Optimizing ankle foot orthosis stiffness in calf muscle weakness

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GENERAL INTRODUCTION
INTRODUCTION

During human gait, the calf muscles control the internal plantar flexor moment around the ankle and are the main providers of propulsive power for forward progression [1]. In neuromuscular disorders, the calf muscles can be weakened [2], which will compromise both functions during gait. Necessary compensations to a weakened calf will change the gait pattern, leading to reduced balance and limitations in walking ability as demonstrated by a higher walking energy cost and reduced walking speed. These limitations may cause fatigue and restrict daily life functioning [3-5]. To counteract these limitations, an ankle foot orthosis (AFO) can be prescribed in persons with neuromuscular disorders to compensate for calf muscle weakness [6]. Yet, evidence about the effectiveness of AFOs on gait in this population is scarce and reported effects vary largely [7]. Simulations [8] and studies in healthy individuals [9] indicate that the effect of the AFOs on walking energy cost depends on the AFO’s ankle stiffness and that differences in AFO ankle stiffness might explain the reported variation in effect. This thesis focusses on how AFO ankle stiffness influences gait and how optimization of AFO ankle stiffness can improve the effectiveness of orthoses for persons exhibiting calf muscle weakness.

The role of the calf muscles in gait

The calf muscles consist of the medial and lateral gastrocnemius muscles and the soleus muscle. The calf muscles are essential for a stable and efficient gait pattern and have two main functions; (1) providing an internal ankle plantar flexion moment to control the tibia inclination during the stance phase of gait and (2) generating power for forward propulsion needed to sustain walking speed [1]. In the first part of the stance phase, the calf muscles contract eccentrically to restrain ankle dorsiflexion and thereby control the forward progression of the tibia. This allows for a forward displacement of the origin of the ground reaction force (i.e. the center of pressure) along the foot. During late stance, the ground reaction force runs in front of the knee in the sagittal plane causing an external knee extension moment that allows the knee extensor muscles to relax [1, 10, 11]. During terminal stance and pre-swing, the calf muscles contract concentrically to generate a large positive power-burst known as the ankle push-off. During the ankle push-off, more than 50% of the propulsive power needed to walk at a constant speed is generated [12, 13]. This mechanical energy is used to move the center of mass forward and upward and accelerate the leg into the swing.

For a normal and efficient gait pattern, the timing and magnitude of ankle push-off are critical [14, 15]. Normally, ankle push-off starts just before the contralateral leg hits the ground. At this instant, the ankle push-off power provides a small upward acceleration of the center-of-mass, which reduces its downward velocity at the moment of heel-strike.
of the contralateral leg. Consequently, contralateral initial contact occurs with a lower downward velocity meaning less energy is lost at foot collision, which is considered a main contributor to the energy efficiency of gait [16]. The continuation of the ankle power-burst after contralateral initial contact accelerates the leg into swing. This energy is suggested to reduce necessary work (flexion) at the hip and therewith the metabolic energy cost during swing [1, 17].

Gait in persons with calf muscle weakness
As the function of the calf muscles is highly important for walking, weakness of these muscles will severely affect the gait pattern [18]. Calf muscles weakness can become manifest due to a large number of different neuromuscular disorders [19], and this thesis focusses on disorders causing non-spastic calf muscle weakness. The most prevalent neuromuscular disorders causing non-spastic calf weakness are Charcot-Marie-Tooth disease (diagnosed in 1 in every 5.000-10.000 people), spinal muscle atrophy (diagnosed in 1 in every 25.000 people) [20], poliomyelitis and post-polio syndrome (in 1 in every 2.500 people) [21], and traumas of the tibial nerve.

The severity of calf muscle weakness differs largely among individuals as it depends on the diagnosis and the progression of the disorder. Consequently, the functional problems that persons with calf muscle weakness encounter range from very mild to severe. Functionally, persons with calf muscle weakness suffer from diminished walking speed and an increased walking energy cost, already in very mild cases [3, 5]. The increase in walking energy cost causes fatigue, which is a major problem in persons with neuromuscular disorders [22, 23]. In addition, calf muscle weakness negatively impacts gait stability [22], which, together with fatigue, results in reduced physical activity and quality of life [4, 24-26].

The increase in walking energy cost is the consequence of two major gait deviations. First, energy cost increases due to the inability of the calf muscles to restrain the forward rotation of the tibia during stance, which would cause the ankle to move into excessive dorsiflexion [3, 18, 27]. To prevent this, the compensatory reaction is to prevent the ground reaction force to progress forward over the foot. As a result, the ground reaction force also remains posterior of the knee, causing an external knee flexion moment during the entire stance phase [3, 27]. Consequently, the quadriceps muscles have to be activated during the whole stance phase, potentially causing overuse symptoms but also substantially increasing the walking energy cost [28, 29].
Neuromuscular disorders exhibiting non-spastic calf muscle weakness

Worldwide, more than 600 neuromuscular disorders are recognized, which vary in etiology and clinical manifestation [52]. Whether or not the calf muscles are affected and if non-spastic weakness of the calf muscles develops depends on the specific disorder.

Neuromuscular disorders are classified according to the location of the disorder within the nerve-muscle complex, namely: disorders of the motor neurons, disorders of motor nerve roots, disorders of the plexus, disorders of peripheral nerve, disorders of neuromuscular transmission and disorders of the muscle itself [6, 52]. Disorders of the plexus and neuromuscular transmission seldom cause non-spastic calf muscle weakness and are therefore not further introduced. The common clinical characteristics of the other groups are shortly introduced below.

Disorders of the motor neurons

If the motor neurons within the spinal cord are affected, patients suffer from muscle weakness, atrophy and hypotonic paresis, while no sensory deficits occur. Examples are poliomyelitis, caused by the poliovirus, and spinal muscular atrophy, which is an inherited disease.

Disorders of the motor nerve roots

In the case of disorders of the nerve roots at the spinal level L4-L5-S1-S2, the calf muscles are affected. Generally, patients first experience pain before muscle weakness and muscle atrophy develop. In most cases, patients also have sensory deficits and reduced reflexes. Disorders of the motor nerve roots can be due to compression of the nerve root such as in radiculopathy or inflammation (e.g. Lyme’s disease).

Disorders of the peripheral nerve

Disorders of the peripheral nerve are also called neuropathies and are characterized by muscle weakness, sensory deficits and hyporeflexia. These disorders can be inherited, like Charcot-Marie-Tooth disease, or acquired by injury or trauma.

Disorders of the muscles

Patients with muscle disorders, called myopathies, suffer from muscle weakness, muscle cramps and myotonia. Not all myopathies result in non-spastic calf muscle weakness. Inclusion body myositis, which is an inflammatory disorder of muscle tissue, is an example of a myopathy included in our studies.
Second, weakness of the calf muscles limits the generation of ankle push-off power, which has been shown to increase walking energy cost [15]. A reduced ankle push-off power results in a lower upward acceleration of the center of mass prior to contralateral heel-strike, and, consequently, in increased energy losses (i.e. negative work) at contralateral heel-strike [15, 16, 30]. To overcome such increment in negative work and decrease in push-off power, compensations are necessary, as positive work must equal negative work when walking at a steady speed. However, such compensations are mechanically less efficient, and thus will increase walking energy cost [16, 31].

In other clinical populations with a reduced ankle push-off power, such as children with cerebral palsy and patients with amputees, compensations by increasing ipsilateral hip joint work have been reported [30, 32, 33]. However, these studies mostly measured the compensations at preferred comfortable walking speed, which limits the comparability with healthy individuals walking at a higher comfortable speed. Furthermore, the compensations in lower limb joint work have not yet been assessed in persons with non-spastic calf muscle weakness. Neither it has been investigated to what extent the amount of compensatory joint work is related to walking energy cost. Therefore, the compensatory strategies for reduced ankle push-off power, and their relation with the elevated walking energy cost as observed in persons with non-spastic calf muscle weakness are not fully understood.

**Ankle foot orthotics**

To normalize the gait pattern and, subsequently, reduce walking energy cost and fatigue in persons with calf muscle weakness, ankle foot orthoses (AFOs) can be provided [6]. An orthosis is “an externally applied device used to modify the structural or functional characteristics of the musculoskeletal system” [34], where AFOs specifically go around the foot and ankle to restrict the movements of the ankle joint. Different AFO designs are available for provision in usual care, which differ in working mechanism. For example, hinged dorsiflexion-stop AFOs have a hinge that only allows for a certain amount of ankle dorsiflexion thereby blocking the tibia progression from a certain angle on, while ventral and dorsal leaf spring AFOs restrain the tibia progression by providing a plantar flexion moment proportional to the AFO’s bending and stiffness. AFO ankle stiffness is defined as the number of Newton\textperiodcentered meter necessary to bend the AFO one degree. Within this thesis, the AFO ankle stiffness is measured using the Bi-articular Reciprocal Universal Compliance Estimator (BRUCE) [35] (see Figure 1). Furthermore, AFOs can differ in materials used, for example carbon fiber or polypropylene, and material thickness, which influences the mechanical properties [7, 36, 37].
Orthotic treatment for calf muscle weakness

In the case of calf muscle weakness, ideally, the AFO supports the functions of the calf muscles in stance, i.e. restraining ankle dorsiflexion and support of the ankle push-off [6, 38]. Although in usual orthotic care a wide variety of AFOs is applied, scientific evidence for the effectiveness of the different designs in non-spastic muscle weakness is scarce [7]. Previously conducted studies are based on small sample sizes and mainly evaluated dorsal and dorsiflexion stop AFOs, while orthopedic shoes were only evaluated in case studies [39]. Dorsal and dorsiflexion-stop AFOs showed beneficial effects with regard to walking speed, ankle range of motion during gait and ankle moment [3, 40, 41], although the reported effect sizes varied largely, from highly effective [3, 42] to hardly effective [7, 43, 44]. This variation in effect is caused, at least partly, by differences in the AFO mechanical properties, such as AFO type, weight, ankle stiffness and footplate stiffness [8]. In lack of clear prescription guidelines for AFOs in this population, the choice for specific mechanical properties is currently the preference of the prescribing physician or orthotist. It is hypothesized that this results in the provision of a variety of AFO types, which differ largely in mechanical properties and, consequently in effect on gait, although this has never been inventoried and studied in persons with non-spastic calf muscle weakness. Furthermore, most AFOs provided in usual orthotic care are not optimally designed for persons with non-spastic calf muscle weakness as they do not support or even negatively affect the generation of ankle push-off power by restricting the ankle range of motion [3, 40].

Energy returning AFOs in calf muscle weakness

Spring-like AFOs with high energy return capacity better match with the impairments of persons with non-spastic calf muscle weakness. These AFO types can, contrary to rigid AFOs, store energy during the stance phase when the ankle moves towards dorsiflexion and release this energy during push-off when the ankle moves towards plantarflexion, thereby supporting the ankle push-off [45-47]. As such, spring-like AFOs can potentially improve walking speed and reduce walking energy cost beyond that of AFO types without spring-like properties [16].

The effect of spring-like AFOs on walking energy cost and gait biomechanics depends highly on the AFOs’ ankle stiffness as indicated by simulations [8] and small studies in healthy subjects [9], persons with multiple sclerosis and stroke [48] and children with cerebral palsy [49]. An increase in ankle stiffness will restrain the ankle range of motion more and thus the ankle dorsiflexion angle, which is the primary aim of an AFO provided for non-spastic calf muscle weakness [48-50]. However, this comes with a serious drawback: reducing the range of motion also limits the generation of ankle power and the amount of energy stored within the dorsal leaf AFO. Therefore, the most effective stiffness for the
reduction in walking energy cost is hypothesized to be a trade-off between sufficiently restraining the ankle dorsiflexion angle, and storing & releasing the maximal amount of energy while minimally obstructing ankle power generation, as previously found in simulations and in studies in healthy subjects [8, 9].

Figure 1: Schematic overview of the BRUCE device. Based on Bregman et al. (2010) [35].

Ideally, the AFO properties are known before provision as the effects of the AFO depend on the properties. To measure the AFO properties the Bi-articular Reciprocal Universal Compliance Estimator (BRUCE) device is used, which is highly reliable [35]. The BRUCE device consists of a dummy leg and foot, which can be changed in thickness and length to match the size of the AFO. The dummy ankle joint and dummy MTP joint are aligned with the locations of the “real” ankle and MTP joint. To measure the AFO ankle stiffness, the dummy leg is manually rotated towards dorsiflexion, while the footplate stiffness is measured by rotating the footplate of the BRUCE. While rotating, the BRUCE device records the angle between the dummy foot and dummy leg (e.g. ankle angle) and the force the AFO produces on the dummy leg. By moving the dummy leg into dorsiflexion, the linear relation between the ankle angle and force produced by the AFO can be determined, from which the stiffness (Nm/degree) is calculated.
Optimization of energy returning AFOs in calf muscle weakness

The most effective stiffness is likely influenced by walking speed and personal characteristics like body mass and the degree of (calf) muscle weakness [7]. These factors determine the strain on the AFO and, consequently, the necessary stiffness to restrain the ankle angle. Considering the large heterogeneity in these characteristics between individuals, it is hypothesized that the AFO ankle stiffness that optimizes between restraining ankle dorsiflexion in stance versus allowing and supporting the push-off power and, consequently, maximally reduces walking energy cost is patient-specific. Yet, the effects of AFO ankle stiffness on gait and walking energy cost at group and individual level in persons with non-spastic calf muscle weakness have never been studied.

In usual orthotic care, the AFO ankle stiffness is not individually optimized, but guided by the preference of the treating physician and/or orthotist. Therefore, as it is assumed that the best AFO stiffness for improving gait is patient specific, individually optimizing the AFO ankle stiffness is hypothesized to improve treatment outcomes, which has been shown in children with cerebral palsy [51], but is not yet studied in persons with neuromuscular disorders demonstrating non-spastic calf muscle weakness.

Aim

This thesis aims to expand our understanding of how AFO ankle stiffness affects gait and if optimization of the AFO ankle stiffness improves treatment outcomes, primarily walking energy cost, in persons with non-spastic calf muscle weakness.

Outline

In Chapter 2, the compensations in lower limb joint work during walking in response to non-spastic calf muscle weakness are studied to expand our knowledge on the gait pattern and the importance of ankle power for walking and walking energy cost in persons with neuromuscular disorders exhibiting non-spastic calf muscle weakness. Chapter 3 evaluates usual orthotic care in the Netherlands by measuring the mechanical properties of currently applied AFOs for non-spastic calf muscle weakness, such as type, weight, ankle stiffness, and footplate stiffness, and evaluating the effect of the provided AFOs on walking speed, walking energy cost and gait biomechanics. In Chapter 4, the effects of optimizing the AFO ankle stiffness of two spring-like AFO types, a spring-hinged AFO and a dorsal leaf AFO, on walking speed, walking energy cost and gait biomechanics are evaluated in a proof-of-concept study among three polio survivors with non-spastic calf muscle weakness. Chapter 5 describes the effect of varying the AFO ankle stiffness of a carbon dorsal leaf AFO in a large sample of persons with neuromuscular disorders exhibiting non-spastic calf muscle weakness on walking speed, walking energy cost, and gait biomechanics.
Chapter 1

To test the main hypothesis of this thesis, that the optimal AFO stiffness for improving gait is patient specific in persons with non-spastic calf muscle weakness, a prospective clinical trial, called PROOF-AFO, was conducted. The protocol of PROOF-AFO trial, which stance for *Precision orthotics: optimizing ankle foot orthoses to improve gait in patients with neuromuscular diseases*, is described in chapter 6. In chapter 7, the results of the PROOF-AFO trial are presented, where stiffness-optimized AFOs are compared to standard non-optimized AFOs provided in usual orthotic care for walking energy cost, walking speed, gait biomechanics and daily physical activity.

Finally, in chapter 8 the main findings of this thesis and clinical implications are discussed.
REFERENCES


