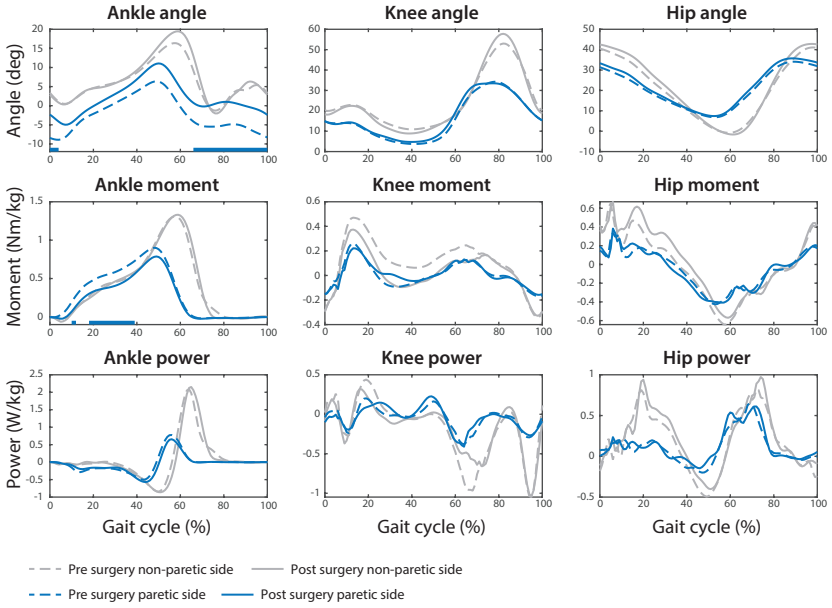


Figure 2. Kinematics and kinetics of the hip, knee, and ankle before and after surgery. Kinematics (n=17) and kinetics (n=13) of the paretic side are shown in blue, whereas the gray lines indicate the non-paretic side. Statistically significant differences are shown by blue bars for the paretic side and grey bars for the non-paretic side.

Plantar pressure measurements

Plantar pressure measurements could be performed in 19 participants. However, three were excluded due to incomplete information on foot shape that prevented normalization. As a result, post-surgical plantar pressure measurements from 16 participants with stroke were compared to those of healthy controls.

Figure 4 presents the normalized plantar pressure patterns of people with stroke post surgery and healthy controls. Lower plantar pressures were found underneath the metatarsal heads in people with stroke compared to healthy controls ($p<0.017$, Figure 4C), whereas slightly higher plantar pressures were found underneath the midfoot and lesser toes in people with stroke compared to healthy controls ($p<0.017$, Figure 4C).

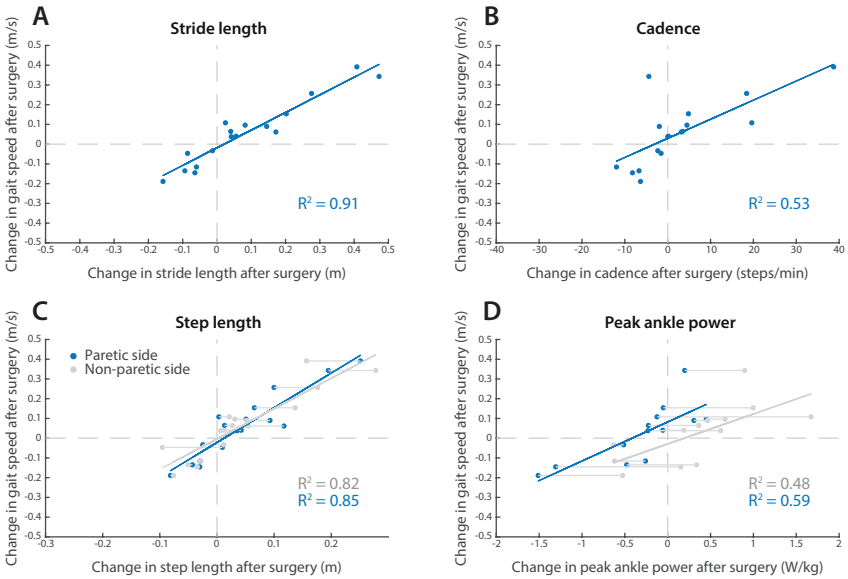


Figure 3. Postoperative changes in gait parameters in relation to changes in gait speed. For stride length (A), cadence (B), step length (C), and peak ankle power (D), the relationships between postoperative changes and changes in gait speed are shown. Coefficients of determination (R^2) are provided for each linear regression analysis. For step length and peak ankle power, each participant is represented by a pair of connected blue and grey dots, corresponding to the values for the paretic and non-paretic sides.

Compared to healthy controls, a slightly more medial center of pressure position in people with stroke post surgery was found between 1-3% (mean Δ: 2.2±0.4%, p=0.047) and 82-93% (mean Δ: 6.6±1.5%, p=0.01) of the stance phase. In contrast, a slightly more lateral center of pressure position in people with stroke was found between 20-31% (mean Δ: 1.7±0.1%, p=0.01) and 57-72% (mean Δ: 3.9±0.3%, p=0.003) of the stance phase (Figure 5).

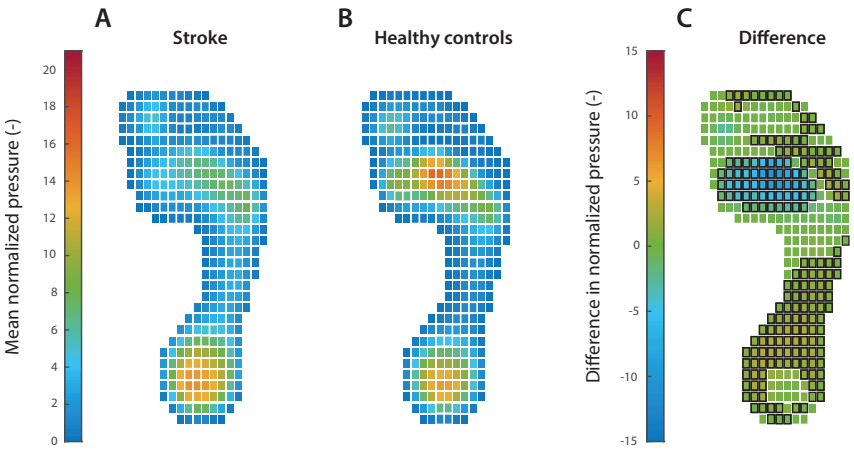


Figure 4. Plantar pressure patterns of people with stroke post surgery and healthy controls. In the two most left panels (A and B), mean normalized plantar pressure patterns are presented for people with stroke post surgery and for healthy controls. In the right panel (C), the difference in mean normalized plantar pressure pattern between people with stroke and healthy controls is illustrated, with positive values indicating higher pressure and negative values indicating lower pressure in people with stroke compared to healthy controls. A black border around a pixel indicates a statistically significant difference ($p<0.017$).

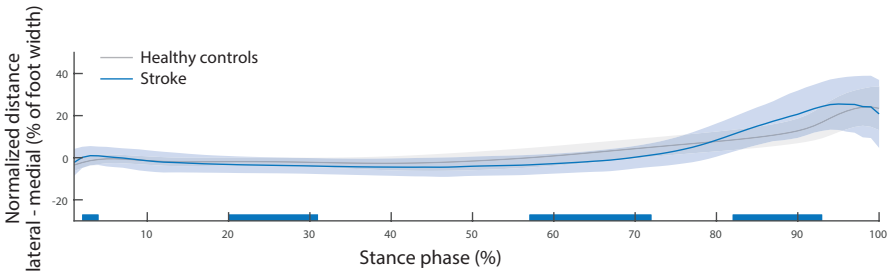


Figure 5. Medio-lateral center of pressure trajectories of people with stroke post surgery and healthy controls. The center of pressure ± standard deviation in medio-lateral direction is shown, with the y-axis representing the normalized distance from the foot's medio-lateral center, expressed as a percentage of foot width. Statistically significant differences are indicated by the blue bars.

Discussion

Consistent with our first hypothesis, reconstructive surgery for pes equinovarus resulted in increased ankle dorsiflexion on the paretic side. In line with our second hypothesis, changes in gait speed were significantly associated with changes in peak ankle power on the non-paretic side. However, such a significant association was also found for the paretic side. The significant interaction between the effects of surgery and body side in peak ankle power revealed that, on the one hand, improved peak ankle power on the non-paretic side coincided with increased gait speed, while, on the other hand, reduced peak ankle power on the paretic side was associated with reduced gait speed. Supporting our third hypothesis, only small differences in medio-lateral center of pressure trajectory were observed between people with stroke after surgery and healthy controls.

The correction of pes equinovarus may improve foot loading on the paretic side, which has been linked to increased paretic step length [5, 6], increased gait speed [5, 6, 9], and enhanced ankle push-off power on the non-paretic side as observed in previous research [5]. However, in the current study, only trends towards improvement of paretic step length and non-paretic peak ankle power were found, while gait speed did not significantly increase. This may be explained by a relatively high pre-surgical self-selected gait speed of the participants in the present study (0.61±0.34 m/s), leaving relatively little room for improvement. Indeed, as previously reported by our group, only individuals with a relatively low initial gait speed demonstrated meaningful improvements after surgery [4].

To address pes equinus deformity, tarsal arthrodesis was frequently combined with a form of Achilles tendon lengthening. This resulted in an increase in ankle dorsiflexion on the paretic side at initial contact and during swing, which is consistent with previous research studying the effect of Achilles tendon lengthening to correct pes equinus [5, 9]. Furthermore, a reduction in internal ankle plantar flexion moment on the paretic side was observed during the stance phase. This finding likely indicates that surgical correction of pes equinus reduces ankle plantar flexion during stance, leading to a posterior shift of the center of pressure. Consequently, the ground reaction force passes closer to the ankle joint center, reducing the moment arm and, in turn, the external dorsiflexion moment. This may explain the observed reduction in internal plantar flexion moment. This interpretation is consistent with previous literature showing that excessive plantar flexion during the loading response and midstance is associated with an increased internal plantar flexion moment [16].

Despite the absence of a significant group level improvement in gait speed, post-surgical changes in gait speed were significantly associated with changes in stride length, cadence, step length on both sides, and peak ankle power on both sides. The association between post-surgical improvements in gait speed and increased peak ankle power on the non-paretic side is consistent with previous research [5] and suggests push-off on the non-paretic side may be suppressed pre surgery as a response to impaired prepositioning and loading capacity of the paretic foot and ankle due to equinovarus deformity. After surgical correction, such latent ankle push-off power on the non-paretic side may be enabled, leading to increased step length and gait speed in some participants. This beneficial effect, however, seems to be counterbalanced by a significant association between diminished gait speed and reduced peak ankle power on the paretic side in other participants. Together, these effects lead to a significant interaction between the effects of surgery and body side at group level, leaving the net effects of surgery on gait speed and ankle power non-significant.

A decrease in peak ankle power on the paretic side has previously been reported in a retrospective study examining the effects of a form of Achilles tendon lengthening in 177 people with stroke [5]. In people with stroke, loss of plantar flexor strength is common [17] and lengthening the plantar flexors may (further) weaken these muscles [17, 18]. In the current study, five participants showed a post-surgical decrease in peak paretic ankle power of more than 0.25 W/kg. Of these, only one had undergone gastrocnemius fasciotomy, while the other four did not undergo any form of pes equinus correction. Accordingly, the observed reduction in peak ankle power in the present study is likely not attributable to the surgical correction of pes equinus, but rather to other factors such as post-surgical immobilization during the casting phase. As ankle push-off is the main source of propulsion during gait, optimal use of available push-off power on both sides of the body is crucial for efficient gait [19]. Therefore, after surgical equinovarus correction, we recommend dedicated gait rehabilitation aimed at optimizing ankle push-off power on the paretic side to at least its pre-surgical level. Additionally, gait rehabilitation should aim to optimize ankle push-off power on the non-paretic side to the level expected for the person's body weight and age.

Previous work has demonstrated a lateralized center of pressure trajectory in people with pes equinovarus [8]. Compared to the substantial differences reported in that study, only small differences in medio-lateral center of pressure trajectory were observed between the participants with stroke and healthy controls in the current study. These minor differences can directly be explained by the surgical intervention, involving arthrodesis of the talonavicular or more tarsal joints as principal procedure.

This intervention induces stabilization of the foot in the frontal plane by correction of pes varus deformity, resulting in normalization of the medio-lateral plantar pressure distribution. Conversely, the mean normalized plantar pressure patterns of people with stroke post surgery still showed some differences from those of healthy controls, more specifically lower pressure underneath the metatarsal heads and higher pressure underneath the midfoot and lesser toes. These differences may be attributable to a lower walking speed in people with stroke, as it has been reported that a reduced walking speed results in increased pressure underneath the midfoot and decreased pressure underneath the first to third metatarsal heads [20].

As this study is an extension of our previous work [4], it shares the same limitation of using a repeated measures study design rather than a randomized clinical trial. However, in our previous work, we demonstrated that potential time effects influencing post-surgical outcomes were minimal, as no differences in outcomes were observed during a 4-month baseline period [4]. Furthermore, nonspecific time effects were unlikely, given that all participants were in the chronic phase after stroke. Therefore, it can be assumed that the observed changes in outcomes over time are attributable to the surgical intervention. Another limitation concerns the use of a normalization method for plantar pressure patterns that was not feasible for individuals with severe deformity. Consequently, it was impossible to compare pre- and post-surgical plantar pressure patterns of people with stroke. Although previous research has demonstrated significant deviations in the plantar pressure patterns of people with pes equinovarus [8], the inability to analyze pre-surgical plantar pressure patterns may have led to inaccurate assumptions about these plantar pressure patterns and, consequently, the effects of surgery. Therefore, future research should focus on creating a normalization method that could be applied to all plantar pressure patterns, even in the absence of complete foot shape information due to severe deformity. Using marker positions as inputs for the normalization process may offer a viable approach [21]. A further limitation is that gait biomechanics could be analyzed for less than half of all participants, primarily because many of them were unable to walk barefoot prior to surgery. Consequently, the true beneficial effects of the surgical equinovarus correction most likely exceed those reported in the current study, as 79% of our participants who were unable to walk barefoot before surgery were able to do so afterwards, indicating substantial barefoot gait improvements as well.

Conclusion

This study revealed an increase in ankle dorsiflexion and a decrease in ankle plantar flexion moment on the paretic side after surgical correction of pes equinovarus in people with chronic stroke. Improved gait speed after surgery was associated with

increased ankle push-off power on the non-paretic side, while decreased gait speed was associated with reduced ankle push-off power on the paretic side. Furthermore, surgery resulted in a normalized medio-lateral center of pressure trajectory. The findings of this study may help optimize pre-operative counseling in clinical practice. Moreover, they may provide guidance for optimizing gait rehabilitation after surgery.

References

[1] Patrick JH, Keenan MAE. Gait analysis to assist walking after stroke. Lancet. 2007;369(9558):256-7. https://doi.org/10.1016/S0140-6736(07)60124-X.

[2] Verdie C, Daviet JC, Borie MJ, Popielarz S, Munoz M, Salle JY, et al. Epidemiology of varus equinus one year after a first cerebral hemisphere stroke. Ann Readapt Med Phys. 2004;47(2):81-6. https://doi.org/10.1016/j.annrmp.2003.10.005.

[3] Nonnekes J, Benda N, Van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.

[4] Bloks BE, Keijsers NLW, Louwerens JWK, Donken CCMA, Geurts ACH, Nonnekes J. Surgical correction of pes equinovarus in people with stroke: Effects on personalized goal attainment and balance and gait capacity. Arch Phys Med Rehabil. 2026;107(6):1364-70. https://doi.org/10.1016/j.apmr.2026.01.015.

[5] Carda S, Bertoni M, Zerbinati P, Rossini M, Magoni L, Molteni F. Gait changes after tendon functional surgery for equinovarus foot in patients with stroke: Assessment of temporo-spatial, kinetic, and kinematic parameters in 177 patients. Am J Phys Med Rehabil. 2009;88(4):292-301. https://doi.org/10.1097/PHM.0b013e318198b593.

[6] Nonnekes J, Kamps M, den Boer J, van Duijnhoven H, Lem F, Louwerens JWK, et al. Tarsal fusion for pes equinovarus deformity improves gait capacity in chronic stroke patients. J Neuroeng Rehabil. 2019;16:102. https://doi.org/10.1186/s12984-019-0572-2.

[7] de l'Escalopier N, Voisard C, Michaud M, Moreau A, Jung S, Tervil B, et al. Evaluation methods to assess the efficacy of equinovarus foot surgery on the gait of post-stroke hemiplegic patients: A literature review. Front Neurol. 2022;13:1042667. https://doi.org/10.3389/fneur.2022.1042667.

[8] Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Plantar pressure in relation to hindfoot varus in people with unilateral upper motor neuron syndrome. J Foot Ankle Res. 2024;17(3):e12041. https://doi.org/10.1002/jfa2.12041.

[9] Giannotti E, Merlo A, Zerbinati P, Prati P, Masiero S, Mazzoli D. Safety and long-term effects on gait of hemiplegic patients in equinovarus foot deformity surgical correction followed by immediate rehabilitation: A prospective observational study. Eur J Phys Rehabil Med. 2019;55(2):169-75. https://doi.org/10.23736/S1973-9087.18.05290-5.

[10] Keijsers NLW. Plantar pressure analysis. The science of footwear. CRC press; 2012. pp. 377-408.

[11] Zeni Jr JA, Richards JG, Higginson JS. Two simple methods for determining gait events during treadmill and overground walking using kinematic data. Gait Posture. 2008;27(4):710-4. https://doi.org/10.1016/j.gaitpost.2007.07.007.

[12] Keijsers NLW, Stolwijk NM, Nienhuis B, Duysens J. A new method to normalize plantar pressure measurements for foot size and foot progression angle. J Biomech. 2009;42(1):87-90. https://doi.org/10.1016/j.jbiomech.2008.09.038.

[13] Pataky TC, Vanrenterghem J, Robinson MA. Zero-vs. one-dimensional, parametric vs. non-parametric, and confidence interval vs. hypothesis testing procedures in one-dimensional biomechanical trajectory analysis. J Biomech. 2015;48(7):1277-85. https://doi.org/10.1016/j.jbiomech.2015.02.051.

[14] Keijsers NLW, Stolwijk NM, Louwerens JWK, Duysens J. Classification of forefoot pain based on plantar pressure measurements. Clin Biomech. 2013;28(3):350-6. https://doi.org/10.1016/j.clinbiomech.2013.01.012.

[15] Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Quantitative assessment of plantar pressure patterns in relation to foot deformities in people with hereditary motor and sensory neuropathies. J Neuroeng Rehabil. 2023;20:65. https://doi.org/10.1186/s12984-023-01172-1.

[16] Perry J, Burnfield JM, Gronley JK, Mulroy SJ. Toe walking: Muscular demands at the ankle and knee. Arch Phys Med Rehabil. 2003;84(1):7-16. https://doi.org/10.1053/apmr.2003.50057.

[17] Waters RL, Perry J, Garland D. Surgical correction of gait abnormalities following stroke. Clin Orthop Relat Res. 1978;(131):54-63. https://doi.org/10.1097/00003086-197803000-00008.

[18] Delp SL, Zajac FE. Force-and moment-generating capacity of lower-extremity muscles before and after tendon lengthening. Clin Orthop Relat Res. 1992;(284):247-59. https://doi.org/10.1097/00003086-199211000-00035.

[19] Winter DA. Energy generation and absorption at the ankle and knee during fast, natural, and slow cadences. Clin Orthop Relat Res. 1983;(175):147-54. https://doi.org/10.1097/00003086-198305000-00021.
[20] Rosenbaum D, Hautmann S, Gold M, Claes L. Effects of walking speed on plantar pressure patterns and hindfoot angular motion. Gait Posture. 1994;2(3):191-7. https://doi.org/10.1016/0966-6362(94)90007-8.
[21] Stebbins JA, Harrington ME, Giacomozzi C, Thompson N, Zavatsky A, Theologis TN. Assessment of sub-division of plantar pressure measurement in children. Gait Posture. 2005;22(4):372-6. https://doi.org/10.1016/j.gaitpost.2004.10.004.



Chapter 8

Summary and general discussion

Summary

The aim of this thesis was to investigate the management of post-stroke gait impairments. The first part of this thesis focused on the assessment of pes equinovarus and stiff knee gait, while in the second part of this thesis, treatment of pes equinovarus was evaluated.

Part I: Assessment of post-stroke gait impairments

Pes equinovarus is one of the most prevalent motor deficits after stroke, often resulting in substantial gait impairments, including reduced foot clearance during the swing phase and decreased stability during the stance phase. Effective clinical management requires thorough assessment of the deformity, including determination of the presence of pes equinus and pes varus throughout the phases of the gait cycle. The detection of pes varus may be performed by multi-segment foot modeling or plantar pressure measurements. In chapter 2, the discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus in people with neurological gait impairments was investigated. In this cross-sectional study comprising 50 participants, hindfoot angles calculated according to four multi-segment foot models were compared with clinical examination of the hindfoot position in the frontal plane at initial contact and midstance. The findings demonstrated a limited capacity of multi-segment foot models to accurately discriminate between hindfoot varus and valgus. This suggests that absolute angles obtained from multi-segment foot model measurements warrant cautious interpretation.

Pes varus can also be assessed using plantar pressure measurements. In chapter 3, plantar pressure patterns were examined in people with unilateral upper motor neuron syndrome (UMNS), including stroke. For a retrospective cohort comprising plantar pressure data of 49 people with UMNS and 586 healthy controls, the ratio between the plantar contact area of the affected and non-affected foot was assessed. Furthermore, normalized plantar pressure patterns and center of pressure trajectories were evaluated. The study revealed spatial and temporal alterations in plantar pressure patterns in individuals with UMNS and dynamic or persistent hindfoot varus deformity, as determined by clinical examination. Based on the discriminative capacity of the evaluated plantar pressure outcome measures, the medio-lateral center of pressure trajectory was proposed as outcome measure for scientifically evaluating surgical interventions targeting hindfoot varus.

Another common gait deviation after stroke is limited knee flexion during the swing phase, often referred to as “stiff knee gait”. For effective clinical management of stiff knee gait, accurately identifying the underlying cause is essential. In chapter 4, a case

series was described, reporting three-dimensional (3D) instrumented gait data from four people with stroke. In clinical practice, rectus femoris spasticity is often intuitively assumed to be the cause of stiff knee gait when rectus femoris activity is observed during the swing phase. However, chapter 4 revealed that rectus femoris activity during swing may be compensatory rather than the cause of limited knee flexion. In the described case series, rectus femoris activity during swing preceded or coincided with knee flexion acceleration, instead of a decelerated knee flexion that one would expect in the case of rectus femoris spasticity. In addition, it coincided with hip flexion motion, while push-off power at the ankle joint was diminished. Taken together, these results suggest that, in these cases, diminished ankle push-off power may have been the primary cause of stiff knee gait, with rectus femoris activity during swing acting as a compensatory mechanism to increase hip (and subsequently knee) flexion. Hence, this chapter highlights the complexity of determining the contribution of rectus femoris spasticity to stiff knee gait in people with stroke, and suggests that biomechanical measures of pre-swing may serve as valuable clinical markers for the assessment of stiff knee gait.

In chapter 5, the role of pre-swing biomechanics on the occurrence of stiff knee gait was further explored. Specifically, based on 3D gait data of 20 healthy controls walking at four different speeds, a predictive model was developed aimed at assessing the contribution of ankle push-off and hip pull-off to knee flexion during swing. The findings demonstrated that pre-swing ankle push-off and hip pull-off together explained a substantial part (58%) of the variance in peak knee flexion among healthy controls. Comparing gait data of 122 people with stroke to the predictive model revealed that, in 65% of participants, peak knee flexion during swing was explained by ankle push-off and hip pull-off. This underscores that inadequate pre-swing biomechanics is a key factor to consider in the evaluation of stiff knee gait, suggesting that assessment should not focus exclusively on rectus femoris spasticity as underlying cause.

Part II: Treatment of pes equinovarus

In the second part of this thesis, the effect of reconstructive surgery for pes equinovarus was evaluated. More specifically, the effect of fusion of one or more tarsal joints, often in combination with a form of Achilles tendon lengthening, was investigated. In chapter 6, the effect of the surgical intervention on personalized goal attainment, balance capacity, and gait speed was investigated in 46 people with stroke. The prospective observational cohort study showed clinically relevant improvements in personalized goal attainment in 78% of participants following surgery. The most frequently reported category of personalized goals was “Improving standing or walking barefoot”, with more than half of the participants reporting at

least one goal in this category. Among these, 88% demonstrated a clinically meaningful improvement of these goals. Furthermore, a clinically meaningful improvement in balance capacity was observed in 52% of participants. A considerable improvement in barefoot gait capacity was observed as well, with 79% of the participants who were not able to walk barefoot before surgery showing the ability to do so afterwards. Additionally, participants with a relatively low gait speed prior to surgery demonstrated increased gait speed after surgery, whereas those with higher baseline gait speed did not show such improvement.

In chapter 7, the effect of reconstructive surgery for pes equinovarus deformity on gait capacity was further investigated by analyzing 3D instrumented gait data and plantar pressure patterns within the same prospective observational cohort as reported on in chapter 6. The findings showed a normalized medio-lateral center of pressure trajectory, indicating correction of pes varus, accompanied by increased ankle dorsiflexion, reflecting correction of pes equinus. Improvements in gait speed following surgery were related to increased push-off power at the ankle joint on the non-paretic side, together with increases in stride length, cadence, and step length on both the paretic and non-paretic sides. In contrast, the slight decrease in gait speed observed in some participants was found to be associated with a reduction in ankle push-off power on the paretic side. In conclusion, part II of this thesis demonstrated that corrective tarsal arthrodesis for pes equinovarus is an effective intervention for people with stroke, improving personalized goal attainment, balance capacity, and barefoot gait capacity.

General discussion

In the following section, I will discuss the findings of this thesis by reflecting on its two parts. First, I will address the assessment of post-stroke gait impairments, as investigated in the first part of this thesis. In particular, I will discuss the value of outcome measures for assessing pes varus and stiff knee gait by reflecting on key quality criteria for outcome measures. Thereafter, I will discuss the treatment of post-stroke gait impairments, focusing on potential directions for future research and outlining clinical recommendations.

Assessment of post-stroke gait impairments

Effective management of post-stroke gait impairments requires comprehensive assessment of motor deficits and their resulting gait deviations. Such assessments rely on the use of appropriate outcome measures, which can inform the development of individualized treatment plans, help predict which patients are likely to benefit

from specific interventions, and enable the evaluation of treatment effectiveness [1-3]. The value of a specific outcome measure for clinical practice or research depends on its ability to meaningfully reflect the motor deficit or gait deviation being assessed. This is determined by three key quality criteria for outcome measures, shown in Figure 1. The first criterion is *validity*, reflecting the extent to which an outcome measure accurately assesses the phenomenon it is intended to measure [1, 4, 5]. Second, an outcome measure should be *reliable*, which means it consistently produces similar results with little random error [4, 5]. The third key quality criterion for outcome measures is *sensitivity*, defined as the ability to detect meaningful differences both between individuals and within individuals across time. In cross-sectional studies, *sensitivity* is similar to discriminative capacity, i.e. the ability to detect differences between individuals [3, 6]. In longitudinal studies, *sensitivity* is similar to responsiveness, i.e. the ability to detect changes over time within individuals [1, 4, 5, 7]. *Sensitivity* is influenced by both *validity* and *reliability*, but it is also affected by possible floor or ceiling effects and by the level of measurement precision, i.e. the number of distinct levels a measure comprises [1, 5, 8]. In addition to the three key quality criteria for outcome measures, two additional criteria are essential for effective implementation in clinical practice or scientific research. The first is *interpretability*, which refers to the degree to which an outcome measure is meaningful and easily understood [5]. The second criterion is *feasibility*, reflecting the degree to which the measurement can be conducted without requiring excessive efforts or resources, or imposing discomfort or burden on either participants or assessors [5].

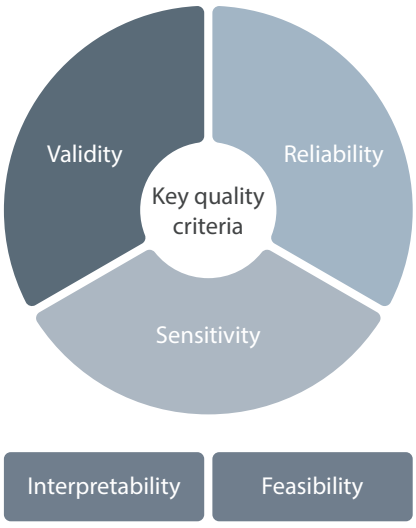


Figure 1. Quality criteria for outcome measures.

In the next section, the quality criteria for outcome measures will be discussed in relation to the assessment of pes equinovarus and stiff knee gait. For pes equinovarus, a current challenge in its assessment lies in determining the presence of pes varus throughout the gait cycle. Methods commonly employed to assess pes varus, including multi-segment foot modeling, biplanar fluoroscopy, plantar pressure measurements, and close-up video recordings, will be reviewed considering the quality criteria mentioned. In the assessment of stiff knee gait, the primary challenge is to identify the most important motor deficit(s) contributing to the gait deviation. In chapter 5 of this thesis, a predictive model was introduced for assessing the influence of inadequate pre-swing biomechanics on stiff knee gait. This model will be discussed in relation to the quality criteria outlined above.

Assessment of pes varus

Multi-segment foot modeling

Three-dimensional instrumented gait analysis is commonly used in both clinical practice and research to assess post-stroke gait impairments [9]. By incorporating a multi-segment foot model into the instrumented gait analysis, pes varus can be assessed through measurement of the frontal plane hindfoot angle with respect to the shank. In terms of *feasibility*, instrumented gait analysis generally requires considerable time for data collection and analysis. However, adding a multi-segment foot model to assess hindfoot varus requires only a few additional markers on the calcaneus and shank. Therefore, when a multi-segment foot model is used solely to evaluate hindfoot motion, the extra time for marker placement and data analysis is minimal, leaving *feasibility* largely unaffected.

Regarding the *validity*, *reliability*, and consequently the *sensitivity* of multi-segment foot models, two primary sources of error associated with 3D instrumented gait analysis should be considered. The first source of error arises from the motion capture system itself, which can introduce inaccuracies in the obtained marker coordinates and, consequently, in the measured joint angles [10]. These errors include systematic errors, such as those caused by image distortion, and random errors, for example due to imprecision in converting marker images into image points [10]. Nonetheless, with proper set-up and calibration, motion capture systems are capable of providing *valid* and *reliable* marker data for measuring (foot) kinematics [9, 11]. The second source of error associated with 3D instrumented gait analysis arises from the use of skin markers placed on anatomical landmarks. These skin markers are used to define segments that are intended to represent the underlying bony anatomy. However, because palpable anatomical landmarks on the foot are limited, individual bones are often grouped into functional segments, which reduces the anatomical specificity and limits the *interpretability* of the measurements. An important consideration

when using a multi-segment foot model to assess pes varus is that skin markers cannot be attached to the talus because of its anatomical location, which prevents direct tracking of talus motion and, consequently, talonavicular joint rotation. Comprehensive assessment of pes varus deformity requires not only measuring calcaneal varus, but also assessing rotations at the talonavicular joint, since a varus position of the calcaneus results from supination of the entire foot, and the talonavicular joint accounts for most of this supination motion [12]. As assessment of talonavicular joint rotation cannot be achieved through multi-segment foot models, their *interpretability* for clinical applications, such as (surgical) treatment planning, is limited. Furthermore, the use of skin markers introduces the risk of marker misplacement, which leads to errors in segment orientation and, consequently, in the joint angles measured [13]. Marker misplacement typically introduces a systematic offset in the absolute joint angles measured, while its effect on the measured kinematic waveform and the range of motion remains limited [14]. As the hindfoot is a relatively small segment with small inter-maker distances, small errors in marker placement lead to relatively large errors in hindfoot angle. Marker placement can be complicated by irregular bony landmarks and variable soft tissue layers covering these landmarks [13]. This challenge is particularly pronounced in cases of ankle-foot deformities, where accurate marker placement becomes more difficult. Previous research on marker misplacement reported a sensitivity of up to 1.6°/mm for hindfoot markers, meaning a 1 mm misplacement can produce a 1.6° error in the frontal plane hindfoot angle [15]. Given that marker placement variability of 0.4-5.3 mm has been reported for hindfoot markers [16], such misplacements inevitably lead to clinically relevant angular errors. A potential approach to reduce the error introduced by marker placement is to guide marker placement by a weight-bearing computed tomography (CT) scan [11, 13]. However, this approach involves radiation exposure and, thus, will be challenging to implement in routine clinical practice due to its limited *feasibility*. Besides marker misplacement, marker-based gait analysis also introduces errors due to soft tissue artifacts, which occurs when skin markers move relative to the underlying bones [17]. As soft tissue artifacts occur at frequencies similar to the bone movement that is intended to measure, distinguishing between them is very hard [17]. Previous research has shown that errors due to soft tissue artifacts can be substantial, with the lateral malleolus marker shifting more than 1 cm relative to the underlying bone morphology during foot movements [18-20]. Furthermore, soft tissue artifacts were found to cause errors of up to 2° in hindfoot angle in the frontal plane across various foot positions [15, 19].

Several studies have evaluated the impact of these sources of error by studying the overall *validity* and *reliability* of multi-segment foot models [21, 22]. Only a few studies directly investigated the *validity* of multi-segment foot models, as this is

challenging due to ethical considerations and the need for a sophisticated experimental setup [21]. A study using bone mounted marker data as the gold standard demonstrated a mean frontal plane hindfoot angle difference of 2.3° over the stance phase [23]. Another study assessed *validity* by comparing a multi-segment foot model to biplanar fluoroscopy measurements and found a root mean squared error of 3.6° [24]. Regarding the *reliability* of multi-segment foot models, several studies have been conducted [21, 22]. However, only a few studies have examined the *reliability* in clinical populations, and just one study has focused specifically on adults with ankle-foot deformities [21]. This study found that, for the hindfoot angle relative to the shank in the frontal plane, variability between sessions and between therapists was twice as large as the variability observed between repeated walking trials within the same measurement session [14]. Standard deviations between trials were less than 3°, whereas the range of hindfoot motion relative to the shank in the frontal plane ranged from 6.9° to 24.1° among participants [14]. Although these findings on *validity* and *reliability* may seem acceptable, chapter 2 of this thesis revealed limited *sensitivity* of multi-segment foot models in detecting inter-subject differences in hindfoot varus/valgus angle, which is an essential requirement for their use in clinical practice and research.

In conclusion, although multi-segment foot models provide a *feasible* addition to regular 3D instrumented gait analysis, their *validity* and *reliability*, and consequently their *sensitivity* to detect meaningful differences in absolute hindfoot varus/valgus angles seem to remain limited. Furthermore, the *interpretability* of the outcomes is complex, as multi-segment foot models do not allow tracking of the talus, which is important for a comprehensive assessment of pes varus deformity. Therefore, although their impact on clinical decision making has not yet been studied, this impact is likely to be limited. Consequently, multi-segment foot models will probably be replaced by techniques that provide more *reliable, valid*, and *sensitive* measurements of pes varus, with reduced complexity in *interpretation*. Since the main limitations of multi-segment foot models stem from the use of skin markers, methods that do not rely on skin markers may serve as an alternative for assessing pes varus during gait.

Biplanar fluoroscopy

An alternative method for assessing pes varus that does not require skin markers is biplanar fluoroscopy, a recently developed technique that enables dynamic tracking of foot bones. Unlike multi-segment foot models, biplanar fluoroscopy allows assessment of the motion of all foot joints, including the talonavicular joint, thereby increasing *interpretability*. Moreover, biplanar fluoroscopy measurements are not affected by soft tissue artifacts or skin marker misplacement.

The technique involves capturing radiographic image sequences from two X-ray sources simultaneously [25-27]. In addition, a static CT scan is required to obtain 3D bone volumes [27]. The CT data and the radiographic images are combined through scientific rotoscoping, a process in which the CT-derived 3D bone model is aligned with its two-dimensional (2D) projections on the two radiographic images [27]. This provides the 3D positions and orientations of the bones over time. To date, biplanar fluoroscopy has been used in several studies investigating (hind)foot kinematics [28-33], including research involving patient populations such as individuals with chronic ankle instability, flat foot, and pes cavus [34].

Currently, the primary limitation to the *validity* and *reliability* of biplanar fluoroscopy is the partly or fully manual scientific rotoscoping process, which introduces human errors [27]. Nonetheless, studies examining the *validity* and *reliability* of biplanar fluoroscopy in measuring (hind)foot kinematics showed good results, with errors less than 1 mm and 1° in position and rotation of the (hind)foot, respectively, for both static and dynamic trials [25-27, 35, 36]. However, its *sensitivity* to detect meaningful differences between or within subjects has yet to be determined. Additionally, up to now, data analysis remains relatively complex and time-consuming, which limits the *feasibility* of this technique, particularly for its use in clinical practice [34]. The development of automated data processing software could address this issue and may also further improve its *validity* and *reliability* [27, 34]. Another factor that is currently restricting the *feasibility* of biplanar fluoroscopy is its requirement of a costly and sophisticated radiographic setup, which is not yet widely accessible [11]. Furthermore, although exposure is limited [26], the technique involves radiation, which is undesirable, especially for applications requiring multiple follow-up measurements.

Taken together, while the current complexity of the technique seems to outweigh its added value, particularly for its use in clinical practice, technological advancements hold promise for improving its *feasibility* in the future. Nonetheless, its impact on clinical decision making remains to be established, and this impact should justify the associated complexity of the technique. In complex ankle-foot deformities where contractures, spasticity, and muscle weakness play a significant role, this method may hold promise to provide valuable guidance for surgical treatment planning in future clinical practice. However, in less complex cases, for example when muscle weakness is the only contributing factor, the technique's complexity may outweigh its potential benefit.

Plantar pressure measurements

A more *feasible* alternative for assessing pes varus during gait is plantar pressure measurement, which provides information on loading of the plantar surface [37-39]. As shown in chapter 3 of this thesis, the medio-lateral center of pressure trajectory is particularly associated with pes varus deformity and may therefore be used to identify pes varus during the stance phase of gait. However, although there is a significant association between plantar pressure and (hind)foot posture, plantar pressure measurements cannot be used to directly measure the hindfoot varus/valgus angle. Instead, it provides data related to the varus/valgus angle, such as the medio-lateral center of pressure trajectory, but these measures only provide an indirect impression of the exact hindfoot position, making *interpretation* more challenging. This limitation may also explain why, in chapter 3 of this thesis, the plantar pressure parameters, including the medio-lateral center of pressure trajectory, did not yield a high *sensitivity* for identifying hindfoot varus.

The *validity*, *reliability* and *sensitivity* of plantar pressure measures depend on the technical specifications of the pressure plate that is used, including its spatial resolution, sampling frequency, and pressure sensor sensitivity [40-42]. Insufficient values for one or more of these parameters may result in information loss and a distorted representation of the plantar pressure pattern. Previous studies have shown good test-retest *reliability* for most outcome measures obtained from plantar pressure measurements, including those related to the medio-lateral center of pressure trajectory [39, 43-45]. This test-retest *reliability* reflects both the random errors introduced by the measurement system and the natural variability of an individual's plantar pressure pattern across repeated tests. However, the *reliability* varied by foot region, with higher *reliability* in areas that typically bear more load, such as the central forefoot, and lower *reliability* in less loaded regions, such as the medial midfoot and toes [39, 43, 44]. The number of trials collected within a session also affected the observed *reliability*, with at least three measurements per session generally considered sufficient to ensure good consistency when the three measurements are averaged [46]. Regarding *validity*, research has demonstrated that plantar pressure parameters are associated with foot posture, with planus feet exhibiting a more medially deviated center of pressure, and cavus feet showing a more laterally deviated center of pressure [37]. This was also demonstrated in chapter 3 of this thesis and in our previous study on plantar pressure patterns in people with cavovarus deformity caused by hereditary motor and sensory neuropathy (HMSN) [47].

In terms of *feasibility*, plantar pressure measurements are relatively fast and easy to perform. Nonetheless, the normalization procedure as used in chapter 3 of this thesis cannot be applied in cases of severe ankle-foot deformity. To address this limitation,

anthropometrically measured foot length and width could be used as input parameters for the normalization method, allowing the plantar pressure patterns to be scaled accordingly. This further development of the normalization method could enhance the *feasibility* of plantar pressure measurements for their use in clinical populations with severe ankle-foot deformities.

In summary, plantar pressure measurement currently seems to be the most *feasible* technique for quantifying foot function during gait, providing *valid* and *reliable* outcomes. As such, it may serve as a valuable complement to standard 3D instrumented gait analysis by providing more detailed information about the ankle-foot complex. However, plantar pressure measurement provides only an indirect measure of hindfoot varus, making clinical *interpretation* challenging. Consequently, its actual contribution to clinical decision-making remains to be determined.

Close-up video recordings

The techniques described above all provide a quantitative measure of pes varus during gait, which is essential for research purposes. However, all three techniques require specialized equipment and quite some level of expertise for proper operation and analysis. In clinical practice, this may not always be *feasible*. A qualitative method for assessing pes varus involves observational gait analysis through close-up video recordings of the hindfoot during gait. This method has the advantage of high *feasibility* and *interpretability*, as it is easy and fast to perform and does not require specialized equipment or expertise to operate it. As for its *validity,* close-up video recordings do not directly reveal the movements of the underlying bone structures, which could lead to misinterpretation, particularly in the presence of excessive callus formation, which can occur with ankle-foot deformities. Moreover, the *validity* and *reliability* of this method are strongly dependent on video quality [48]. Thereby, the angle of the 2D video recording relative to the hindfoot is crucial for obtaining accurate measurements. To minimize optical errors, the camera must be positioned with its optical axis perpendicular to the imaging plane [48, 49]. Video quality also depends on proper lighting, camera stability, frame rate, and resolution [48, 50]. In addition, image size is important, ensuring the required portion of the individual is within the camera's field of view, which can be a challenge in frontal plane measurements, as the subject size decreases in the case of overground walking [48, 50].

Overall, video based gait observation provides qualitative, 2D information on hindfoot posture during gait, offering an important complement to standard 3D instrumented gait analysis for assessing pes varus. Although it does not provide quantitative information on foot function, this technique may still offer sufficient

insight for evaluating pes varus in clinical practice. For research purposes, however, quantitative outcome measures remain necessary. Markerless motion capture, an emerging gait analysis technique, may provide a solution by enabling 3D quantification of joint angles from 2D video recordings [51-53]. However, the value of this technique for evaluating pes varus has yet to be determined.

Assessment of stiff knee gait

In contrast to the assessment of pes (equino)varus deformity, the main challenge in assessing stiff knee gait does not lie in detecting the gait deviation, i.e. the reduction in knee flexion during swing, as this can accurately be quantified by 3D instrumented gait analysis [9, 54]. Rather, the challenge lies in identifying the underlying motor deficit of this gait deviation. Stiff knee gait may result from several underlying motor deficits, including spasticity of the quadriceps muscles and diminished ankle push-off and/or hip pull-off. Consequently, accurate identification of the underlying motor deficit(s) in each individual is essential for effective management. In chapter 5 of this thesis, a predictive model was developed to assess the influence of reduced ankle push-off and hip pull-off on knee flexion during swing. By determining the contribution of inadequate pre-swing biomechanics to stiff knee gait on an individual level, this model holds the potential to improve treatment selection by determining whether inadequate pre-biomechanics should be a target of treatment. However, before the model can be applied in clinical practice, its *validity*, *reliability*, and *sensitivity* to detect meaningful differences should first be established. Examining its *validity* could be accomplished by evaluating the effects of interventions targeting quadriceps spasticity in a cohort of individuals with stroke. A retrospective cohort design would be suitable, as multiple clinical trials on these interventions have already been performed, allowing the use of existing data without imposing additional burden on participants. Within such trials, each participant should have received both intramuscular botulinum toxin and placebo injections in a randomized order, with peak knee flexion, ankle push-off, and hip pull-off assessed using instrumented gait analysis before and after each injection. The predicted treatment effects from the model, defined as the difference between the actual peak knee flexion angle and the peak knee flexion angle predicted based on ankle push-off and hip pull-off, should be compared to the actual observed treatment effects to evaluate the performance of the predictive model. Using statistical measures including the Pearson correlation coefficient and root mean squared error, the difference between the predicted and observed change in swing phase peak knee flexion can be examined to indicate *validity*. Furthermore, the model's *reliability* can be evaluated using Bland-Altman analysis of the two baseline measurements prior to injection. The *sensitivity* of the model to detect meaningful differences should be evaluated by calculating the model's positive and negative predictive values [55]. A high positive

predictive value would imply that most people predicted to achieve an improvement in knee flexion following spasticity treatment actually attained such improvement. Here, improvement would be defined as an increase in knee flexion exceeding the minimal clinically important difference (MCID) for sagittal plane knee range of motion (ROM) on the affected side in people with chronic stroke, reported to be 8.5° [54].

After examining the *validity*, *reliability*, and *sensitivity*, the *feasibility* and *interpretability* of the model should be enhanced by developing a stepwise assessment algorithm incorporating the predictive model to serve as a guideline for clinical practice. The initial step of this assessment algorithm should involve the evaluation of gait speed. As individuals with a low gait speed are unlikely to present quadriceps spasticity, as this is typically triggered by rapid knee flexion, it is presumed that insufficient pre-swing biomechanics is the primary cause of stiff knee gait in these people. The second step should involve the evaluation of ankle and hip work using 3D instrumented gait analysis. Individuals with a low value for combined ankle and hip work are unlikely to exhibit quadriceps spasticity as well, because of the lack of rapid hip and knee flexion during pre-swing. Accordingly, also in these people, it is presumed that insufficient pre-swing biomechanics is the primary cause of stiff knee gait. The final step of the assessment algorithm should consist of the predictive model as presented in chapter 5 of this thesis, determining whether inadequate pre-swing biomechanics contributes to stiff knee gait.

Treatment of post-stroke gait impairments

Following the assessment of post-stroke gait deviations and their underlying motor deficits, treatment should focus on improving gait capacity. Gait capacity improvements can be achieved through restitution or substitution of function, through reconstruction of impaired body functions and structures, and/or through applying external mobility aids. Restitution of function refers to the spontaneous neurological repair processes following stroke that lead to restoration of motor selectivity and coordination of the affected trunk and extremity muscles [56]. Typically, restitution of function occurs shortly after stroke and levels off within the first ten weeks [57]. Gait capacity can also be improved through substitution of function, which can be achieved by physical training. This promotes motor learning of compensatory strategies to overcome motor deficits [58]. To ensure effectiveness, it is essential that physical training is both task-specific and context-specific [58, 59]. The extent to which gait capacity can be restored through restitution and substitution of function is limited and depends on the severity of the initial sensorimotor deficits [60]. Further gait capacity improvements can be accomplished through the use of external mobility aids, such as assistive devices (e.g. a crutch or a walker) and/or

orthotic devices (e.g. ankle-foot orthosis (AFO) or orthopedic footwear). Importantly, in the chronic phase post stroke, gait capacity improvements can also be achieved by reconstruction of impaired body functions and structures, such as surgical procedures to restore a more normal ankle-foot alignment.

According to the theoretical framework as presented in Figure 2 of the general introduction of this thesis, gait independence, gait adaptability, and the gait pattern after stroke are influenced by trunk control, standing balance control, and motor control of both the paretic and non-paretic leg [61]. Gait independence and gait adaptability can both be improved by enhanced standing balance control. Research has shown that, in the chronic phase after stroke, standing balance control can be improved by physical training [59, 62]. According to the theoretical framework, this enhanced standing balance control can be achieved through improved trunk control. In the chronic phase after stroke, core stability exercises have shown to improve trunk control [63], most likely by enhanced innervation of the axial muscles through descending neurons from the unaffected hemisphere. In addition to improved trunk control, standing balance control, and consequently gait independence and gait adaptability, can be enhanced through substitution by the non-paretic leg. Since the non-paretic leg is primarily controlled by the unaffected hemisphere, it can compensate for the motor deficits of the paretic leg. This notion is supported by studies showing that functional recovery of gait is not necessarily related to improved motor control of the paretic leg [64, 65]. Through training, the non-paretic leg can be used more effectively and efficiently for standing balance control and during gait [65], which implies an inherent increase in motor control asymmetry [66]. Lastly, gait capacity improvements can be achieved by reduction of the "learned non-use" phenomenon, which refers to the disuse of latent motor control of the paretic leg [67]. Because restitution is only possible in the acute and early subacute phases after stroke, training in the chronic phase generally does not restore motor control of the paretic leg. This means that, for individuals with pes equinovarus or stiff knee gait, targeted training interventions are generally insufficient to improve the gait pattern. Indeed, research provides no conclusive evidence that training consistently improves paretic leg pre-swing biomechanics [68], highlighting the difficulty in treating stiff knee gait when it is caused by reduced ankle push-off and/or hip pull-off. However, there is evidence that high-intensity exercise training and combined strength and cardiorespiratory training can improve motor function of the paretic leg [59], which suggests a reduction of learned non-use. In line with this, some studies have reported improvements in pre-swing biomechanics following training [68], suggesting that if stiff knee gait arises from learned non-use of paretic push-off and/or pull-off, training interventions may be effective. Therefore, rehabilitation should focus on reducing this learned non-use phenomenon, if present. An important area for future research

may be the development of methods for identifying post-stroke individuals exhibiting learned non-use, who may benefit from targeted training aimed at improving the gait pattern. In the absence of learned non-use, efforts to improve the gait pattern should focus on reconstruction of paretic leg motor functions through medical-technical interventions. The next section will discuss such interventions for the treatment of pes equinovarus deformity.

Treatment of pes equinovarus

Medical-technical interventions for the treatment of pes equinovarus include conservative treatment options such as orthopedic footwear, which can promote plantigrade loading, offer support during standing and walking, and relieve pain. Furthermore, in the case of dynamic pes equinus due to weakness of the ankle dorsiflexors or muscular imbalance around the ankle joint, an AFO can aid in improving the gait pattern. However, a key limitation of these conservative interventions is that they cannot be worn all the time, which poses problems during activities such as showering or walking to the bathroom at night. Furthermore, when a structural deformity is present, such interventions may have limited effectiveness. Consequently, reconstruction of impaired body functions and structures through corrective surgery represents an important treatment option to consider.

Chapters 6 and 7 of this thesis revealed that reconstructive tarsal arthrodesis for pes equinovarus deformity is an effective intervention for improving personalized goal attainment, balance capacity, and gait capacity. This indicates that tarsal arthrodesis represents an important treatment option with the potential to improve quality of life. As an alternative to tarsal arthrodesis, pes equinovarus can surgically be corrected through soft tissue procedures, such as split anterior tibial tendon transfer and release or transfer of the posterior tibial tendon, often combined with Achilles tendon lengthening. These are procedures widely reported in the literature [69, 70]. Nevertheless, in our experience, the outcomes of soft tissue interventions can be difficult to predict in people with stroke, due to the presence of spasticity and muscle weakness. Alternatively, tarsal bone fusion procedures have been proposed in the literature [69, 71, 72]. However, no research has been done on how the outcomes of corrective tarsal arthrodesis compare to those of other surgical interventions for pes equinovarus, including soft tissue procedures. Therefore, further research is needed that directly compares tarsal arthrodesis with soft tissue procedures to develop and refine evidence-based guidelines for the surgical management of pes equinovarus deformity in people with stroke.

Another direction for future research is to evaluate the long-term effects of reconstructive tarsal arthrodesis for pes equinovarus deformity. Chapters 6 and 7 of

this thesis present an evaluation of the effects of surgical intervention after one year. However, post-surgical rehabilitation may not have been fully completed in every person at this point in time. Consequently, assessing long-term outcomes after an additional year could yield further insights into the effects of surgical intervention. Additionally, it would be valuable to investigate whether the improvements in personalized goal attainment, balance capacity, and gait capacity as observed one year after surgery persist in the very long term, for example, up to ten years after surgery. As tarsal arthrodesis leads to redistribution of forces within the foot, resulting in increased midfoot loading, it may cause long-term degenerative joint changes [73-75]. However, it remains unclear whether these changes have an impact on functional mobility. To gain further insight into the effects of surgical intervention, evaluating its cost-effectiveness compared to conservative treatment would be valuable. Although surgery is a more expensive treatment, it likely reduces the need for additional conservative treatment in the long term [76]. Future studies should therefore conduct a cost-utility analysis to compare healthcare costs and quality-adjusted life years (QALYs), reflecting life expectancy and quality of life, for surgical versus conservative interventions [77]. The EuroQOL five dimensions questionnaire (EQ-5D) could be used to measure health-related quality of life (HRQOL), which covers aspects of well-being and illness [78]. Such research would offer greater insight into the effectiveness of surgical intervention for treating post-stroke pes equinovarus.

When targeted training before surgery yields little gait capacity improvement, surgical restoration of a more normal ankle-foot alignment may create a new window of opportunity for gait training. Reduced pain, improved stability, and increased confidence may allow for higher training intensity, which could, in turn, contribute to further improvement of gait capacity. Additionally, improved loading of the paretic foot resulting from enhanced plantigrade support may open new avenues for training and improving standing balance control and gait adaptability. In people with stroke, falls and fall-related injuries are prevalent and can have serious consequences for functional mobility [79]. One of the major risk factors for falls in people with stroke is a reduced or delayed ability to execute reactive stepping responses to balance perturbations [79, 80]. Prior research has shown that people with stroke have impaired reactive stepping responses, particularly for side steps towards the paretic side [81]. In the presence of pes equinovarus deformity, reactive stepping is even more compromised as a result of diminished plantigrade support. Because falls in people with stroke commonly occur towards the paretic side [79], improving reactive side step responses of the paretic leg is crucial for fall prevention. Previous research demonstrated that perturbation-based balance training could enhance the reactive stepping response of the paretic leg [82]. Following reconstructive surgery for pes

equinovarus, the resulting improvement in plantigrade support of the paretic foot may offer a favorable window for such perturbation-based balance training, facilitating further gains in stepping responses. Future studies on the development and evaluation of training programs aimed at improving standing balance control and gait adaptability after surgical correction of pes equinovarus are therefore recommended. Previously, we initiated a study on gait training in individuals with pes equinovarus following stroke at our rehabilitation clinic, but we had to discontinue it due a lack of participant enrollment, which was largely attributable to long travel distances to the clinic in combination with the reduced functional capacity observed after stroke. Implementing standing balance and gait adaptability training in a home-based setting may therefore enhance feasibility and, in turn, lead to improved balance and gait outcomes.

Clinical recommendations

Chapters 6 and 7 of this thesis revealed meaningful improvements in personalized goal attainment, balance capacity, and gait capacity following reconstructive surgery for pes equinovarus. However, the effectiveness of surgical correction is contingent upon five critical factors in clinical practice, in line with the Dutch clinical practice guidelines for the treatment of cerebral and/or spinal spasticity in adults ("Richtlijn cerebrale en/of spinale spasticiteit bij volwassenen") [83]. The first critical factor is that both conservative treatment options and surgical treatment options should be carefully considered for the management of pes equinovarus to determine the most optimal treatment strategy for each individual. Importantly, surgical intervention should not be viewed as a last resort following the failure of all conservative treatments, which remains common in many clinical practices. As surgery can have lasting beneficial effects and may reduce the need for additional conservative interventions, it is recommended that surgical options are always considered, even early in the chronic phase after stroke. A second critical factor for the effectiveness of surgical intervention is that it is carried out in a specialized clinic with acknowledged expertise. Preferably, corrective surgery for pes equinovarus in people with stroke is centralized in a small number of specialized, dedicated centers in the Netherlands, as this promotes the concentration of knowledge and skills. Moreover, this allows for easier monitoring of complications and failures, which can contribute to continuous improvement of the quality of surgical interventions and peri- and postoperative care. In each specialized center, consideration of surgery should always be discussed by an experienced multidisciplinary team, including a rehabilitation physician, orthopedic surgeon, and physiotherapist. The third critical factor for surgical effectiveness is that patients are properly informed about what to expect from the intervention, as well as the associated risks of complications, enabling shared decision-making. This should involve personalized goal setting prior to surgery,

ensuring that the chosen (surgical) treatment option aligns with the pre-defined personalized goals. Chapter 6 of this thesis revealed that personalized goals were attained in most participants after surgery, whereas improvements in balance and gait capacity were particularly evident in people who were severely affected. For people with severe leg motor impairments, surgical intervention would therefore often be recommendable. For people who are less severely affected, the indication for surgery depends even more on their personalized goals. If the primary aim of people with a relatively high gait speed is to further improve their gait speed, other interventions, such as external mobility aids, may be more appropriate. But if the goal is to improve standing or walking barefoot, reduce pain, or improve walking on uneven surfaces, surgical intervention will probably yield meaningful improvements and should therefore be considered. As a fourth critical factor, a comprehensive assessment of post-stroke gait deviations and their underlying motor deficits should be conducted prior to surgery, enabling informed clinical decision-making that considers the most appropriate type of surgical intervention, possible alternative conservative treatment options, and a risk analysis of possible adverse effects of surgery on non-operated body parts. Moreover, the effects of the surgical intervention should be evaluated, with consideration of the personalized goals established prior to surgery. The fifth and final critical factor for surgical effectiveness is the provision of appropriate post-surgical rehabilitation. Close collaboration between the treatment team in the specialized clinic and experienced healthcare therapists in the community should facilitate care closer to patients' homes. In order to maximize treatment outcomes, gait and balance training should be a key component of this post-surgical rehabilitation.

References

[1] Roach KE. Measurement of health outcomes: Reliability, validity and responsiveness. J Prosthet Orthot. 2006;18(6):8-12. https://doi.org/10.1097/00008526-200601001-00003.

[2] Kirshner B, Guyatt G. A methodological framework for assessing health indices. J Chronic Dis. 1985;38(1):27-36. https://doi.org/10.1016/0021-9681(85)90005-0.

[3] Hankins M. How discriminating are discriminative instruments? Health Qual Life Outcomes. 2008;6:36. https://doi.org/10.1186/1477-7525-6-36.

[4] Gadotti IC, Vieira ER, Magee DJ. Importance and clarification of measurement properties in rehabilitation. Braz J Phys Ther. 2006;10(2):137-46. https://doi.org/10.1590/S1413-35552006000200002.

[5] Fitzpatrick R, Davey C, Buxton MJ, Jones DR. Evaluating patient-based outcome measures for use in clinical trials. Health Technol Assess. 1998;2(14)

[6] Guyatt GH, Kirshner B, Jaeschke R. Measuring health status: What are the necessary measurement properties? J Clin Epidemiol. 1992;45(12):1341-5. https://doi.org/10.1016/0895-4356(92)90194-r.

[7] García de Yébenes Prous MJ, Salvanés FR, Ortells LC. Responsiveness of outcome measures. Reumatol Clin. 2008;4(6):240-7. https://doi.org/10.1016/S1699-258X(08)75545-1.

[8] Brock KA, Goldie PA, Greenwood KM. Evaluating the effectiveness of stroke rehabilitation: Choosing a discriminative measure. Arch Phys Med Rehabil. 2002;83(1):92-9. https://doi.org/10.1053/apmr.2002.27348.

[9] Bonato P, Feipel V, Corniani G, Arin-Bal G, Leardini A. Position paper on how technology for human motion analysis and relevant clinical applications have evolved over the past decades: Striking a balance between accuracy and convenience. Gait Posture. 2024;113:191-203. https://doi.org/10.1016/j.gaitpost.2024.06.007.

[10] Chiari L, Della Croce U, Leardini A, Cappozzo A. Human movement analysis using stereophotogrammetry: Part 2: Instrumental errors. Gait Posture. 2005;21(2):197-211. https://doi.org/10.1016/j.gaitpost.2004.04.004.

[11] Leardini A, Stebbins J, Hillstrom H, Caravaggi P, Deschamps K, Arndt A. ISB recommendations for skin-marker-based multi-segment foot kinematics. J Biomech. 2021;125:110581. https://doi.org/10.1016/j.jbiomech.2021.110581.

[12] Lundberg A. Kinematics of the ankle and foot: In vivo roentgen stereophotogrammetry. Acta Orthop Scand. 1989;60:Suppl.233. https://doi.org/10.3109/17453678909154185.

[13] Della Croce U, Leardini A, Chiari L, Cappozzo A. Human movement analysis using stereophotogrammetry: Part 4: Assessment of anatomical landmark misplacement and its effects on joint kinematics. Gait Posture. 2005;21(2):226-37. https://doi.org/10.1016/j.gaitpost.2004.05.003.

[14] Deschamps K, Staes F, Bruyninckx H, Busschots E, Matricali GA, Spaepen P, et al. Repeatability of a 3D multi-segment foot model protocol in presence of foot deformities. Gait Posture. 2012;36(3):635-8. https://doi.org/10.1016/j.gaitpost.2012.04.007.

[15] Schallig W, van den Noort JC, Piening M, Streekstra GJ, Maas M, van der Krogt MM, et al. The Amsterdam Foot Model: A clinically informed multi-segment foot model developed to minimize measurement errors in foot kinematics. J Foot Ankle Res. 2022;15:46. https://doi.org/10.1186/s13047-022-00543-6.

[16] Deschamps K, Roosen P, Birch I, Dingenen B, Bruyninckx H, Desloovere K, et al. A novel device for standardizing marker placement at the calcaneus. J Am Podiatr Med Assoc. 2014;104(1):43-9. https://doi.org/10.7547/0003-0538-104.1.43.

[17] Leardini A, Chiari L, Della Croce U, Cappozzo A. Human movement analysis using stereophotogrammetry: Part 3. Soft tissue artifact assessment and compensation. Gait Posture. 2005;21(2):212-25. https://doi.org/10.1016/j.gaitpost.2004.05.002.

[18] Birch I, Deschamps K. Quantification of skin marker movement at the malleoli and talar heads. J Am Podiatr Med Assoc. 2011;101(6):497-504. https://doi.org/10.7547/1010497.

[19] Schallig W, Streekstra GJ, Hulshof CM, Kleipool RP, Dobbe JGG, Maas M, et al. The influence of soft tissue artifacts on multi-segment foot kinematics. J Biomech. 2021;120:110359. https://doi.org/10.1016/j.jbiomech.2021.110359.

[20] Shultz R, Kedgley AE, Jenkyn TR. Quantifying skin motion artifact error of the hindfoot and forefoot marker clusters with the optical tracking of a multi-segment foot model using single-plane fluoroscopy. Gait Posture. 2011;34(1):44-8. https://doi.org/10.1016/j.gaitpost.2011.03.008.
[21] Leardini A, Caravaggi P, Theologis T, Stebbins J. Multi-segment foot models and their use in clinical populations. Gait Posture. 2019;69:50-9. https://doi.org/10.1016/j.gaitpost.2019.01.022.
[22] Deschamps K, Staes F, Roosen P, Nobels F, Desloovere K, Bruyninckx H, et al. Body of evidence supporting the clinical use of 3D multisegment foot models: A systematic review. Gait Posture. 2011;33(3):338-49. https://doi.org/10.1016/j.gaitpost.2010.12.018.
[23] Nester C, Jones RK, Liu A, Howard D, Lundberg A, Arndt A, et al. Foot kinematics during walking measured using bone and surface mounted markers. J Biomech. 2007;40(15):3412-23. https://doi.org/10.1016/j.jbiomech.2007.05.019.
[24] Kessler SE, Rainbow MJ, Lichtwark GA, Cresswell AG, D'Andrea SE, Konow N, et al. A direct comparison of biplanar videoradiography and optical motion capture for foot and ankle kinematics. Front Bioeng Biotechnol. 2019;7:199. https://doi.org/10.3389/fbioe.2019.00199.
[25] Wang B, Roach KE, Kapron AL, Fiorentino NM, Saltzman CL, Singer M, et al. Accuracy and feasibility of high-speed dual fluoroscopy and model-based tracking to measure in vivo ankle arthrokinematics. Gait Posture. 2015;41(4):888-93. https://doi.org/10.1016/j.gaitpost.2015.03.008.
[26] Cross JA, McHenry BD, Molthen R, Exten E, Schmidt TG, Harris GF. Biplane fluoroscopy for hindfoot motion analysis during gait: A model-based evaluation. Med Eng Phys. 2017;43:118-23. https://doi.org/10.1016/j.medengphy.2017.02.009.
[27] Maharaj JN, Kessler S, Rainbow MJ, D'Andrea SE, Konow N, Kelly LA, et al. The reliability of foot and ankle bone and joint kinematics measured with biplanar videoradiography and manual scientific rotoscoping. Front Bioeng Biotechnol. 2020;8:106. https://doi.org/10.3389/fbioe.2020.00106.
[28] Roach KE, Foreman KB, Barg A, Saltzman CL, Anderson AE. Application of high-speed dual fluoroscopy to study in vivo tibiotalar and subtalar kinematics in patients with chronic ankle instability and asymptomatic control subjects during dynamic activities. Foot Ankle Int. 2017;38(11):1236-48. https://doi.org/10.1177/1071100717723128.
[29] de Asla RJ, Wan L, Rubash HE, Li G. Six DOF in vivo kinematics of the ankle joint complex: Application of a combined dual-orthogonal fluoroscopic and magnetic resonance imaging technique. J Orthop Res. 2006;24(5):1019-27. https://doi.org/10.1002/jor.20142.
[30] Lenz AL, Nichols JA, Roach KE, Foreman KB, Barg A, Saltzman CL, et al. Compensatory motion of the subtalar joint following tibiotalar arthrodesis: An in vivo dual-fluoroscopy imaging study. J Bone Joint Surg Am. 2020;102(7):600-8. https://doi.org/10.2106/JBJS.19.01132.
[31] Campbell KJ, Wilson KJ, LaPrade RF, Clanton TO. Normative rearfoot motion during barefoot and shod walking using biplane fluoroscopy. Knee Surg Sports Traumatol Arthrosc. 2016;24:1402-8. https://doi.org/10.1007/s00167-014-3084-4.
[32] Wang C, Geng X, Wang S, Ma X, Wang X, Huang J, et al. In vivo kinematic study of the tarsal joints complex based on fluoroscopic 3D-2D registration technique. Gait Posture. 2016;49:54-60. https://doi.org/10.1016/j.gaitpost.2016.06.009.
[33] Roach KE, Wang B, Kapron AL, Fiorentino NM, Saltzman CL, Bo Foreman K, et al. In vivo kinematics of the tibiotalar and subtalar joints in asymptomatic subjects: A high-speed dual fluoroscopy study. J Biomech Eng. 2016;138(9):091006. https://doi.org/10.1115/1.4034263.
[34] Ye D, Sun X, Zhang C, Zhang S, Zhang X, Wang S, et al. In vivo foot and ankle kinematics during activities measured by using a dual fluoroscopic imaging system: A narrative review. Front Bioeng Biotechnol. 2021;9:693806. https://doi.org/10.3389/fbioe.2021.693806.
[35] Iaquinto JM, Tsai R, Haynor DR, Fassbind MJ, Sangeorzan BJ, Ledoux WR. Marker-based validation of a biplane fluoroscopy system for quantifying foot kinematics. Med Eng Phys. 2014;36(3):391-6. https://doi.org/10.1016/j.medengphy.2013.08.013.
[36] Ito K, Hosoda K, Shimizu M, Ikemoto S, Kume S, Nagura T, et al. Direct assessment of 3D foot bone kinematics using biplanar X-ray fluoroscopy and an automatic model registration method. J Foot Ankle Res. 2015;8(1):21. https://doi.org/10.1186/s13047-015-0079-4.

[37] Buldt AK, Allan JJ, Landorf KB, Menz HB. The relationship between foot posture and plantar pressure during walking in adults: A systematic review. Gait Posture. 2018;62:56-67. https://doi.org/10.1016/j.gaitpost.2018.02.026.
[38] Arzehgar A, Nia RGNN, Hoseinkhani M, Masoumi F, Sayyed-Hosseinian S-H, Eslami S. An overview of plantar pressure distribution measurements and its applications in health and medicine. Gait Posture. 2025;117:235-44. https://doi.org/10.1016/j.gaitpost.2024.12.022.
[39] Rogers A, Morrison SC, Gorst T, Paton J, Freeman J, Marsden J, et al. Repeatability of plantar pressure assessment during barefoot walking in people with stroke. J Foot Ankle Res. 2020;13(1):39. https://doi.org/10.1186/s13047-020-00407-x.
[40] Giacomozzi C, Keijsers N, Pataky T, Rosenbaum D. International scientific consensus on medical plantar pressure measurement devices: Technical requirements and performance. Ann Ist Super Sanita. 2012;48(3):259-71. https://doi.org/10.4415/Ann_12_03_06.
[41] Razak AHA, Zayegh A, Begg RK, Wahab Y. Foot plantar pressure measurement system: A review. Sensors. 2012;12(7):9884-912. https://doi.org/10.3390/s120709884.
[42] Rosenbaum D, Becker HP. Plantar pressure distribution measurements. Technical background and clinical applications. Foot Ankle Surg. 1997;3(1):1-14. https://doi.org/10.1046/j.1460-9584.1997.00043.x.
[43] Xu C, Wen X-X, Huang L-Y, Shang L, Cheng X-X, Yan Y-B, et al. Normal foot loading parameters and repeatability of the Footscan® platform system. J Foot Ankle Res. 2017;10(1):30. https://doi.org/10.1186/s13047-017-0209-2.
[44] Gurney JK, Kersting UG, Rosenbaum D. Between-day reliability of repeated plantar pressure distribution measurements in a normal population. Gait Posture. 2008;27(4):706-9. https://doi.org/10.1016/j.gaitpost.2007.07.002.
[45] Hafer JF, Lenhoff MW, Song J, Jordan JM, Hannan MT, Hillstrom HJ. Reliability of plantar pressure platforms. Gait Posture. 2013;38(3):544-8. https://doi.org/10.1016/j.gaitpost.2013.01.028.
[46] Deepashini H, Omar B, Paungmali A, Amaramalar N, Ohnmar H, Leonard J. An insight into the plantar pressure distribution of the foot in clinical practice: Narrative review. Pol Ann Med. 2014;21(1):51-6. https://doi.org/10.1016/j.poamed.2014.03.003.
[47] Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Quantitative assessment of plantar pressure patterns in relation to foot deformities in people with hereditary motor and sensory neuropathies. J Neuroeng Rehabil. 2023;20:65. https://doi.org/10.1186/s12984-023-01172-1.
[48] Fatone S, Stine R. Capturing quality clinical videos for two-dimensional motion analysis. J Prosthet Orthot. 2015;27(1):27-32. https://doi.org/10.1097/JPO.0000000000000051.
[49] Areblad M, Nigg BM, Ekstrand J, Olsson KO, Ekström H. Three-dimensional measurement of rearfoot motion during running. J Biomech. 1990;23(9):933-40. https://doi.org/10.1016/0021-9290(90)90358-A.
[50] Michelini A, Eshraghi A, Andrysek J. Two-dimensional video gait analysis: A systematic review of reliability, validity, and best practice considerations. Prosthet Orthot Int. 2020;44(4):245-62. https://doi.org/10.1177/0309364620921290.
[51] Mündermann L, Corazza S, Andriacchi TP. The evolution of methods for the capture of human movement leading to markerless motion capture for biomechanical applications. J Neuroeng Rehabil. 2006;3:6. https://doi.org/10.1186/1743-0003-3-6.
[52] Lam WWT, Tang YM, Fong KNK. A systematic review of the applications of markerless motion capture (MMC) technology for clinical measurement in rehabilitation. J Neuroeng Rehabil. 2023;20:57. https://doi.org/10.1186/s12984-023-01186-9.
[53] Wade L, Needham L, McGuigan P, Bilzon J. Applications and limitations of current markerless motion capture methods for clinical gait biomechanics. PeerJ. 2022;10:e12995. https://doi.org/10.7717/peerj.12995.
[54] Guzik A, Drużbicki M, Wolan-Nieroda A, Turolla A, Kiper P. Estimating minimal clinically important differences for knee range of motion after stroke. J Clin Med. 2020;9(10):3305. https://doi.org/10.3390/jcm9103305.
[55] Monaghan TF, Rahman SN, Agudelo CW, Wein AJ, Lazar JM, Everaert K, et al. Foundational statistical principles in medical research: Sensitivity, specificity, positive predictive value, and negative predictive value. Medicina. 2021;57(5):503. https://doi.org/10.3390/medicina57050503.

[56] Bernhardt J, Hayward KS, Kwakkel G, Ward NS, Wolf SL, Borschmann K, et al. Agreed definitions and a shared vision for new standards in stroke recovery research: The stroke recovery and rehabilitation roundtable taskforce. Int J Stroke. 2017;12(5):444-50. https://doi.org/10.1177/1747493017711816.
[57] Kwakkel G, Kollen B, Twisk J. Impact of time on improvement of outcome after stroke. Stroke. 2006;37(9):2348-53. https://doi.org/10.1161/01.STR.0000238594.91938.1e.
[58] Langhorne P, Bernhardt J, Kwakkel G. Stroke rehabilitation. Lancet. 2011;377(9778):1693-702. https://doi.org/10.1016/S0140-6736(11)60325-5.
[59] Veerbeek JM, van Wegen E, van Peppen R, van der Wees PJ, Hendriks E, Rietberg M, et al. What is the evidence for physical therapy poststroke? A systematic review and meta-analysis. PloS One. 2014;9(2):e87987. https://doi.org/10.1371/journal.pone.0087987.
[60] Prabhakaran S, Zarahn E, Riley C, Speizer A, Chong JY, Lazar RM, et al. Inter-individual variability in the capacity for motor recovery after ischemic stroke. Neurorehabil Neural Repair. 2007;22(1):64-71. https://doi.org/10.1177/1545968307305302.
[61] Weerdesteyn VGM, Nonnekes JH, Fleuren JFM, Buurke JH, van Duijnhoven HJR, Geurts ACH. Herstel van loopvaardigheid na een CVA - een theoretisch framework. Nederlands Tijdschrift voor Revalidatiegeneeskunde. 2022;44(1):8-11.
[62] van Duijnhoven HJR, Heeren A, Peters MAM, Veerbeek JM, Kwakkel G, Geurts ACH, et al. Effects of exercise therapy on balance capacity in chronic stroke: Systematic review and meta-analysis. Stroke. 2016;47(10):2603-10. https://doi.org/10.1161/STROKEAHA.116.013839.
[63] Gamble K, Chiu A, Peiris C. Core stability exercises in addition to usual care physiotherapy improve stability and balance after stroke: A systematic review and meta-analysis. Arch Phys Med Rehabil. 2021;102(4):762-75. https://doi.org/10.1016/j.apmr.2020.09.388.
[64] den Otter AR, Geurts ACH, Mulder TH, Duysens J. Gait recovery is not associated with changes in the temporal patterning of muscle activity during treadmill walking in patients with post-stroke hemiparesis. Clin Neurophysiol. 2006;117(1):4-15. https://doi.org/10.1016/j.clinph.2005.08.014.
[65] Buurke JH, Nene AV, Kwakkel G, Erren-Wolters V, IJzerman MJ, Hermens HJ. Recovery of gait after stroke: What changes? Neurorehabil Neural Repair. 2008;22(6):676-83. https://doi.org/10.1177/1545968308317972.
[66] Kwakkel G, Wagenaar RC. Effect of duration of upper- and lower-extremity rehabilitation sessions and walking speed on recovery of interlimb coordination in hemiplegic gait. Phys Ther. 2002;82(5):432-48. https://doi.org/10.1093/ptj/82.5.432.
[67] Taub E, Uswatte G, Mark VW, Morris DM. The learned nonuse phenomenon: Implications for rehabilitation. Eura Medicophys. 2006;42(3):241-55.
[68] Alingh JF, Groen BE, van Asseldonk EHF, Geurts ACH, Weerdesteyn V. Effectiveness of rehabilitation interventions to improve paretic propulsion in individuals with stroke – A systematic review. Clin Biomech. 2020;71:176-88. https://doi.org/10.1016/j.clinbiomech.2019.10.021.
[69] Allart É, Sturbois-Nachef N, Salga M, Rosselin C, Gatin L, Genêt F. Neuro-orthopedic surgery for equinovarus foot deformity in adults: A narrative review. J Foot Ankle Surg. 2022;61(3):648-56. https://doi.org/10.1053/j.jfas.2021.11.012.
[70] Salga M, Gatin L, Deltombe T, Gustin T, Carda S, Marque P, et al. International recommendations to manage poststroke equinovarus foot deformity validated by a panel of experts using delphi. Arch Phys Med Rehabil. 2023;104(3):372-9. https://doi.org/10.1016/j.apmr.2022.07.020.
[71] Nonnekes J, Benda N, van Duijnhoven H, Lem F, Keijsers N, Louwerens JWK, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: A review. JAMA Neurol. 2018;75(6):751-8. https://doi.org/10.1001/jamaneurol.2017.5041.
[72] Martin KD, Jastifer J, Scott D, Grzeskiewicz E. Spastic equinovarus foot deformity. J Am Acad Orthop Surg. 2024;32(16):e785-e94. https://doi.org/10.5435/JAAOS-D-23-01007.
[73] Southwell RB, Sherman FC. Triple arthrodesis: A long-term study with force plate analysis. Foot Ankle. 1981;2(1):15-24. https://doi.org/10.1177/107110078100200103.
[74] So LWN, Kuong EE, To K-TM, Leong JCY, Chow W. Long-term outcome after Lambrinudi arthrodesis: How they're doing after three decades. J Orthop Surg. 2019;27(1):2309499019826492. https://doi.org/10.1177/2309499019826492.

[75] Pell RF, Myerson MS, Schon LC. Clinical outcome after primary triple arthrodesis. J Bone Joint Surg Am. 2000;82(1):47-57. https://doi.org/10.2106/00004623-200001000-00006.
[76] Reddy S, Kusuma S, Hosalkar H, Keenan MA. Surgery can reduce the nonoperative care associated with an equinovarus foot deformity. Clin Orthop Relat Res. 2008;466(7):1683-7. https://doi.org/10.1007/s11999-008-0250-3.
[77] Detsky AS, Naglie IG. A clinician's guide to cost-effectiveness analysis. Ann Intern Med. 1990;113(2):147-54. https://doi.org/10.7326/0003-4819-113-2-147.
[78] Gusi N, Olivares PR, Rajendram R. The EQ-5D health-related quality of life questionnaire. Handbook of Disease Burdens and Quality of Life Measures: Springer; 2010. p. 87-99.
[79] Weerdesteijn V, de Niet M, van Duijnhoven HJR, Geurts ACH. Falls in individuals with stroke. J Rehabil Res Dev. 2008;45(8):1195-214. https://doi.org/10.1682/JRRD.2007.09.0145.
[80] Mansfield A, Inness EL, Wong JS, Fraser JE, McIlroy WE. Is impaired control of reactive stepping related to falls during inpatient stroke rehabilitation? Neurorehabil Neural Repair. 2013;27(6):526-33. https://doi.org/10.1177/1545968313478486.
[81] de Kam D, Roelofs JMB, Bruijnes AKBD, Geurts ACH, Weerdesteyn V. The next step in understanding impaired reactive balance control in people with stroke: The role of defective early automatic postural responses. Neurorehabil Neural Repair. 2017;31(8):708-16. https://doi.org/10.1177/1545968317718267.
[82] van Duijnhoven HJR, Roelofs JMB, den Boer JJ, Lem FC, Hofman R, van Bon GEA, et al. Perturbation-based balance training to improve step quality in the chronic phase after stroke: A proof-of-concept study. Front Neurol. 2018;9:980. https://doi.org/10.3389/fneur.2018.00980.
[83] Federatie Medisch Specialisten. Richtlijn cerebrale en/of spinale spasticiteit bij volwassenen. Available from: https://richtlijnendatabase.nl/richtlijn/cerebrale_en_of_spinale_spasticiteit_bij_volwassenen/cerebrale_en_of_spinale_spasticiteit_-_startpagina.html.

Chapter 9

Nederlandse samenvatting

Summary in Dutch

Nederlandse samenvatting

Mensen die een beroerte hebben doorgemaakt ervaren vaak problemen met lopen. Een veelvoorkomende oorzaak hiervan is een "pes equinovarus", een standsafwijking waarbij de voet in spits- en varusstand staat. Een pes equinovarus leidt vaak tot instabiliteit tijdens het lopen, wat het dagelijks functioneren aanzienlijk kan beïnvloeden. Een ander veelvoorkomend loopprobleem bij mensen die een beroerte hebben doorgemaakt is verminderde knieflexie tijdens de zwaaifase van het lopen, aangeduid als "stiff knee gait". Stiff knee gait kan verschillende onderliggende oorzaken hebben en bemoeilijkt het van de grond houden van de voet tijdens de zwaaifase van het lopen, waardoor het risico op struikelen en vallen toeneemt.

Om loopproblemen na een beroerte effectief te kunnen behandelen is een grondige evaluatie van de loopproblemen essentieel. Het eerste deel van dit proefschrift richt zich op methoden voor het evalueren van loopproblemen na een beroerte, met specifieke focus op de evaluatie van pes equinovarus en stiff knee gait. In het tweede deel van dit proefschrift komt de behandeling van pes equinovarus aan bod, in het bijzonder chirurgische correctie van de standsafwijking.

Deel I: Evaluatie van loopproblemen na een beroerte

Voor een adequate evaluatie van pes equinovarus is het essentieel vast te stellen of en tijdens welke fasen van het lopen de standsafwijking van de voet optreedt. Er bestaan verschillende methoden om een varusstand van de voet tijdens het lopen te detecteren. Eén van deze methoden is het gebruik van een multi-segment voetmodel, dat tijdens een driedimensionale (3D) geïnstrumenteerde gangbeeldanalyse kan worden toegepast om de stand van de achtervoet in het frontale vlak te meten. In **hoofdstuk 2** van dit proefschrift is de discriminatieve capaciteit van multi-segment voetmodellen onderzocht voor het detecteren van varus- en valgusstanden van de achtervoet tijdens het lopen bij mensen met diverse neurologische loopproblemen. De uitkomsten van multi-segment voetmodellen werden vergeleken met een klinische beoordeling van de stand van de achtervoet. De resultaten lieten zien dat de overeenstemming beperkt was, wat duidt op geringe discriminatieve capaciteit van multi-segment voetmodellen voor het detecteren van varus- en valgusstanden van de achtervoet tijdens het lopen.

Een andere methode om een varusstand van de achtervoet te evalueren is door middel van een voetdrukplaat, waarmee de drukverdeling onder de voet tijdens het lopen kan worden gemeten. In **hoofdstuk 3** van dit proefschrift is de drukverdeling onder de voet tijdens het lopen geanalyseerd bij mensen met niet-aangeboren hersenletsel, waaronder een beroerte. De resultaten lieten spatiële en temporele

verschillen in drukverdeling onder de voet zien bij mensen met een varusstand van de achtervoet, ten opzichte van gezonde controlepersonen en ten opzichte van mensen met niet-aangeboren hersenletsel zonder varusstand van de achtervoet. Een varusstand van de achtervoet kon het beste gedetecteerd worden door analyse van het traject van het aangrijpingspunt van de grondreactiekracht ("center of pressure") in medio-laterale richting. Dit suggereert dat deze maat een waardevolle uitkomstmaat zou kunnen zijn om het effect van interventies te evalueren.

Om stiff knee gait effectief te kunnen behandelen is het essentieel om de onderliggende oorzaak van de verminderde knieflexie te achterhalen. Twee veelvoorkomende oorzaken van stiff knee gait zijn spasticiteit van de rectus femoris en een verminderde energieproductie rondom de enkel en heup voorafgaand aan de zwaaifase. In **hoofdstuk 4** van dit proefschrift worden vier casus gepresenteerd met 3D gangbeeldanalyse data van mensen die een beroerte hebben doorgemaakt. De resultaten van deze gangbeeldanalyses lieten zien dat activiteit van de rectus femoris tijdens de zwaaifase van het lopen niet altijd gepaard gaat met een vermindering in knieflexie. Dit suggereert dat rectus femoris activiteit tijdens de zwaaifase van het lopen niet altijd hinderlijke spasticiteit hoeft te zijn, maar ook compensatoir zou kunnen zijn voor het versterken van de heupflexie en daarmee ook de knieflexie. Daarnaast lieten de casus een verminderde energieproductie rondom het enkelgewricht voorafgaand aan de zwaaifase zien. Uit de resultaten werd geconcludeerd dat het interpreteren van rectus femoris activiteit tijdens de zwaaifase van het lopen in relatie tot stiff knee gait complex kan zijn en dat een verminderde energieproductie voorafgaand aan de zwaaifase een belangrijke determinant kan vormen bij de evaluatie van stiff knee gait.

In **hoofdstuk 5** van dit proefschrift is de rol van energieproductie voorafgaand aan de zwaaifase op verminderde knieflexie tijdens het lopen verder onderzocht. Op basis van gangbeeldanalyse data van gezonde deelnemers werd een predictief model ontwikkeld waarmee de knieflexie tijdens de zwaaifase kan worden voorspeld aan de hand van de energieproductie rondom de enkel en heup voorafgaand aan de zwaaifase. Vervolgens werd dit model vergeleken met data van mensen na een beroerte, waarbij in de meerderheid van de gevallen de knieflexie kon worden voorspeld op basis van de energieproductie. Dit suggereert dat de energieproductie rondom de enkel en heup een belangrijke determinant vormt bij de evaluatie van stiff knee gait, en dat klinische evaluatie niet uitsluitend gericht moet zijn op rectus femoris spasticiteit als onderliggende oorzaak.

Deel II: Behandeling van pes equinovarus

In het tweede deel van dit proefschrift werd het effect van chirurgische correctie van pes equinovarus onderzocht bij mensen die een beroerte hebben doorgemaakt. De chirurgische correctie bestond uit een voetwortelartrodese, vaak in combinatie met andere ingrepen, zoals een achillespeesverleging. In **hoofdstuk 6** werd specifiek het effect van chirurgische correctie op persoonlijke doelen, balanscapaciteit, en loopcapaciteit op blote voeten onderzocht. De resultaten lieten klinisch relevante verbeteringen in persoonlijke doelen en balanscapaciteit zien na de chirurgische ingreep. Daarnaast werd er een aanzienlijke verbetering van loopcapaciteit gevonden, aangezien veel mensen die voor de operatie niet op blote voeten konden lopen, dit na de operatie wel konden. Bovendien bleek dat mensen met een relatief lage loopsnelheid voorafgaand aan de operatie vooruit gingen in loopsnelheid na de operatie.

In **hoofdstuk 7** van dit proefschrift werd het effect van chirurgische correctie van pes equinovarus verder onderzocht door de loopcapaciteit nader te bestuderen. Hiervoor werden de resultaten van 3D gangbeeldanalyses en voetdrukmetingen geanalyseerd binnen hetzelfde cohort als in hoofdstuk 6. Na de interventie werd een genormaliseerd traject van het aangrijpingspunt van de grondreactiekracht in medio-laterale richting gevonden, evenals verbeteringen in kinematica en kinetica van het enkelgewricht. Daarnaast gingen verbeteringen in loopsnelheid gepaard met een toename in schredelengte, cadans en staplengte aan zowel de aangedane als niet-aangedane zijde, evenals met een verbeterde afzet rondom de enkel aan de niet-aangedane zijde. Bij mensen die een lichte achteruitgang in loopsnelheid lieten zien na de operatie werd een lichte afname in afzet rondom de enkel aan de aangedane zijde gevonden. Concluderend laat het tweede deel van dit proefschrift zien dat chirurgische correctie van pes equinovarus middels voetwortelartrodese een effectieve behandeling is bij mensen die een beroerte hebben doorgemaakt, met verbeteringen in persoonlijke doelen, balanscapaciteit, en loopcapaciteit op blote voeten.

In **hoofdstuk 8** worden de resultaten van dit proefschrift samengevat en bediscussieerd. Daarbij wordt er specifiek ingegaan op de waarde van verschillende meetmethoden voor het evalueren van pes equinovarus en stiff knee gait. Tevens wordt de behandeling van pes equinovarus besproken, waarbij mogelijke richtingen voor toekomstig onderzoek worden beschreven en aanbevelingen voor de klinische praktijk worden gedaan.

Appendices

Dankwoord | Acknowledgements
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List of publications
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Donders Graduate School
Theses Sint Maartenskliniek

Dankwoord

Het dankwoord. Misschien niet het belangrijkste onderdeel van een proefschrift, maar laten we eerlijk zijn: voor velen is het wel het eerste waar ze naartoe bladeren. En kijk eens aan: je hebt het gevonden!

Allereerst wil ik graag alle deelnemers aan de verschillende studies bedanken. Ondanks dat deelname vaak veel van jullie vroeg, waren jullie bereid om mee te doen, vaak met als motivatie om anderen met vergelijkbare problemen in de toekomst te helpen. Dat vind ik bewonderingswaardig en daar heb ik veel respect voor. Daarnaast wil ik jullie bedanken voor de openheid, het vertrouwen en de vele fijne gesprekken die we tussen de metingen door hebben gevoerd.

Mijn promotieteam: Sander, Noël, Jorik en Jan Willem. Heel erg bedankt voor de oprecht hele leuke en leerzame tijd die ik de afgelopen jaren met jullie heb gehad. Jullie enthousiasme en passie voor onderzoek werken inspirerend en motiverend. Bovendien hebben jullie ieder vanuit jullie eigen expertise en perspectief een unieke en onmisbare bijdrage geleverd aan dit proefschrift. Dankzij jullie kritische blik, begeleiding en betrokkenheid is dit werk geworden wat het nu is.

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Uiteraard wil ik ook de leden van de manuscriptcommissie, prof. dr. I.C.M. van der Geest, prof. dr. J.H.P. Houdijk en dr. M.M. van der Krogt, bedanken voor de tijd en aandacht die zij hebben besteed aan het beoordelen van mijn proefschrift. Ook wil ik prof. dr. V.G.M. Weerdesteyn, dr. E.C. Prinsen en dr. J.F.M. Fleuren bedanken voor hun bereidheid om tijdens mijn verdediging plaats te nemen in de oppositie.

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Als laatste wil ik mijn lieve familie, vrienden en natuurlijk Ties bedanken voor alle liefde, steun en mooie momenten, vaak juist niet gerelateerd aan dit proefschrift. Zonder jullie was dit proefschrift er niet geweest.

About the author



Bente Bloks was born on September 18, 1996, in Utrecht, The Netherlands. After completing her secondary education at O.R.S. Lek & Linge in Culemborg in 2014, Bente enrolled in the Bachelor's programme Human Movement Sciences at the Vrije Universiteit Amsterdam. During her bachelor research project, she studied the time-dependent adaptation of step frequency following perturbations in walking speed. In 2017, Bente obtained her Bachelor's degree and subsequently started the Master's programme Biomedical Engineering at the Delft University of Technology. During her Master's studies, Bente dedicated a year to Project MARCH, a student-led initiative focused on developing an exoskeleton for individuals with spinal cord injury. Within the project, she was responsible for several aspects of the exoskeleton development, including the generation of gait patterns. Bente later completed an internship at Hocoma AG in Switzerland, where she worked in the development department on gait trajectories for the Lokomat robotic gait training device. For her Master's thesis, Bente studied the effects of excessive medio-lateral trunk movements on ankle plantar flexor work during gait in healthy individuals. After earning her Master's degree in 2020, Bente began her PhD at the Sint Maartenskliniek and Radboud university medical center in 2021, where she focused on the management of post-stroke gait impairments.

List of publications

Bloks BE, Nonnekes J, Louwerens JWK, Geurts ACH, Keijsers NLW. Improved gait biomechanics following surgical correction of pes equinovarus in individuals with stroke. *Submitted.*

Bloks BE, van de Wiel LM, Wilders LM, Louwerens JWK, Geurts ACH, Nonnekes J, Keijsers NLW. Discriminative capacity of multi-segment foot models in identifying hindfoot varus and valgus in people with neurological gait impairments. *Submitted.*

Bloks BE, Keijsers NLW, Louwerens JWK, Donken CCMA, Geurts ACH, Nonnekes J. Surgical correction of pes equinovarus in people with stroke: Effects on personalized goal attainment and balance and gait capacity. Arch Phys Med Rehabil. 2026; 107(6):1364-1370.

van Oorschot W, **Bloks B**, Kamphuis J, Pieterse A, Geurts A, Keijsers N, Nonnekes J, Stiff knee gait after stroke: The potential compensatory role of mid-swing rectus femoris activity. Case Rep Neurol Med. 2025; 2025(1):7703081.

Bloks BE, Keijsers NLW, van Oorschot W, Geurts AC, Nonnekes J. Towards a precision rehabilitation approach for post-stroke stiff knee gait. Clin Biomech. 2025; 127: 106587.

Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Plantar pressure in relation to hindfoot varus in people with unilateral upper motor neuron syndrome. J Foot Ankle Res. 2024; 17(3):e12041.

Bloks BE, Wilders LM, Louwerens JWK, Geurts AC, Nonnekes J, Keijsers NLW. Quantitative assessment of plantar pressure patterns in relation to foot deformities in people with hereditary motor and sensory neuropathies. J Neuroeng Rehabil. 2023; 20:65.

Portfolio

Name PhD candidate: Bente E. Bloks
Graduate School: Donders Graduate School

Courses	Organizer	Year	ECTS
Graduate School Day	Donders Graduate School	2021, 2022	0.5
eBROK course	Nederlandse Federatie van Universitair Medische Centra	2021	1.5
Graduate School Introduction Day	Donders Graduate School	2021	0.25
Projectmanagement voor Promovendi	Radboud University	2021	2
Writing Scientific Articles	Radboud University	2022	3
Scientific Integrity Course	Donders Graduate School	2022	0.25
Presenting and Poster Pitching	Radboud University	2022	1.5
Sensorimotor Control and Learning	Radboud University	2022-2023	6
Academic English and Pronunciation	Radboud University	2023	1.5
Design and Illustration	Radboud University	2023	1
Career development workshop "The next step in my career"	Radboud university medical center	2024	1

Lectures, webinars, workshops, other		Organizer	Year
Other	GRAIL operator training	Sint Maartenskliniek	2021
Workshop	Data visualization	Sint Maartenskliniek	2022
Workshop	Communication - "writing for scientists" and "talking to the media"	The Online Scientist	2022
Workshop	PhD Information Event: Getting Started with Adobe Illustrator	Donders	2022
Lecture	Loop Expertise Centrum (LEC) scholingsavond	Sint Maartenskliniek	2022*, 2022*, 2025*
Workshop	Presenteren	Spies & Spreken	2023
Lecture	"Voetdrukmetingen voor het evalueren van voetfunctie tijdens het lopen: van wetenschap naar klinische praktijk"	Voetentraining	2023*

Lectures, webinars, workshops, other		Organizer	Year
Workshop	Introduction course R	Sint Maartenskliniek	2024
Workshops/lectures	Career Event for PhD candidates and postdocs	Radboud university medical center	2024
Webinar	"Theia markerless: van installatie tot klinische toepassing", "Lopen onder druk: voetdrukmeting in de klinische gangbeeldanalyse"	SMALLL	2024, 2025*
Workshop	LinkedIn	Sint Maartenskliniek	2025
Lecture	Image Recognition	Sint Maartenskliniek	2025
Workshop	PhD Information Event: How to Finish Your PhD	Donders Graduate School	2025
Lecture	Presentation physiotherapy unit	Sint Maartenskliniek	2025*
Workshop	Graphical abstracts	Sint Maartenskliniek	2026
Meeting	Research Content Meeting (ReCoMe)	Sint Maartenskliniek	2021-2026
Meeting	Research lunch and lab lunch	Sint Maartenskliniek	2021-2026
Webinar	Online lectures	The Interdisciplinary Consortium for Clinical Movement Sciences & Technology (ICMS)	2022-2025
Workshop/meeting	Patient participation training, workshops, and meetings	Sint Maartenskliniek	2022-2025
Workshop/lecture	Meet the PhD	Sint Maartenskliniek	2022-2025*
Meeting	Topspecialistische Zorg en Onderzoek (TZO) meeting	Sint Maartenskliniek	2022-2026
Meeting	Pitches & Drinks	Sint Maartenskliniek	2023-2026

*=presenter

Conferences and symposia	Role	Location	Year
International Society of Posture and Gait Research (ISPGR) World Congress	Poster presentation	Montreal, Canada	2022
Web conference CMT (HMSN) & HNPP, Spierziekten Nederland	Invited lecture	Online	2022
Symposium Loop Expertise Centrum (LEC)	Attendee	Nijmegen, The Netherlands	2022
Society for Movement Analysis Laboratories in the Low Lands (SMALLL) Congress	Poster presentation	Hasselt, Belgium	2022
Vereniging voor Bewegingswetenschappen Nederland (VvBN) PhD Day	Attendee	Nijmegen, The Netherlands	2022
International Foot and Ankle Biomechanics (i-FAB) Congress	Oral presentation and poster presentation	Bordeaux, France	2023
International Congress on Neurorehabilitation and Neural Repair (NNR)	Invited lecture	Maastricht, The Netherlands	2023
Society for Movement Analysis Laboratories in the Low Lands (SMALLL) Congress	Organizing committee member	Nijmegen, The Netherlands	2023
The TechMed Event: Shaping a Healthy Future	Attendee	Enschede, The Netherlands	2023
The Interdisciplinary Consortium for Clinical Movement Sciences & Technology (ICMS) Annual Event	Poster presentation	Nijmegen, The Netherlands	2023
Annual Meeting of the European Society for Movement Analysis in Adults and Children (ESMAC)	Oral presentation	Oslo, Norway	2024
Symposium Loop Expertise Centrum (LEC)	Invited lecture	Nijmegen, The Netherlands	2024
European Foot & Ankle Society (EFAS) Congress	Poster presentation	Brussels, Belgium	2024
Dutch Congress of Rehabilitation Medicine (DCRM)	Invited lecture	Utrecht, The Netherlands	2024
The Interdisciplinary Consortium for Clinical Movement Sciences & Technology (ICMS) Annual Event	Attendee	Enschede, The Netherlands	2025
Nederlandse Orthopaedische Vereniging (NOV) Jaarcongres	Oral presentation	Utrecht, The Netherlands	2025

Conferences and symposia	Role	Location	Year
International Society of Biomechanics (ISB) Congress	Oral presentation	Stockholm, Sweden	2025
Spierziektecongres	Invited lecture	Arnhem, The Netherlands	2025
Society for Movement Analysis Laboratories in the Low Lands (SMALLL) Congress	Attendee	Rotterdam, The Netherlands	2025

Project supervision	Year	Duration
Bachelor students Biomedical Sciences – Radboud University (3 students)	2022, 2023	6 months
Master student Technical Medicine – University of Twente (1 student)	2022-2023	1 year

Research data management

Ethics and privacy

This thesis is based on the results of research involving human participants, which were conducted in accordance with the ICH-GCP guidelines (Good Clinical Practice). Chapter 2 was part of a larger study for which a statement that the study was not subject to the Dutch Medical Research Involving Human Subjects Act (WMO) was obtained from the recognized Medical Ethics Review Committee "METC Oost-Nederland" (2023-16337). For the study presented in chapters 6 and 7, the recognized Medical Ethics Review Committee "METC Oost-Nederland" had given approval to conduct this study (file number: NL77992.091.21). No medical-ethical approval was required for the studies presented in chapters 3, 4, and 5, as the data used for these studies were previously collected as part of routine clinical care and were used in pseudonymized form.

For chapters 2, 4, 6, and 7, written informed consent was obtained from participants to collect and process their data. For chapters 3 and 5, data were previously collected in the context of healthcare. To ensure responsible reuse of healthcare data, specific informed consent procedures were followed that are aligned with applicable laws, regulations, and the national Code of Conduct for Health Research. These data were used in pseudonymized form. Consent was not obtained for sharing of the (pseudonymized) data for future research for any of the chapters.

According to the Dutch legislation, data collection from electronic patient files was performed by personnel with a treatment relationship with the patients (chapters 3, 4 and 5), and by the researchers upon consent by the study participants (chapters 2, 4, 6, and 7). The privacy of the participants in all studies was warranted by the use of pseudonymization. The pseudonymization key was stored on a secured network drive and was only accessible to the members of the project who needed access to it for their role in the project. The pseudonymization key was stored separately from the research data.

Data collection and storage

Data for chapter 2 were collected through gait laboratory experiments, using a motion capture system (Vicon Motion Systems Ltd., UK). For chapters 3, 4, and 5, data collected previously in the context of healthcare from gait laboratory experiments with a motion capture system (Vicon Motion Systems Ltd., UK) and a plantar pressure plate (RSscan, Belgium) were used. For chapters 6 and 7, data were collected through experiments, including gait laboratory experiments (Vicon Motion Systems Ltd., UK and RSscan, Belgium), clinical tests, and standardized interviews/questionnaires.

Furthermore, data were collected through electronic Case Report Forms (eCRF) of a prospective data collection in Castor EDC. For all chapters, subject characteristics were collected from electronic patient files (HiX) and stored in Castor EDC for chapters 6 and 7 and on a secure department server for chapters 2, 3, 4, and 5. Data were converged to MATLAB (The MathWorks, Inc., USA).

Pseudonymized data were stored and analyzed on the department server and in Castor EDC, which is only accessible by project members working at the Sint Maartenskliniek. These secure storage options safeguard the availability, integrity and confidentiality of the data. Paper (hardcopy) data are stored in cabinets at the department.

Data sharing according to the FAIR principles

Chapters 3, 4, 5, and 6 are published with open access. Chapters 2 and 7 have been submitted for publication and are currently under review.

The data used for this thesis are owned by the Sint Maartenskliniek, not by the Radboudumc. Data are archived by the Sint Maartenskliniek. Data are not suitable for sharing because no informed consent for sharing was obtained for any of the chapters. Questions about the data can be addressed to the secretariat of the Research department of the Sint Maartenskliniek (email address: secretariaat.research@maartenskliniek.nl). All data remain available for 20 years after termination of the studies.

Donders Graduate School

For a successful research Institute, it is vital to train the next generation of scientists. To achieve this goal, the Donders Institute for Brain, Cognition and Behaviour established the Donders Graduate School in 2009. The mission of the Donders Graduate School is to guide our graduates to become skilled academics who are equipped for a wide range of professions. To achieve this, we do our utmost to ensure that our PhD candidates receive support and supervision of the highest quality.

Since 2009, the Donders Graduate School has grown into a vibrant community of highly talented national and international PhD candidates, with over 500 PhD candidates enrolled. Their backgrounds cover a wide range of disciplines, from physics to psychology, medicine to psycholinguistics, and biology to artificial intelligence. Similarly, their interdisciplinary research covers genetic, molecular, and cellular processes at one end and computational, system-level neuroscience with cognitive and behavioural analysis at the other end. We ask all PhD candidates within the Donders Graduate School to publish their PhD thesis in de Donders Thesis Series. This series currently includes over 750 PhD theses from our PhD graduates and thereby provides a comprehensive overview of the diverse types of research performed at the Donders Institute. A complete overview of the Donders Thesis Series can be found on our website: https://www.ru.nl/donders/donders-series.

The Donders Graduate School tracks the careers of our PhD graduates carefully. In general, the PhD graduates end up at high-quality positions in different sectors, for a complete overview see https://www.ru.nl/donders/destination-our-former-phd. A large proportion of our PhD alumni continue in academia (>50%). Most of them first work as a postdoc before growing into more senior research positions. They work at top institutes worldwide, such as University of Oxford, University of Cambridge, Stanford University, Princeton University, UCL London, MPI Leipzig, Karolinska Institute, UC Berkeley, EPFL Lausanne, and many others. In addition, a large group of PhD graduates continue in clinical positions, sometimes combining it with academic research. Clinical positions can be divided into medical doctors, for instance, in genetics, geriatrics, psychiatry, or neurology, and in psychologists, for instance as healthcare psychologist, clinical neuropsychologist, or clinical psychologist. Furthermore, there are PhD graduates who continue to work as researchers outside academia, for instance at non-profit or government organizations, or in pharmaceutical companies. There are also PhD graduates who work in education, such as teachers in high school, or as lecturers in higher education. Others continue in a wide range of positions, such as policy advisors, project managers, consultants, data scientists, web- or software developers, business owners, regulatory affairs specialists,

engineers, managers, or IT architects. As such, the career paths of Donders PhD graduates span a broad range of sectors and professions, but the common factor is that they almost all have become successful professionals.

For more information on the Donders Graduate School, as well as past and upcoming defenses please visit: http://www.ru.nl/donders/graduate-school/phd/

Theses SMK

Haegens, L. (2026) Informed and Activated Patients for Medication Self-Management: eHealth-enhanced Strategies. Radboud University Nijmegen, Nijmegen. The Netherlands.

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