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Early observations of the effects of lateral wedge orthoses on lower limb muscle length and potential for exacerbating spasticity

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Abstract
Lateral wedge orthoses are often prescribed to correct varus deformity after stroke. Spasticity is implicated in varus deformity and is caused by velocity-related muscle length changes, so a lateral wedge may affect spasticity by manipulating foot and ankle kinematics which, in turn, may alter the length of these muscles. We sought to test this theory in healthy participants. Eight volunteers walked with no wedge and with lateral wedges of 5 and 8.5 degrees in both shoes. Qualysis motion capture system collected kinematic data and SIMM musculoskeletal modeling software calculated muscle tendon length of plantarflexor and peroneal muscle groups using 3-D ankle and knee joint angle data. The wedges increased ankle eversion ($p < 0.0001$) and total excursion of tibialis posterior, peroneus longus and brevis by 13–29% ($p < 0.05$). Muscle length of peroneus longus and brevis increased by $<1\%$ ($p < 0.005$). Potentially clinical meaningful effects were found for tibialis posterior (15%), peroneus brevis (23%) and peroneus tertius (13%). Further research is required to be conclusive and to explore the effects of lateral wedge orthoses in patients with stroke. If such changes are seen in people with stroke, a change in orthotic prescription practice could be indicated as lateral wedge orthoses may exacerbate spasticity.

Keywords: *Lateral wedge orthoses, spasticity, stroke, musculoskeletal modelling*

Introduction

Foot orthoses are often prescribed for patients with stroke, to prevent or correct deformities, alleviate symptoms and improve function.[1,2] However, the effects of orthoses are controversial. Orthotic devices are designed routinely to optimize biomechanics but their effect on neuromuscular impairments such as spasticity is rarely considered.[3] There is however, clinical and objective evidence that orthoses can modify foot and ankle biomechanics[4] and elicit neuromuscular responses.[5] The most widely used foot orthosis for patients with stroke is the lateral wedge orthosis which is prescribed to improve balance

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and gait and correct varus deformity.[6–8] Previous studies have demonstrated that wedge-shaped orthoses can influence electromyographic activity of lower limb muscles in the normal population.[9,10] If this is also the case in patients with neurological conditions, then orthotic design could positively or adversely affect muscle activity and consequently, function. We hypothesized that a lateral wedge orthosis could affect the neuromuscular function of the foot and ankle, specifically spasticity, after stroke. Spasticity is a velocity-dependent increase in muscle activity,[11] which is thought to be an important impairment that limits function in many people with stroke and other neurological conditions. It is known that plantarflexor (medial gastrocnemius and soleus muscles) activity increases at a much lower lengthening velocity in people with stroke than healthy controls.[12,13] Thus, if a lateral wedge alters foot and ankle kinematics it could change the pattern and velocity of lengthening in these muscles and thus alter the spasticity, positively or negatively. As a precursor to the study of stroke patients we sought to test this theory on healthy participants using musculoskeletal modelling techniques. Healthy participants were recruited to eliminate the influence of pathologies or uncontrolled compensations that may occur in the stroke population.

The purpose of this study was to explore the effect of different degrees of lateral wedge on the origin-insertion length and lengthening velocity of seven plantarflexor and peroneal muscles in healthy participants.

Methods

Study design and participants

A randomized cross-over trial design was used. Walking with no wedge was the control condition and the order in which the participants wore two wedge orthoses was randomized. Participants were excluded if they had orthopaedic or neurological deficits impairing foot and ankle function or mobility. Eight men with a mean age of 33.5 ± 4.5 years and a mass and height of 71.4 ± 10.6 kg and 1.70 ± 0.06 m, respectively, were recruited from staff and students of the School of Health Care Professions at the University of Salford. Ethical approval was obtained from the University's Research Ethics Committee.

Equipment. A 10-camera Qualysis Proreflex system (Qualisys Medical, 2003) was used to obtain three-dimensional coordinate kinematic data of the thigh, shank and foot. The measurement frequency of cameras was set at 100 Hz. Kinetic data were collected using four AMTI force plates (model BP400600, AMTI, Watertown, MA, USA) embedded in walkway at a frequency of 240 Hz and synchronized with camera system.

Intervention. Two lateral heel and sole wedge orthoses (5 and 8.5 degrees) were compared with the 'no wedge' control condition. These were chosen as they are routinely prescribed in clinical practice. They were made from high density vinyl acetate with reference to the size of the participants' feet. The wedge was graded to zero at the base of the 5th metatarsal and attached to the base of an EVA flat based neutral insole (Algeos, Liverpool, UK) (Figure 1).

Procedure. Participants who met inclusion criteria provided informed consent and participated in a single testing session. A standardized shoe was used for all participants to avoid confounding results from differing footwear. Sixteen retro-reflective markers, either individual or as rigid clusters of markers, were attached to the shoes, shank and thigh of the

right and left legs. The Calibrated Anatomical System Technique (CAST) was adopted to establish a suitably anatomical model of the foot and lower limb. Table I and Figure 2 give details of the marker placement.

One trial was collected with the participant standing to define segments and establish the relationship between anatomical and tracking markers, then several practice walking trials were conducted to determine the starting position from which right and left foot could be placed consistently on the first and second force plates, respectively, and to enable participants to adapt to the orthosis and laboratory environment. After completing the practice period, participants walked over the 10 m walkway at a self-selected speed with no wedge and each of the wedges. Ten trials were recorded for each condition.

Data analysis. Kinematic data were processed using the calibration data in standing to calculate ankle, knee and hip joint centres to define segmental coordinate systems for the foot, shank and thigh. Kinematic and kinetic data for each walking trial were smoothed using a 4th order Butterworth low-pass filter with a cut-off frequency of 6 and 15 Hz, respectively, after which joint ankle angles were calculated using Cardan angles. All kinematic calculations were implemented with using Visual3D (C-motion, USA). Initial contact and toe off were determined as the points at which the vertical ground reaction force exceeded a

Figure 1. The lateral heel and sole wedge orthoses: no wedge (right), 5° (middle), 8.5° (left).

Table I. Anatomical and tracking markers.

Segments	Anatomical markers	Tracking markers
Thigh	1. One marker on the trochanter; 2. Two markers on the medial and lateral femur epicondyles.	A cluster of four markers on the lateral aspect of thigh
Shank	1. Two markers on the medial and lateral femur epicondyles; 2. Two markers on the most medial and lateral aspects of malleolus (MMAL and LMAL, respectively).	A cluster of four markers on the lateral aspect of shank
Total foot (a single segment)	1. MMAL and LMAL; 2. One marker on the distal head of first metatarsal over the shoes (SD1MT); 3. One marker on the distal head of fifth metatarsal over the shoes (SD5MT).	1. One marker on the posterior and superior part of shoes; 2. SD1MT; 3. SD5MT.

Figure 2. Anatomical and tracking markers.

threshold of 8 N. The kinematic data was then expressed as a function of stance phase and exported to SIMM musculoskeletal modeling software which was used to calculate muscle-tendon length of seven lower leg muscles during stance phase. SIMM creates graphics-based models of the musculoskeletal system that quantify the effects of joint kinematics, musculoskeletal geometry and muscle-tendon parameters on muscle-tendon lengths, moment arms, muscle forces and joint moments.[15] It calculates muscle length over time as a function of joint angle and takes into account sagital and frontal plane motion of the ankle and knee joints. The muscle-tendon length data were scaled to the participants' height, expressed as a percentage of stance phase and analyzed in Matlab to determine the following variables for each condition: maximum length; total excursion (maximum length minus minimum length); and maximum lengthening velocity. The selected muscles were medial and lateral gastrocnemius, soleus, tibialis posterior and peroneus brevis, longus and tertius.

SPSS v16 was used to conduct statistical analyses. Normal data distribution (in all parameters) was confirmed by the Shapiro-Wilk test ($p > 0.05$). Repeated measures ANOVA, with Bonferroni adjustment for multiple comparisons as a post hoc test, investigated the effects of lateral wedges on kinematic, muscle length and lengthening variables. *P*-values less than 0.05 were considered statistically significant.

Results

The effect on walking speed

The wedges had no effect on walking speed. Mean walking speed without a wedge was $1.33 \pm 0.12\ \mathrm{m \cdot s^{-1}}$ and it did not change significantly when the wedges were introduced; changes were within 3% of the walking speed with no wedge (control condition).

The effect on foot and ankle kinematics

A lateral wedge orthosis increased frontal plane motion of the ankle (Figure 3): Maximum ankle eversion during stance increased following the introduction of the wedges with greater eversion produced when participants walked with the lateral wedges. The angles were $11.4^\circ \pm 3.5^\circ$, $11.9^\circ \pm 3.0^\circ$ and $12.6^\circ \pm 2.9^\circ$ for the control (no wedge), 5° wedge and 8.5° wedge, respectively ($p < 0.0001$). Post-hoc analysis revealed significant differences between the 8.5° and control, and between 8.5° and 5° wedge conditions.

Effects on the lower leg muscle length

Total excursion and maximum muscle length tended to increase with the greater larger wedge. The 8.5° wedge showed significant increases ($p < 0.05$) in total excursion of tibialis posterior, peroneus longus and peroneus brevis by 13%, 29% and 23%, respectively (Table II), compared to the control (no wedge). Maximum muscle length increased significantly in peroneus longus and peroneus brevis but the differences were less than 1%. Figure 4 shows the effect of the lateral wedges on proneus brevis muscle tendon length.

Maximum lengthening velocity increased for all tested muscles (Table II); by 2–23% when wearing the 8.5° wedge compared with the control but the differences did not reach statistical significance.

Discussion

The aim of this study was to make an initial exploration of the effect of lateral wedge orthoses on the muscle-tendon complex in lower leg muscles of healthy participants with a view to informing orthotic prescription for patients with stroke. The results indicate that a lateral wedge orthosis can influence the muscle lengthening properties of the muscles

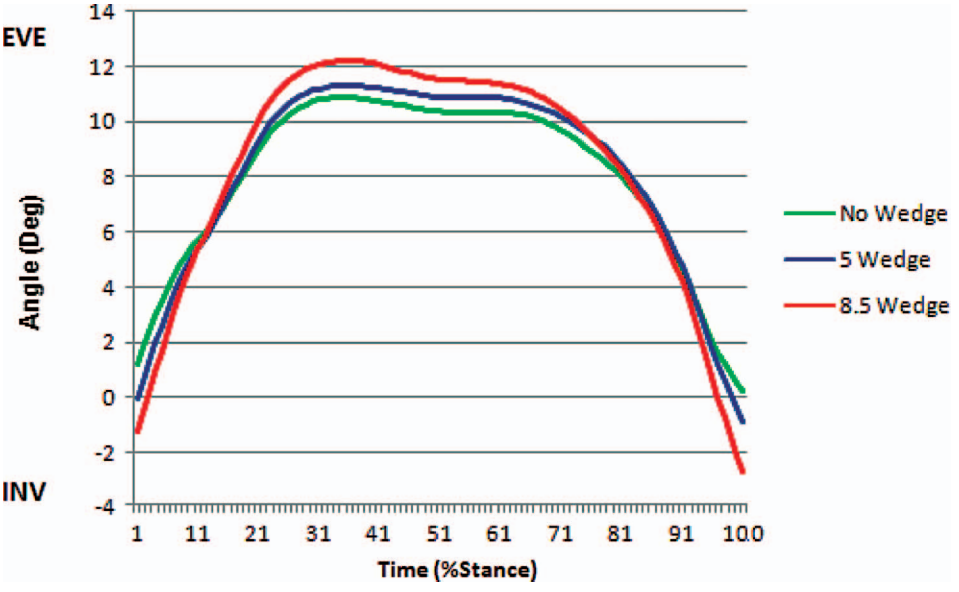

Figure 3. Mean ankle kinematic pattern in the frontal plane during stance phase in no wedge (green), 5° wedge (blue), and 8.5° wedge (red) conditions. Positive angles are eversion.

Table II. Mean values of the peak of lengthening velocity (m/s) and total excursion (mm) for the selected muscles, $^{**}p < 0.001$.

Muscle	No wedge	5° wedge	8.5° wedge
Medial gastrocnemius			
Lengthening velocity	0.055 ± 0.010	0.055 ± 0.011	0.057 ± 0.010
Excursion	18.97 ± 2.49	18.89 ± 2.22	19.07 ± 2.19
Lateral gastrocnemius			
Lengthening velocity	0.058 ± 0.012	0.057 ± 0.012	0.059 ± 0.011
Excursion	19.56 ± 2.59	19.47 ± 2.19	18.79 ± 2.23
Soleus			
Lengthening velocity	0.051 ± 0.014	0.051 ± 0.012	0.053 ± 0.011
Excursion	18.71 ± 2.52	18.61 ± 2.31	19.66 ± 2.36
Tibialis posterior			
Lengthening velocity	0.026 ± 0.005	0.027 ± 0.004	0.030 ± 0.004
Excursion	7.11 ± 1.23	7.41 ± 1.25	8.01 ± 1.13**
Peroneus brevis			
Lengthening velocity	0.021 ± 0.013	0.026 ± 0.011	0.026 ± 0.010
Excursion	5.59 ± 1.96	6.2 ± 1.87	7.21 ± 1.43**
Peroneus longus			
Lengthening velocity	0.021 ± 0.010	0.024 ± 0.009	0.022 ± 0.009
Excursion	6.52 ± 2.09	7.00 ± 2.16	8.00 ± 1.6**
Peroneus tertius			
Lengthening velocity	0.115 ± 0.020	0.125 ± 0.029	0.130 ± 0.032
Excursion	10.06 ± 1.94	10.44 ± 1.67	10.92 ± 1.72

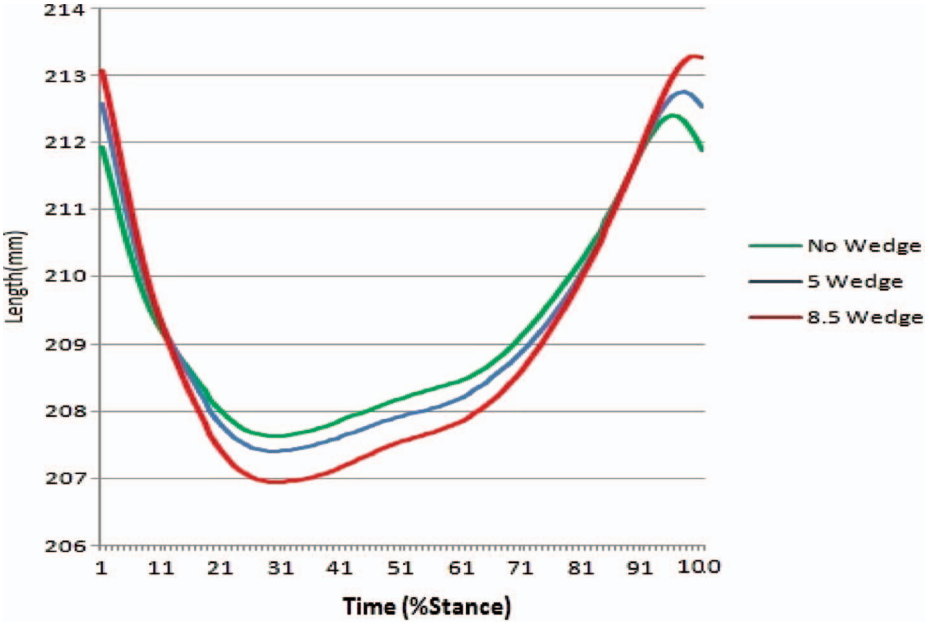

Figure 4. The effect of lateral wedges on peroneus brevis muscle tendon length.

acting over the ankle and foot with greater increases with the larger wedge. As such they provide 'proof-of-concept' that a lateral wedge orthosis can effect muscle lengthening and thus could influence spasticity. If this is the case, then using a lateral wedge may be detrimental for people with spasticity, although non-significant, lengthening velocities increased with the lateral wedges by up to 23%. Such changes would be sufficient to exacerbate a spastic response in people with stroke as our previous work has shown that the lengthening velocity at which a spastic response (a dramatically increase in muscle activity) is elicited is only 14% less than in healthy control participants.[12] Further research is required to investigate the effects of a lateral wedge on the balance and gait of patients with and without spasticity following stroke and to develop optimal orthotic designs.

There was also a trend for maximum muscle length and total excursion to increase when wearing the lateral wedge. Increased muscle stiffness and shortening are frequent complications after stroke, particularly around the foot and ankle, and are caused by adaptive changes in the intrinsic mechanical properties of muscles and tendons secondary to immobility.[16] In such cases, a device which facilitated muscle lengthening would be desirable. Our findings that a lateral wedge may increase spasticity (which is detrimental) and muscle length (which is beneficial) suggest that a lateral wedge might be most effectively prescribed in cases where muscle stiffness is a problem in the absence of spasticity. However, these components (spasticity and stiffness) are most commonly found together in upper motor neurone conditions[17] and it is difficult to distinguish between them in the clinical settings;[18] the prescription of a lateral wedge orthosis may be better suited to patients with a lower motor neurone pathology, for whom spasticity is not a feature. Further studies to address this clinical issue are planned.

This pilot, proof-of-concept study has several limitations. Firstly, the most likely explanation for the lack of consistent statistical significance is the sample size; the comparisons were under-powered. Power calculations show that a sample size of 15 participants would provide 80% power to demonstrate a significant increase in maximum lengthening velocity of tibialis posterior, for example. Research is ongoing to power comparisons to provide a definitive answer. Secondly, healthy participants were recruited and people with stroke respond to a lateral wedge orthosis differently. However we felt, ethically, it was necessary to test the impact of potentially detrimental intervention in healthy participants before a clinical population.

Another consideration is whether the participants' self-selected walking speed may have confounded the results. Walking speed affects muscle-tendon length and lengthening velocities; the slower the speed, the shorter the peak muscle-tendon length and the slower the lengthening velocity.[19] Our healthy participants walked at a self-selected speed to follow the normal pattern of walking and prevent any uncontrolled compensations. As each was compared to his own baseline (walking with no orthotic) and so any differences are unlikely to be purely due to differences in walking speed. However, further testing with a clinical population is needed to replicate our findings at slower walking speeds. Finally, we used a standard musculoskeletal model of the lower limb which does not consider subject-specific musculoskeletal geometry and assumes that the foot acts as a rigid body. Furthermore, we described changes in muscle kinematics without measuring spasticity directly, to confirm whether changes occurred at this level, muscle activity should be also recorded.

Conclusion

These results indicate that a lateral wedge orthosis can influence muscle lengthening properties in some plantarflexor muscles of healthy volunteers. If the same results are

observed in a larger study of patients with stroke, this would raise concerns regarding the prescription of lateral wedge orthoses in this population as they would suggest that a lateral wedge may exacerbate spasticity in the lower limb. Further research is indicated to assess this hypothesis in a clinical population before firm conclusions can be drawn.

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