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ORIGINAL REPORT

INFLUENCE OF FUNCTIONAL ELECTRICAL STIMULATION OF THE HAMSTRINGS ON KNEE KINEMATICS IN STROKE SURVIVORS WALKING WITH STIFF KNEE GAIT

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Objective: **To explore whether functional electrical stimulation of the hamstrings results in improved knee kinematics in chronic stroke survivors walking with a stiff knee gait.**

Design: **Quasi-experimental.**

Subjects: **Sixteen adult chronic stroke survivors.**

Methods: **Survivors received functional electrical stimulation of the hamstrings, 3 times a week for 1 h during a period of 5 weeks. 3D kinematics was calculated before the training period and after 5 weeks of training. Knee kinematics of walking without stimulation before the training period was compared with walking with stimulation after 5 weeks of training. (intervention effect). In addition, knee kinematics of walking without stimulation before the training period was compared with walking without stimulation after the training period (therapeutic effect).**

Results: **The intervention effect showed a significant increase, of mean 8.7° (standard deviation (SD) 8.3, $p = 0.001$), in peak knee flexion. The therapeutic effect showed a significant increase in peak knee flexion, of mean 3.1° (SD 4.7, $p = 0.021$)**

Conclusion: **The results of this exploratory study shows an increase in knee kinematics in swing after functional electrical stimulation of the hamstrings in stroke survivors walking with a stiff knee gait. The largest improvement in peak knee flexion in swing was seen when participants walked with hamstring stimulation. Participants with low neurological impairment responded better to hamstring stimulation, and there are indications that the effect of hamstring stimulation can be predicted during a single session. The effect of functional electrical stimulation is comparable to that of more invasive treatment options, such as botulinum toxin or soft-tissue surgery. This makes functional electrical stimulation a feasible treatment option for daily clinical practice.**

Key words: stroke; walking; kinematics; functional electrical stimulation; hamstrings; stiff knee gait.

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LAY ABSTRACT

This exploratory study examined the effect of functional electrical stimulation of the hamstrings on knee kinematics and walking speed in 16 chronic stroke survivors walking with a stiff knee gait. Participants were measured before and after 5 weeks of training with functional electrical stimulation. There was an increase in peak knee flexion in the swing phase after the training period, while walking with functional electrical stimulation. Participants with low neurological impairment responded better to hamstring stimulation, and there are indications that the effect of hamstring stimulation can be predicted during a single session. The effect of functional electrical stimulation is comparable to that of more invasive treatment options, such as botulinum toxin or soft-tissue surgery. Functional electrical stimulation is therefore a feasible treatment option for daily clinical practice.

Stiff knee gait is an abnormal movement pattern commonly observed in stroke survivors. It is characterized by reduced peak knee flexion (PKF) during the swing phase. The limited knee flexion may cause toe dragging or energy-inefficient compensatory movements (1), compromising the stability of the gait and increasing the risk of falling (2). The pathophysiology of stiff knee gait is only partly understood, and several hypotheses are postulated in the literature. The role of overactivity of the rectus femoris during the swing phase is often cited (3–5). Other possible causes of stiff knee gait are increased forces generated by the vasti (6), decreased hip flexion moments (7) and decreased ankle plantar flexion moments (8). However, the exact mechanisms remain unclear and seem to be multifactorial.

Different treatment options for stiff knee gait are available that are aimed at influencing the overactivity of the rectus femoris. These options include chemodenervation of the rectus femoris (9) and rectus femoris transfer (10, 11). The indication for chemodenervation or RF transfer treatment is related to overactivity of the rectus femoris in pre-swing or swing. This means that only those patients who exhibit this overactivity are eligible for this type of treatment. Electrical stimula-

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tion of the calf and/or hamstring muscles (12, 13) is a treatment option that might be suitable for all patients, irrespective of the cause of stiff knee gait, as it might directly assist in achieving sufficient knee flexion. Studies on the effect of electrical stimulation of the calf or hamstrings on the hemiplegic gait pattern are, however, scarce and most of the studies stimulated 2 or more muscle groups in order to influence gait kinematics.

One practical problem of stimulating 2 or more muscles is the timing of the stimulation, as the proper timing of muscle contraction may differ between muscles. Providing adequate timing is easier when stimulating only one muscle, which makes it more feasible in clinical practice. From a clinical point of view, it is therefore interesting to investigate whether knee flexion during swing can be improved by stimulating only one muscle group.

The primary aim of the present study was to explore whether 5 weeks of functional electrical stimulation of the hamstrings results in improved knee kinematics during the swing phase in chronic stroke subjects with a stiff knee gait. The study compared: (*i*) walking without electrical stimulation pre-intervention with walking with electrical stimulation post-intervention (*intervention effect*). The secondary aims were to compare: (*ii*) walking without electrical stimulation pre-intervention with walking without electrical stimulation post-intervention (*therapeutic effect*); and (*iii*) walking without stimulation pre-intervention with walking with stimulation pre-intervention (*immediate effect*). Furthermore, based on clinically experience, the study explored: (*iv*) the relationship between the immediate effect and the intervention effect. Finally, based on the findings of Hanlon & Anderson (14) who found a positive relationship between walking speed and knee kinematics, the study aimed to explore: (*v*) the influence of walking speed on the intervention effect of stimulation.

METHODS

Study design

The study was designed as an exploratory prospective quasi-experimental study (Fig. 1). It was approved by the local Medical Ethics Committee (MEC Twente).

Study population

A convenience sample of adult chronic stroke survivors (>6 months after stroke) were recruited for participation at Roessingh Centre for Rehabilitation (RCR), Enschede, the Netherlands. Inclusion was based on: visible diminished knee flexion during the swing phase, ability to walk without physical support, ability to complete a 3.5-h assessment, and ability to understand and follow verbal instructions. Exclusion criteria were: a pacemaker, metal implants in the paretic leg, or orthopaedic problems or progressive diseases influencing the walking pattern. Participants were allowed to continue their regular treatment during the study and received oral and written information about the study before they decided to participate.

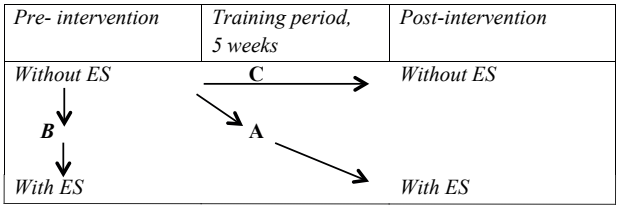

Fig. 1. Study design. A: intervention effect (post-intervention with electrical stimulation (ES) minus pre-intervention without ES). B: immediate effect (pre-intervention with ES minus pre-intervention without ES). C: therapeutic effect (post-intervention without ES minus pre-intervention without ES).

Intervention

Patients were treated with electrical stimulation for 1 h, 3 times a week, for a period of 5 weeks (15 h in total). Therapy was provided by a senior physical therapist with longstanding experience (over 20 years) in the use of electrical stimulation in the stroke population. Each session consisted of walking with electrical stimulation of the hamstrings of the paretic leg. Participants walked at a comfortable speed indoors at the physical therapy department and were allowed to stop or rest when necessary.

The Odstock 2-channel footswitch controlled stimulator system (Odstock Medical Limited, Salisbury, UK) was used for stimulation. Self-adhesive skin surface electrodes, with a size of 50×100 mm (CefarCompex Medical, Lund, Sweden) were placed on the mediolateral aspect of the hamstrings (15). A footswitch was used to trigger the stimulation between heel off and toe off. The indifferent electrode was placed approximately 5 cm above the knee crease and the active electrode was placed approximately 10 cm above the indifferent electrode. During an initial, pre-intervention session the location of the foot switch, the locations for electrode placement and stimulation settings (amplitude, pulse duration) were determined for maximum optimization of the walking pattern. These locations remained the same during the 15 sessions. The pulse duration varied between 0.125 and 0.475 s. The stimulation frequency was 40 Hz.

Experimental protocol

All participants were tested pre- and post-intervention, both during walking without and with stimulation. During the pre-intervention session, anthropometric data were collected.

Participants were instructed to walk at their natural, comfortable speed. During both evaluations and training period, participants used the same walking aids, orthoses and shoes.

Participant characteristics

The Rivermead Mobility Index (16), Functional Ambulation Category (17), Motricity index (18) and the Duncan Ely test (19) were administered only at the pre-intervention assessment to determine participants' characteristics. Furthermore, the use of an ankle foot orthosis (AFO) and walking aids were recorded. In addition, adverse events during the experiment, such as blisters, skin problems or intolerance of the stimulation, were recorded.

Table I. Participant characteristics

Participants (dropouts), *n*	16 (0)
Age, years, mean (SD)	57.6 (10.3)
Sex (male/female), *n*	14/2
Time after stroke, years, mean (SD)	5.8 (5.4)
Paretic side (left/right), *n*	7/9
Use of walking aids, walking with/without, *n*	6/10
Use of ankle foot orthosis, walking with/without, *n*	8/8
Duncan Ely, median, (IQR)	1 (1–2)
Functional Ambulation Categories, median, (IQR)	5 (4–5)
Rivermead Mobility Index, (0–15), mean (SD)	12.2 (0.9)
Motricity Index lower extremity, (0–99), mean (SD)	64.7 (17.8)

SD: standard deviation; IQR: interquartile range.

Kinematics

To determine knee kinematics and preferred walking speed, an infrared opto-electronic 3D-motion analysis system (VICON MX + 6 MX13 cameras, frame rate 100 Hz; Vicon Motion Systems, Oxford, UK) was used. Participants walked a 10-m walkway. A standard marker-placement (Plug in Gait model) was used and one person made all the marker placements. To normalize data to the gait cycle, initial contact and toe off events were detected. A minimum of 10 strides were analysed and averaged for each participant to determine PKF during swing, knee range of motion (minimum stance phase vs maximum swing phase) and walking speed.

Statistical analysis

All variables showed sufficient closeness to a normal distribution, as determined visually by a senior statistician.

To identify the effect of ES on knee kinematics and walking speed, data for the 3 walking conditions were compared: (*i*) the intervention effect (pre-intervention without ES vs post-intervention with ES); (*ii*) the therapeutic effect (pre-intervention without ES vs post-intervention without ES); and (*iii*) the immediate effect (pre-intervention without ES vs pre-intervention with ES). Data were analysed with paired samples *t*-test. (*iv*) To predict the intervention effect in one try-out session (*immediate effect*) the correlation between knee kinematics of the intervention effect and those of the immediate effect were calculated using Pearson's correlation. (*v*) To explore the influence of walking speed on the effect of stimulation the correlation between knee kinematics of the intervention effect and the intervention effect for walking speed were calculated using Pearson's correlation.

Table III. Correlations

Outcome	Intervention effect peak knee flexion	Intervention effect knee range of motion
Intervention effect walking speed	R = 0.12, $p = 0.650$	R = 0.09, $p = 0.741$
Immediate effect peak knee flexion	R = 0.75, $p = 0.001$*	R = 0.74, $p = 0.001$*

*Denotes a statistically significant difference between the conditions. $p \leq 0.05$.

Participant characteristics and outcome measures were described with descriptive statistics using mean and standard deviation (SD). All statistical analyses were performed using SPSS 19.0 for Windows. A $p \leq 0.05$ was considered statistically significant. A correlation above 0.7 was considered relevant.

RESULTS

Participants

A total of 16 chronic stroke survivors were included in the study (Table I). All participants attended all therapy sessions and there were no dropouts. Eight patients walked with an AFO, 5 patients walked with a cane, and 1 patient walked using a quad cane. No adverse events were reported.

Kinematics and walking speed

The kinematic parameters analysed for the different testing conditions are shown in Table II. The kinematic parameters and walking speed of the intervention effect, therapeutic effect and immediate effect are shown in Table II.

Correlations

The calculated correlations are shown in Table III.

DISCUSSION

The aim of the present study was to quantify the effect of hamstring stimulation during the swing phase of

Table II. Knee kinematics and walking speed

	Pre-intervention		Post-intervention		Difference effect		
Outcome measure	Without ES Mean (SD)	With ES Mean (SD)	Without ES Mean (SD)	With ES Mean (SD)	Intervention effect Mean (SD)	Therapeutic effect Mean (SD)	Immediate effect Mean (SD)
Peak knee flexion (°)	29.1 (9.0)	34.6 (12.1)	32.2 (11.6)	37.9 (13.4)	8.7 (8.3) $p = 0.001$* (CI 4.3; 13.2)	3.1 (4.7) $p = 0.021$* (CI 0.5; 5.6)	5.5 (7.0) $p = 0.007$* (CI 1.7; 9.2)
Knee range total cycle (°)	27.7 (8.0)	33.2 (10.4)	30.2 (9.6)	35.9 (10.7)	8.2 (7.7) $p = 0.001$* (CI 4.0; 12.2)	2.5 (4.0) $p = 0.027$* (CI 0.3; 8.9)	5.5 (6.5) $p = 0.004$* (CI 2.0; 8.9)
Walking speed (m/s)	0.86 (0.22)	0.95 (0.24)	0.91 (0.21)	0.97 (0.23)	0.11 (0.10) $p = 0.000$* (CI 0.06; 0.17)	0.05 (0.06) $p = 0.005$* (CI 0.02; 0.08)	0.09 (0.09) $p = 0.001$* (CI 0.04; 0.14)

*Denotes a statistically significant difference between the conditions. $p \leq 0.05$. (Paired sample *t*-test).
SD: standard deviation; ES: electrical stimulation; CI: confidence interval.

gait in chronic stroke survivors with a stiff-knee gait. The results showed that walking with stimulation of the hamstrings after 5 weeks of training resulted in a statistically significant increase in PKF and knee range of motion in chronic stroke survivors with a stiff knee gait. In addition, a statistically significant therapeutic and immediate effect was found for PKF and knee range of motion. Self-selected walking speed increased statistically significantly with hamstring stimulation (intervention effect).

It is not known what the clinically meaningful difference in PKF is for treatment options for stiff knee gait. Therefore, it is debatable whether the statistically significant increase of 3° as a therapeutic effect is clinically meaningful for the patient. The intervention effect of hamstring stimulation is considerably larger (8.7°). Thus, hamstring stimulation might be regarded more as an assistive device than a therapeutic device, as the effect of functional continuous stimulation (comparison of pre with stimulation and post with stimulation) is much larger than when it is used only as a training device (comparison of pre without stimulation and post without stimulation).

Although this study found a positive result for hamstring stimulation at the group level, there was a strong heterogeneity in effect at the individual level (see Fig. 2 for the exemplary data for 3 individuals). In other words, there were participants in whom a large intervention effect was seen and there were participants in whom no or only a small intervention effect was seen. The 4 participants with the largest response showed an improvement of more than 16° in PKF during swing. From a clinical point of view, insight into the participant characteristics that distinguish this subpopulation from the overall study population is of interest. All 4 large responders walked without an AFO or walking aid, had a high score on the Motricity Index (>69) of the lower extremity, and had a low spasticity score of the rectus femoris measured with the Duncan Ely Test (score = 1). Therefore, responders with the largest improvement appeared to have low neurological impairment and were able to adapt their walking pattern to the electrical stimulation. Following this line of thought, patients with severe neurological impairments may have limited ability to adapt their walking pattern to incorporate the hamstring stimulation.

A strong statistically significant correlation was found between the knee kinematics of the immediate effect and the knee kinematics of the intervention effect. Participants with a large immediate response also showed a large response after 5 weeks of training and for participants in whom the immediate response was low, the response after 5 weeks of training was also low. Clinically this might be a crucial issue. It means that the effect of functional electrical stimulation of the hamstrings at the individual level can be predicted with a high probability during a single session.

A statistically significant increase in walking speed was found, which may have contributed to an increase in knee kinematics (14), reducing the true effect of hamstring stimulation. However, a non-significant correlation between the change in walking speed and the knee kinematics (PKF and knee range of motion) was found. Furthermore, van Hedel et al. (20) showed that, in healthy subjects, an increase in walking speed of 0.9–1.0 m/s, which is the magnitude of the differences that we found, led to an increase in PKF of 1–2°. Based on this, it can be concluded that positive influence of the increased walking speed on knee kinematics is negligible in our study, and that the described differences are the result of the electrical stimulation.

In addition, it is debatable whether the significant improvement in walking speed, of 0.11 m/s from mean 0.86 (SD 0.22) to 0.97 (SD 0.23) m/s (intervention effect), was clinically meaningful for the patients, as there is no consensus on the magnitude of the minimal

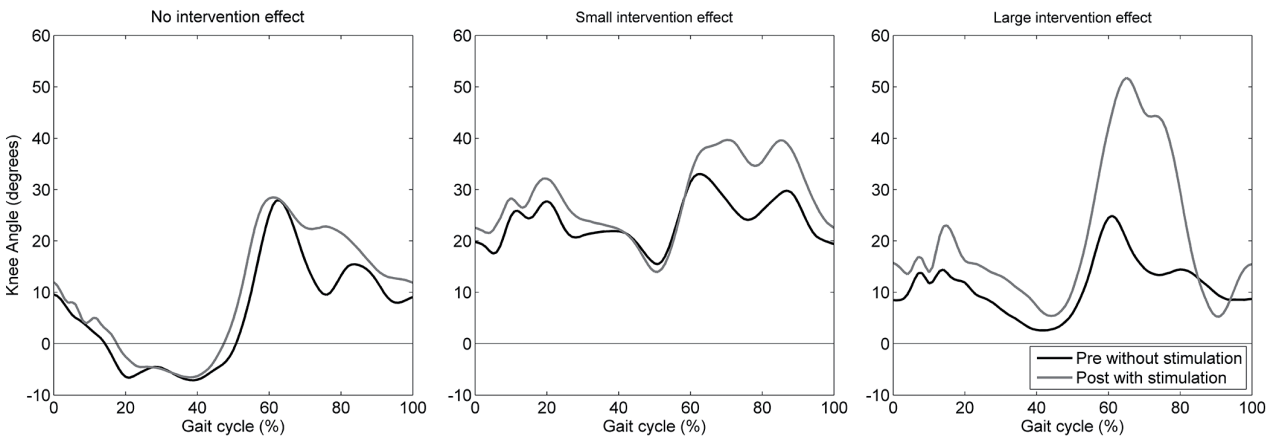

Fig. 2. Heterogeneity in intervention effect.

clinical important difference. Tilson et al. (21) concluded that a meaningful improvement in comfortable walking speed in subacute stroke patients is >0.16 m/s, whereas Perera et al. (22) stated that a change of 0.1 m/s in walking speed was a significant clinical difference.

To our knowledge, this is the first study to examine the effect of stimulating only one muscle (hamstring), instead of more muscles, on knee kinematics during the swing phase of gait for the treatment of stiff knee gait. The results of this study can therefore only be compared with the results of studies that stimulated 2 or more muscles. Kesar et al. (12) stimulated the dorsal and plantar flexors of the ankle, but found no significant improvement in PKF. Mann et al. (13) investigated the effect of stimulation of hamstring muscles in addition to common peroneal nerve stimulation. Of the 6 participants who were stimulated, one showed a substantial improvement in knee flexion and 3 showed a general improvement (not specified) in knee flexion response. Mann et al. (13) did not mention PKF or knee range of motion. Daly et al. (23) used intramuscular electrodes to stimulate 8 lower limb muscles, including the hamstrings, for gait training in combination with a 3-month treatment protocol, consisting of weight-supported-treadmill training, strength and coordination training and overground training in chronic stroke. They found a significant improvement in PKF of 10.0° ($p=0.02$) during walking without stimulation in the group who also received functional neuromuscular stimulation, in comparison with the group without neuromuscular stimulation treatment (PKF±1.4°, $p=0.84$). This significant therapeutic increase in PKF of 10° is larger than the significant therapeutic increase of mean 3.1° (SD 4.7, $p=0.021$) in our study. Although our study and the study of Daly et al. (23) are difficult to compare, a possible explanation for the larger increase in PKF in the study of Daly could be the difference in training intensity. In the literature, other treatment options for stiff knee gait have been investigated. A systematic review performed by Tenniglo et al. (24) reported that chemodenervation of the rectus femoris resulted in a significant pooled improvement of 7.4° in PKF in stroke patients with a stiff knee gait. However, no significant difference in knee flexion range of motion was reported in this review. Namdari et al. (25) examined the effect of rectus femoris transfer in combination with fractional lengthening of the vasti in adults with stroke, and found an increased knee flexion during swing, changing from 8° preoperatively (range 0–15°) to 33° postoperatively (range 20–50°). Lennon et al. (26) found no improvement in PKF after physiotherapy based on the Bobath concept in adults with stroke.

Overall, it seems that the intervention effect of hamstring stimulation on knee kinematics is comparable or slightly better compared with the other treatment options for stiff knee gait. However, comparison between studies is difficult, because literature about the treatment of stiff knee gait is scare and very diverse in methodology. In addition, inclusion criteria were different and the diversity of reasons for a stiff knee gait may have affected the results.

Study limitations and future research

A limitation of the present study was the lack of a control group. Furthermore, both participants and assessors were not blinded in the present study.

Despite the general increase in PKF and knee range of motion, not all participants in the present study responded equally to hamstring stimulation. The multiple reasons mentioned for stiff knee gait, such as overactivity of the rectus femoris in the swing phase (3–5) or a lack of push off from the gastrocnemius moments (8, 27), may have influenced the effect of hamstring stimulation. In addition, generalization of the present study results to the broader stroke population is difficult, because of the relatively small study population.

Future research with more participants (control group, randomization, blinding) should deepen our understanding about the aetiology of stiff knee gait and evaluate how interventions can influence the causative factors of stiff knee gait.

Conclusion

This exploratory study shows an increase in knee kinematics in the swing phase after functional electrical stimulation of the hamstrings in stroke survivors walking with a stiff knee gait.

The largest improvement in peak knee flexion in the swing phase is seen when participants walked with the hamstring stimulation. Participants with low neurological impairment responded better to hamstring stimulation and there are indications that the effect of hamstring stimulation can be predicted during a single session.

The effect of functional electrical stimulation is comparable to that of more invasive treatment options, such as botulinum toxin (BTX) or soft-tissue surgery. Functional electrical stimulation is therefore a feasible treatment option for daily clinical practice.

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REFERENCES

1. Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. Clin Orthop Relat Res 1993: 139–147.
2. Weerdesteyn V, de Niet M, van Duijnhoven HJ, Geurts AC. Falls in individuals with stroke. J Rehabil Res Dev 2008; 45: 1195–1213.
3. Perry J. Distal rectus femoris transfer. Dev Med Child Neurol 1987; 29: 153–158.
4. 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: 835–840.
5. Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. J Biomech 1996; 29: 723–733.
6. 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: 1189–1196.
7. Kerrigan DC, Bang MS, Burke DT. An algorithm to assess stiff-legged gait in traumatic brain injury. J Head Trauma Rehabil 1999; 14: 136–145.
8. Kerrigan DC, Burke DT, Nieto TJ, Riley PO. Can toe-walking contribute to stiff-legged gait? Am J Phys Med Rehabil 2001; 80: 33–37.
9. 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: 56–61.
10. Perry J. Distal rectus femoris transfer. Dev Med Child Neurol 1987; 29: 153–158.
11. Gage JR, Perry J, Hicks RR, Koop S, Werntz JR. Rectus femoris transfer to improve knee function of children with cerebral palsy. Dev Med Child Neurol 1987; 29: 159–166.
12. Kesar TM, Perumal R, Reisman DS, Jancosko A, Rudolph KS, Higginson JS, et al. Functional electrical stimulation of ankle plantarflexor and dorsiflexor muscles: effects on poststroke gait. Stroke 2009; 40: 3821–3827.
13. Mann G, Burridge J, Ewins D, McLellan D, Swain I, Taylor P. Optimising two-channel stimulation to improve walking following stroke. 5th Annual Conference of the International FES Society; June; Aalborg, 2000.
14. Hanlon M, Anderson R. Prediction methods to account for the effect of gait speed on lower limb angular kinematics. Gait Posture 2006; 24: 280–287.
15. Baker LL, Weedeich CL, McNeal DR, Newsam CJ, Waters RL. Neuromuscular electrical stimulation. A practical guide. 4th edn. Downey, CA: Los Amigos Research and Educaton Institute, Inc.; 2000, p. 169–713.
16. Collen FM, Wade DT, Robb GF, Bradshaw CM. The Rivermead Mobility Index: a further development of the Rivermead Motor Assessment. Int Disabil Stud 1991; 13: 50–54.
17. Holden MK, Gill KM, Magliozzi MR, Nathan J, Piehl-Baker L. Clinical gait assessment in the neurologically impaired. Reliability and meaningfulness. Physical Ther 1984; 64: 35–40.
18. Collin C, Wade D. Assessing motor impairment after stroke: a pilot reliability study. J Neurol Neurosurg Psychiatry 1990; 53: 576–579.
19. Marks MC, Alexander J, Sutherland DH, Chambers HG. Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. Dev Med Child Neurol 2003; 45: 763–768.
20. van Hedel HJ, Tomatis L, Muller R. Modulation of leg muscle activity and gait kinematics by walking speed and bodyweight unloading. Gait Posture 2006; 24: 35–45.
21. Tilson JK, Sullivan KJ, Cen SY, Rose DK, Koradia CH, Azen SP, et al. Meaningful gait speed improvement during the first 60 days poststroke: minimal clinically important difference. Physical Ther 2010; 90: 196–208.
22. Perera S, Mody SH, Woodman RC, Studenski SA. Meaningful change and responsiveness in common physical performance measures in older adults. J Am Geriatr Soc 2006; 54: 743–749.
23. Daly JJ, Roenigk KL, Butler KM, Gansen JL, Fredrickson E, Marsolais EB, et al. Response of sagittal plane gait kinematics to weight-supported treadmill training and functional neuromuscular stimulation following stroke. J Rehabil Res Develop 2004; 41: 807–820.
24. 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 Physical Med Rehabil 2014; 95: 576–587.
25. Namdari S, Pill SG, Makani A, Keenan MA. Rectus femoris to gracilis muscle transfer with fractional lengthening of the vastus muscles: a treatment for adults with stiff knee gait. Physical Ther 2010; 90: 261–268.
26. Lennon S, Ashburn A, Baxter D. Gait outcome following outpatient physiotherapy based on the Bobath concept in people post stroke. Disabil Rehabil 2006; 28: 873–881.
27. 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: 294–300.