

Response of sagittal plane gait kinematics to weight-supported treadmill training and functional neuromuscular stimulation following stroke

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Abstract—After stroke, persistent gait deficits cause debilitating falls and poor functional mobility. Gait restoration can preclude these outcomes. Sixteen subjects (>12 months poststroke) were randomized to two gait training groups. Group 1 received 12 weeks of treatment, 4 times a week, 90 min per session, including 30 min strengthening and coordination, 30 min over-ground gait training, and 30 min weight-supported treadmill training. Group 2 received the same treatment, but also used functional neuromuscular stimulation (FNS) with intramuscular (IM) electrodes (FNS-IM) for each aspect of treatment. Outcome measures were kinematics of gait swing phase. Both groups showed no significant pre-/posttreatment gains in peak swing hip flexion. Group 1 (no FNS) had no significant gains in other gait components at posttreatment or at follow-up. Group 2 (FNS-IM) had significant gains in peak swing knee flexion and mid-swing ankle dorsiflexion ($p < 0.05$) that were maintained for 6 months.

Key words: brain ischemia, electrical stimulation therapy, gait, gait disorders, kinematics, motor learning, neurologic, rehabilitation, treadmill training.

INTRODUCTION

After stroke, persistent gait deficits cause debilitating falls and poor functional mobility. Gait restoration can preclude these outcomes. Under normal circumstances,

limb advancement during swing phase is achieved through flexion of the hip, knee, and ankle joints in the sagittal plane [1]. The normal swing phase limb-flexion pattern helps minimize the energy cost of walking [2–3].

Despite conventional rehabilitation following stroke, persistent swing phase limb-flexion deficits can cause falls, elevated energy cost of gait, and compromised walking

Abbreviations: ANOVA = analysis of variance, BWSTT = body-weight-supported treadmill training, CNS = central nervous system, COM = center of mass, FM = Fugl-Meyer, FNS = functional neuromuscular stimulation, FNS-IM = functional neuromuscular stimulation with intramuscular electrodes, IM = intramuscular, NO-FNS = no functional neuromuscular stimulation, SD = standard deviation, 3-D = three-dimensional, TT = treadmill training, VCM = Vicon Clinical Manager.

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walking endurance [3–5]. Abnormal swing phase after stroke can be characterized by reduced peak flexion values at all or any one of the lower-limb joints; a delay in the flexion at the hip, knee, or ankle; and/or lack of progression of flexion throughout the swing phase at the hip, knee, or ankle [6]. In the presence of these motor impairments, compensatory strategies can result in a number of characteristic gait patterns, including a “stiff-legged” swing phase in which the involved limb is dragged behind the torso [7], circumduction of the involved limb at the hip, hiking or elevating the involved side of the pelvis, vaulting onto the toes during stance on the uninvolved limb, or lateral leaning to the uninvolved side during swing on the involved side [8–9].

One promising gait training method following stroke is the use of body-weight-supported treadmill training (BWSTT). Visintin et al. compared the use of BWSTT with treadmill training (TT) alone for patients in the early months following stroke (mean 73 days) [10]. They found that the group that had BWSTT showed greater gains compared with the group that had TT for active joint movement, basic mobility, walking speed, and walking endurance. Visintin and others reported promising results for BWSTT, although none reported that subjects in the acute phase (<6 months) had gait restoration to normal. Changes in gait kinematics were not reported. Functional neuromuscular stimulation (FNS) with intramuscular (IM) electrodes (FNS-IM) is a second promising gait training intervention after stroke. In prior work, Daly et al. showed that FNS-IM produced gains in volitionally performed swing phase gait components in response to FNS-IM [4,11]. These gait component gains were obtained for subjects in the chronic phase after stroke (>12 months). This work was promising because volitional motor control was restored sufficiently to be reflected in gains in the volitional execution of the complex pattern used in the swing phase of gait. However, these gains were realized only after 6 to 18 months of treatment.

Treatment 6 to 18 months long is not economically practical. Therefore, this study tested response to 3 months of treatment with FNS-IM used in combination with other promising therapies. The treatment protocol consisted of strength and coordination training, over-ground gait training, and BWSTT. One group received no FNS (NO-FNS), while the other group used FNS-IM during all aspects of treatment.

METHODS

Subjects

Sixteen subjects in the chronic phase after stroke (>12 months) were enrolled. Subjects were assigned to one of two treatment groups through a randomization procedure that incorporated stratification according to stroke severity with the use of the Fugl-Meyer (FM) Coordination Scale [12–14]: severely involved, FM = 0 to 17, or moderately involved, FM = 18 to 28; mildly involved were not accepted into the study (FM = 29 to 34). The FM scale was used so that comparable numbers of moderately and severely involved subjects were assigned to the two treatment groups. Additional inclusion criteria were chronic, persistent swing phase gait deficits, characterized by absent or attenuated limb flexion during swing phase; ability to follow two-level commands; and endurance to participate in 90 min of therapy 4 days per week. Exclusion criteria included peripheral neuropathy, acute degenerative diseases of the musculoskeletal or nervous systems, allergy to the electrode materials, and use of a cardiac pacemaker. The following subject characteristics were recorded: stroke type, stroke location, years since stroke, and age. Subjects provided informed consent in accordance with the Declaration of Helsinki, and the study was approved by the Institutional Review Board of the Cleveland Department of Veterans Affairs Medical Center (Committee on Human Subjects Protection in Research).

Treatment Procedures

Subjects were treated for 3 months, 4 sessions per week, 90 min per session. Both groups received the following treatment times: 30 min strength and coordination training, 30 min over-ground gait training, and 30 min BWSTT. One group received NO-FNS. The other group used FNS-IM during all three types of treatment. This treatment protocol was developed from pilot work and past studies [4,11,15].

Strength and Coordination Training

Exercises were provided that targeted the lower-limb flexors. The exercises were designed to improve both strength and coordination of isolated joint movement. An example of isolated joint movement is performance of ankle dorsiflexion throughout its full range without any movement occurring at other joints. The treatment program was progressed in a number of ways. Initially exercises

were performed at a single joint. Then we assigned movement at two joints simultaneously, counter to mass limb flexion or extension. An example of a two-joint exercise is simultaneous ankle dorsiflexion and knee extension. A second example of exercise progression is the following positional sequence for exercises: seated position, standing position, and dynamic conditions. Dynamic conditions included weight shifting and limb movements during weight shifting.

Over-Ground Gait Training

Over-ground gait training included stepping and walking with the use of parallel bars, quad cane, straight cane, and no assistive device (depending on patient ability). Subjects received demonstrations of correct movement patterns, tactile and verbal cues, and practice in self-evaluation of performance accuracy of swing phase gait components.

Body Weight-Supported Treadmill Training

BWSTT was implemented on the BIODEX 500 (Shirley, New York) treadmill and harness apparatus. Subjects were progressed through the protocol by gradually decreasing body weight support as follows: 30 percent, 20 percent, 10 percent, 0 percent. Therapists performed movement analysis to determine the subject's readiness to progress. Criterion for decreasing the body weight support was the subject's ability to maintain normal upright alignment of the torso, pelvis, and stance limb during the stance phase of gait. Initial walking speed was selected with the same criterion and was progressively increased to 2.0 mph. The treadmill protocol began with a 2 min walk. A 5 min rest period was provided, followed by a second 2 min walk. Five iterations of the walk-rest sequence were conducted on the first day (a total of 10 min of walking). At the next visit, each of the five walk periods was increased to 3 min (a total of 15 min), and so on. If the subject was unable to start or progress at this rate, he/she was progressed at a rate that was comfortable. As soon as the subject could walk for two 10 min walks with a 5 min rest in between, the walk and rest periods were increased and decreased, respectively, in an incremental fashion until the subject could walk continuously for up to 30 min.

FNS-IM Intervention

For the group that received FNS-IM, subjects were implanted with IM electrodes in the following muscles:

tibialis anterior, peroneus longus/brevis, short head of the biceps femoris, semitendinosus, and semimembranosus or long head of the biceps femoris. With the use of conscious sedation, a needle insertion technique was used to place the electrodes at the motor point of each muscle. A detailed description regarding the IM electrodes and the implantation protocol appears elsewhere [16–17].

Subjects received FNS-IM and gait training protocols developed by Daly and colleagues [4,11,15]. The protocols in this study were targeted to enhance voluntary control of knee and ankle dorsiflexion during gait swing phase. Therapists used a specialized computer program to individualize the FNS patterns [18]. The subject used his/her own stimulator with the individualized patterns to practice FNS-assisted movements and gait components. The stimulator was controlled with a four-button hand switch. FNS-driven exercise was used to treat weakness, muscle fatigue, and impaired coordination of isolated joint movement. FNS-IM was used to activate muscles in normal combinations and at the appropriate time for gait component practice and gait training.

We evaluated FNS-driven or assisted muscle function and determined acceptable stimulation parameter ranges to produce the first stimulation pattern for a patient. The activation level for each muscle was set first according to patient comfort and then according to the movement desired. For most subjects and most muscles, we used the maximum comfortable level of activation to approximate normal joint movement excursion. For the remainder of muscles, we stimulated at a level well below the maximum comfortable level, since the desired joint movement was obtained at the lesser level. FNS-driven flexion angles for gait training were no greater than normal. Stimulation parameter ranges were 30 Hz, 4 mA to 20 mA, and 5 μ s to 150 μ s pulsewidth.

Outcome Measures: Kinematic Gait Components During Swing Phase

Data Collection and Processing

Data were collected in three sessions: pretreatment, posttreatment, and follow-up (6 months after the end of treatment). At each session, gait kinematics were measured during volitional, over-ground walking at a self-selected speed. During data collection, subjects did not use FNS-IM or orthotic devices.

Gait kinematics were measured with the Vicon 370 (Oxford Metrics, UK), a computerized, three-dimensional

(3-D) video data acquisition system. The system included seven charge-coupled device cameras strategically configured on a 30 ft walkway, a personal computer, and software for collection and initial analysis of the data (Vicon Clinical Manager [VCM]). We used the modified Helen Hayes kinematic model for marker placement [19]. Markers were placed at the following locations: sacrum, anterior superior iliac spines, thighs, lateral epicondyles, shanks, lateral malleoli, and fifth metatarsals, as recommended with use of the Vicon 370 [20]. As the subject walked, the 3-D position coordinates for all the markers were recorded at a sampling rate of 60 Hz.

The Vicon 370 VCM software then reconstructed the motion of the limb segments of the body during walking. At each data collection session, we collected data for 30 strides. The gait events of heel strike and toe-off were identified by the computer operator with the stop-frame feature and frame-by-frame inspection of the stick figure. The kinematic data (referenced to percentage of the gait cycle; e.g., 0% = right heel contact; 100% = subsequent right heel contact) was transferred to MATLAB (The MathWorks, Natick, MA) in an ASCII format. We identified each gait component of interest and calculated mean and standard deviation (SD) for each subject for each gait event of interest (described in the next section) with MATLAB. Blinded evaluators collected and analyzed data.

Three Swing Phase Gait Component Measures and Timing of Performance

Three limb flexion gait components were chosen as indicators of swing phase limb advancement in the sagittal plane: peak swing hip flexion, peak swing knee flexion, and mid-swing ankle dorsiflexion. Peak swing hip flexion was defined as the maximum hip flexion during swing phase. Peak swing knee flexion was defined as the maximum knee flexion during swing phase. Mid-swing ankle dorsiflexion was defined as the point in the gait cycle at which the swing ankle crosses the stance ankle. These gait components were chosen because they are often impaired for patients following stroke and because they responded to FNS-IM in prior work during a longer treatment protocol [e.g., 4,11,15].

Additionally, we investigated whether a posttreatment change occurred regarding the timing of peak hip flexion, peak knee flexion, and mid-swing ankle dorsiflexion. Since the timings of interest were within the swing phase, we normalized the data to percentage of the swing phase.

Data Analysis

Although we stratified subjects into the two treatment groups according to severe or moderate categories of stroke severity, we assessed possible initial group differences by analyzing an analysis of variance (ANOVA) model to compare the actual initial scores of the FM Coordination Scale between the two treatment groups. FM score was the dependent variable and group membership was the independent variable. To further assess possible initial group differences, we compared the two groups regarding the initial values for each of the three swing phase gait components. For example, we analyzed an ANOVA model with initial peak swing knee flexion as the dependent variable and group membership as the independent variable.

For each of the two groups, we compared pre- versus posttreatment values within the groups by analyzing the Wilcoxon Rank Sum model [21]. We chose this model because the sample size was small and the pre-/posttreatment results were not normally distributed. The Wilcoxon Rank Sum Test is useful for repeated measures for a small sample size for which data is not normally distributed [21]. Given the limitations of these data, we did not make a direct comparison of gains between the two treatment groups. Rather, we compared pre- versus posttreatment values for the first group. Then we compared pre- versus posttreatment values for the second group. Similarly, we compared posttreatment and follow-up values.

RESULTS

Initial Subject Characteristics

Subject characteristics are listed in **Table 1**. Fifteen of the sixteen enrolled subjects completed the treatment protocol of the study. One subject was not able to complete the study because his social support system was compromised. Prior to treatment, no significant differences existed between the two treatment groups for age ($F_{1,14} = 1.001, p = 0.34$), years following stroke ($F_{1,14} = 0.044, p = 0.84$), stroke severity (FM score, $p = 0.27$) (**Table 1**), or gait components (peak swing hip flexion, $p = 0.55$, peak swing knee flexion, $p = 0.31$; mid-swing ankle dorsiflexion, $p = 0.55$). Ages for the NO-FNS group were 50, 53, 54, 68, 72, 76, and 81 years. Ages for the FNS-IM group were 43, 46, 62, 62, 62, 65, 67, and 68 years. Years poststroke for the NO-FNS group were 2, 2,

Table 1.
Subject characteristics.

Group	Fugl-Meyer Coordination Scale Score* (Mean \pm SD)	Stroke Type		Stroke Location		Years Poststroke*		Age Range*	
		Ischemic	Hemorrhagic	Cortical	Subcortical	1–2	>2	45–59	60
NO-FNS	19.71 \pm 5.02	6	1	3	4	4	3	3	4
FNS-IM	22.5 \pm 4.28	6	2	3	5	4	4	2	6

*No significant group difference; $p > 0.23$, $\alpha = 0.05$.
SD = standard deviation

NO-FNS = no functional neuromuscular stimulation
FNS-IM = functional neuromuscular stimulation with intramuscular electrodes

2, 3, 4, 4, and 9. Years poststroke for the FNS-IM group were 1, 1, 1, 1, 2, 5, 6, and 15.

Ten subjects were able to complete the follow-up testing at 6 months after the end of treatment, four in the NO-FNS group and six in the FNS-IM group. Healthcare status changes resulted in noncompliance in follow-up testing, including the following: deceased, compromised general health, and compromised health of the caregiver.

Treatment Response

Figures 1 to 5 provide an overall perspective of the results, showing the mean \pm SD of hip, knee, and ankle kinematics for the entire gait cycle for the NO-FNS group (Figures 1 [pretreatment] and 2 [posttreatment]), for the FNS-IM group (Figures 3 [pretreatment] and 4 [posttreatment]), and healthy adults (Figure 5). These data and all the results presented here were obtained exclusively under the condition of over-ground volitional walking (i.e., neither BWSTT nor FNS was used for testing).

For each of the two treatment protocols, Table 2 shows the pre-/posttreatment values of three swing phase gait components. There was no gain in peak swing phase hip flexion in response to either of the two treatment protocols. For the NO-FNS group, there was no significant gain in peak swing knee flexion or mid-swing ankle dorsiflexion. In contrast, in response to FNS-IM, there were significant gains in volitional peak swing knee flexion and volitional mid-swing ankle dorsiflexion. The range of posttreatment peak swing knee flexion values was 16° to 56°. Summarizing, treatment without FNS did not produce a significant gain in any swing phase gait component, whereas FNS-IM produced a significant gain in two swing phase gait components.

Figure 6 illustrates the posttreatment gains in gait kinematics for the group receiving FNS-IM. Figure 6(b) shows the significant gain in peak swing knee flexion. Mean pretreatment peak swing knee flexion ($23.5^\circ \pm$

14.6°) improved to a mean posttreatment value of $33.5^\circ \pm 15.9^\circ$ ($p = 0.02$; normal peak knee flexion = $62.55^\circ \pm 6.14^\circ$ [22–23]). Figure 6(c) shows a significant increase in mid-swing ankle dorsiflexion during swing phase. Mean pretreatment mid-swing ankle dorsiflexion was $-1.3^\circ \pm 9.0^\circ$ and posttreatment was $5.0^\circ \pm 3.9^\circ$ ($p = 0.04$). Normal mid-swing ankle dorsiflexion is $1.72^\circ \pm 4.03^\circ$ [22–23]. The posttreatment mean was 3.28° higher than normal in absolute value (5.0° versus normal, 1.72°), and within 1 SD of normal (normal mean, $1.72^\circ \pm 4.03^\circ$, SD = 5.75°). Figure 6(a) shows the pre- and posttreatment peak swing hip flexion. Mean pretreatment peak swing hip flexion was $24.7^\circ \pm 13.0^\circ$ and posttreatment was $31.3^\circ \pm 10.1^\circ$ ($p = 0.06$). Normal hip flexion is $36.55^\circ \pm 4.24^\circ$ [23].

Table 3 shows the pre-/posttreatment results regarding the timing of performance for peak hip flexion, peak knee flexion, and mid-swing ankle dorsiflexion. For the NO-FNS group, there was no significant change in timing for performance of the three gait components. For the FNS-IM group, there was a significant posttreatment improvement in timing for peak swing hip flexion ($79.7\% \pm 8.6\%$ to $68.9\% \pm 12.6\%$, $p = 0.01$, normal = 70% [1]). For the FNS-IM group, there was no significant posttreatment change for either peak swing knee flexion or mid-swing ankle dorsiflexion (normal peak swing knee flexion = 30%, normal mid-swing ankle dorsiflexion = 54% [1]).

Posttreatment Follow-Up

Table 4 shows the gait component kinematic values for posttreatment versus follow-up for each of the two treatment protocols. At the end of the follow-up period, there was no significant change in any of the three gait components for either the NO-FNS group (p -values ranged from 0.34 to 0.75). The FNS-IM group also showed no change in gait pattern, maintaining their gains (p -values ranged from 0.27 to 0.46).

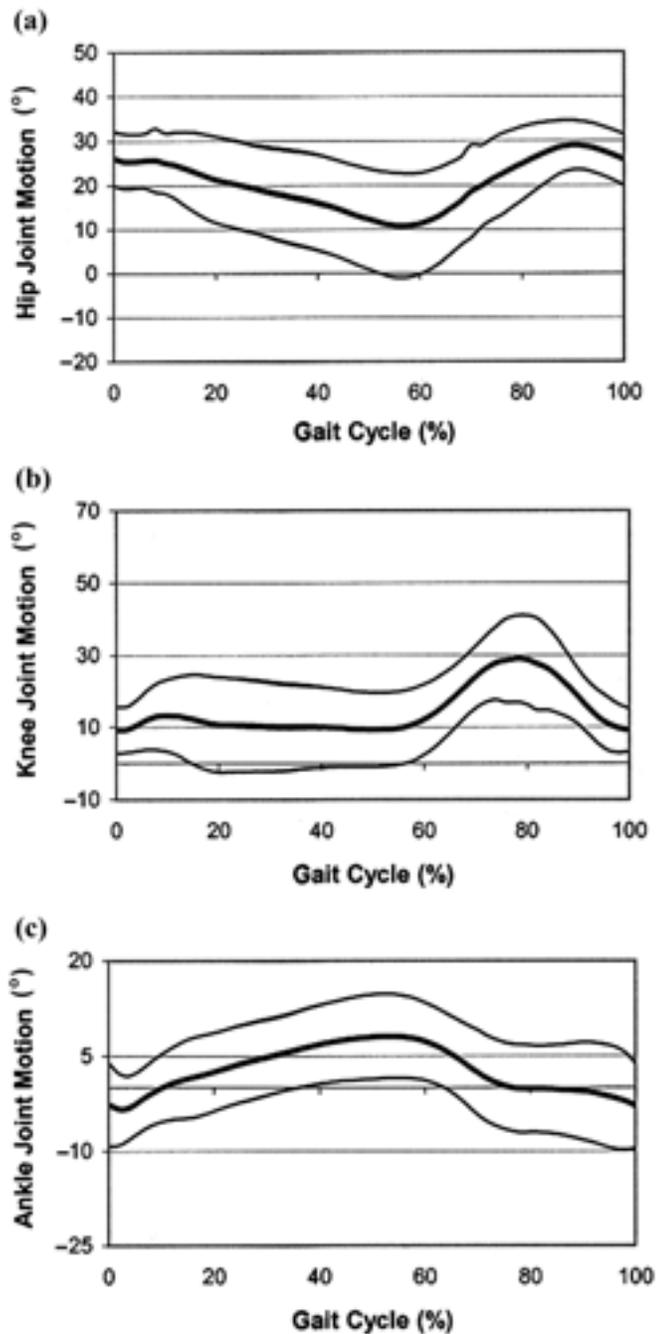


Figure 1. Sagittal plane gait kinematics, pretreatment, Group 1 (no treatment with functional neuromuscular stimulation): (a) hip, (b) knee, and (c) ankle.

DISCUSSION

Requirements for normal gait include muscle strength, coordination, and endurance. In the current study, the first 30 min of each session were strength and

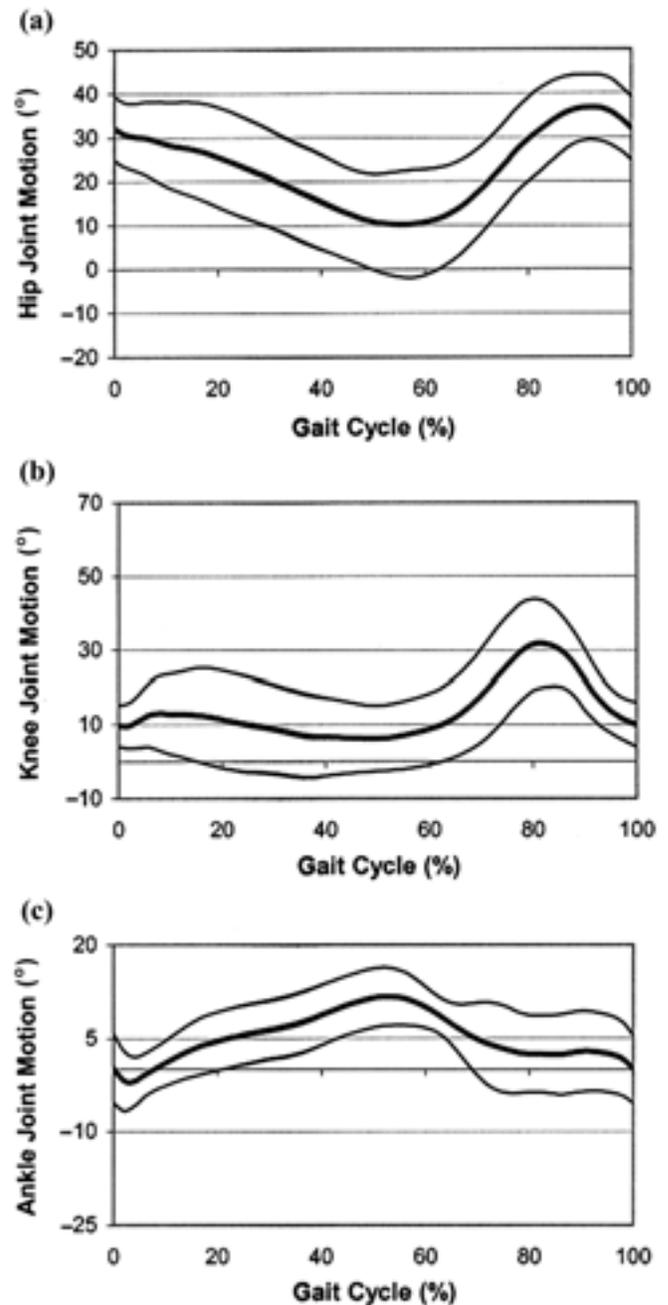


Figure 2. Sagittal plane gait kinematics, posttreatment, Group 1 (no treatment with functional neuromuscular stimulation): (a) hip, (b) knee, and (c) ankle.

coordination training. Increasingly challenging strength and coordination exercises were used to prepare subjects to progress in the motor learning protocol to the more complex movements comprising gait components. The second portion of each session was over-ground gait training. Subjects practiced joint movements in the

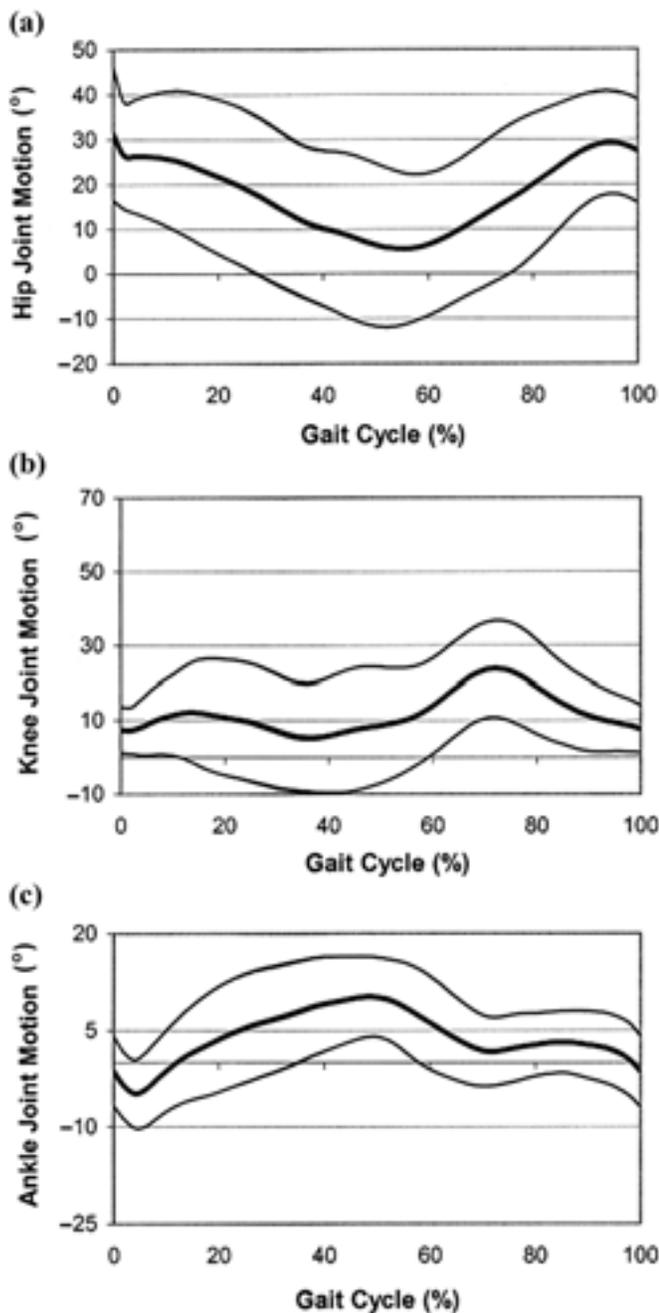


Figure 3. Sagittal plane gait kinematics, pretreatment, Group 2 (treated with functional neuromuscular stimulation [FNS] with intramuscular electrodes): (a) hip, (b) knee, and (c) ankle. No FNS activated for testing.

sequence required for gait swing phase. The third portion of each session included BWSTT, endurance training, and repetition of movement.

Both treatment groups received the advantages of gait practice with BWSTT. BWSTT enabled practice of

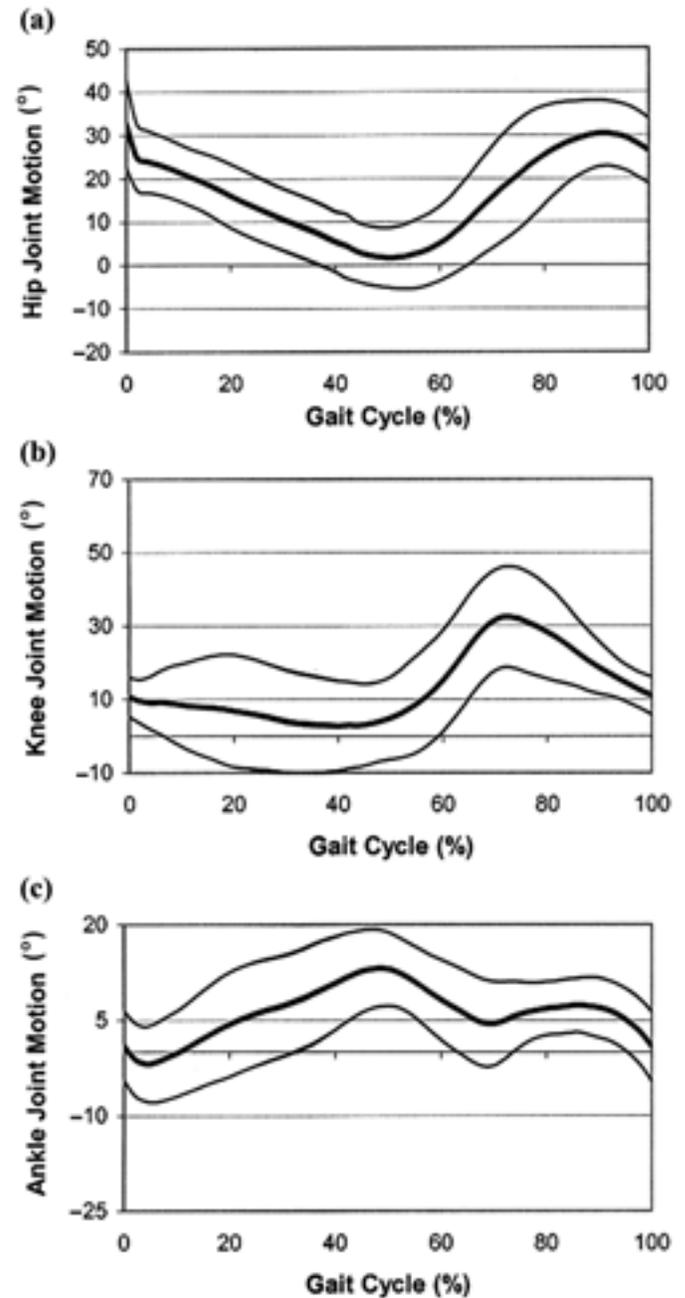


Figure 4. Sagittal plane gait kinematics, posttreatment, Group 2 (treated with functional neuromuscular stimulation [FNS] with intramuscular electrodes): (a) hip, (b) knee, and (c) ankle. No FNS activated for testing.

the following characteristics: more symmetrical gait pattern as indicated by single-limb stance ratio and single-limb loading ratio [24]; more symmetrical weight shifting compared with the use of parallel bars [25]; more symmetrical muscle activation of tibialis and quadriceps,

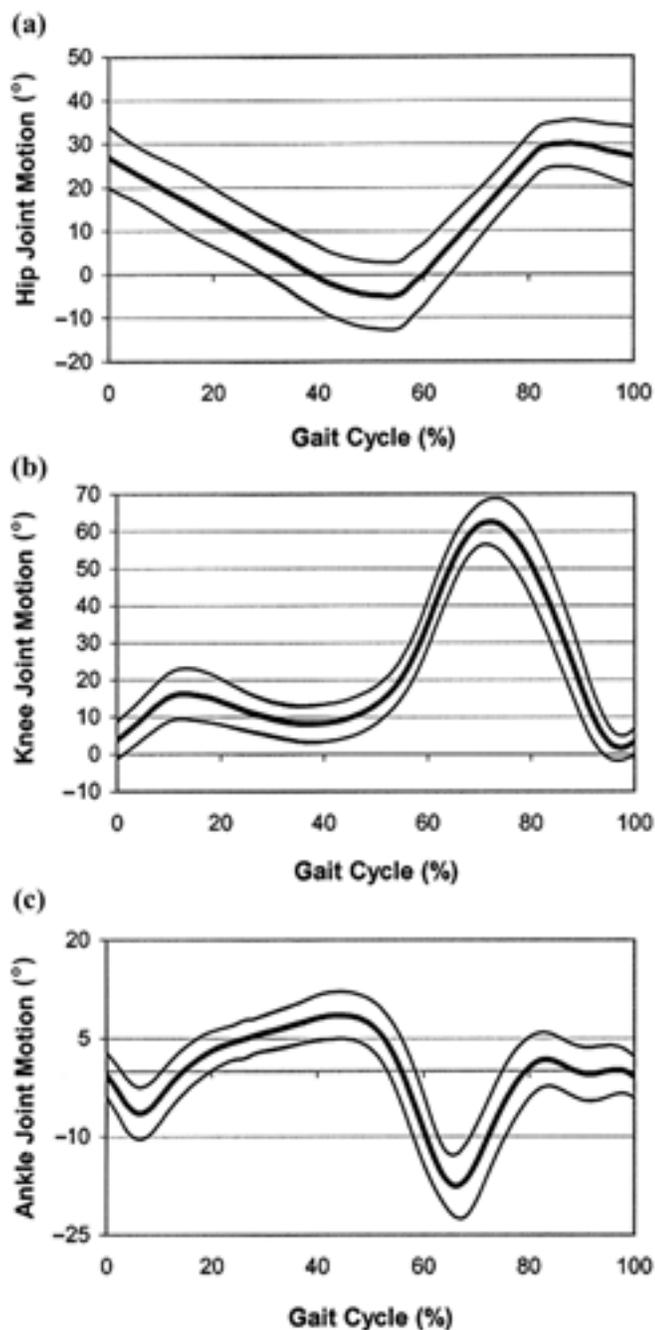


Figure 5.

Sagittal plane gait kinematics, healthy adults: (a) hip, (b) knee, and (c) ankle. Data in graphs obtained from reference source. *Source:* Winter DA. The biomechanics and motor control of human gait. Waterloo, Ontario, Canada: University of Waterloo Press, Dana Porter Library; 1987. p. 121.

according to surface electromyographic signal acquisition [26]; more normal walking training speeds than would be possible with over-ground walking [27]; reduc-

tion in oxygen demand and heart rate compared with over-ground gait training [28]; balance safety, mitigating the fear of falling [29]; and gradations of weight support, walking speed, and duration of walking [10].

Others reported that these advantages of BWSTT produced posttreatment gains in some impairment measures and in some temporal gait characteristics following stroke. For chronic cases (>6 months), researchers reported treatment gains in single-limb and double-limb support times [26] and in walking speed in a 10 m test [27,30]. For subjects in the acute phase, comparative gains with respect to other treatments were reported in response to BWSTT for impairment, disability, and ambulatory status, although spontaneous recovery and treatment times were not controlled [5,31]. In a randomized controlled trial for subjects in the acute phase after stroke, Visintin and colleagues compared BWSTT with TT alone [10]. They found a significant advantage for the BWSTT treatment ($p < 0.03$ for measures of active joint movement and basic mobility and walking endurance) that persisted for 3 months. These studies did not report kinematic gains in gait pattern or restoration of gait to normal. However, consistent with the positive results reported by others, the use of BWSTT probably contributed to the kinematic gains obtained in the current study for Group 2, FNS-IM.

The FNS-IM group demonstrated significant gains in swing phase gait components, while the NO-FNS group did not. A number of reasons might explain this occurrence. During the strength and coordination training in this study, FNS-IM provided treatment advantages not experienced by the NO-FNS group. The usefulness of FNS following stroke has been reported for many years for treating muscle weakness [15,32–40], limited passive range of joint motion [41–43], and limited active range of joint movement [44–45]. In the current study, FNS-IM electrically induced muscle activation and provided a means to strengthen muscles that were not initially under volitional control [4,15]. Similarly, FNS-IM repetitively activated muscles and provided a means to condition muscles that were not initially under volitional control (addressing muscle fatigue). Further, electrically induced muscle activation provided a means to practice coordinated movement of the limb. For example, for normal gait, at late stance, the hip is in an extended position, while knee flexion is simultaneously required in preparation for swing phase. Following stroke, many patients are unable to flex the knee when the hip is in an extended

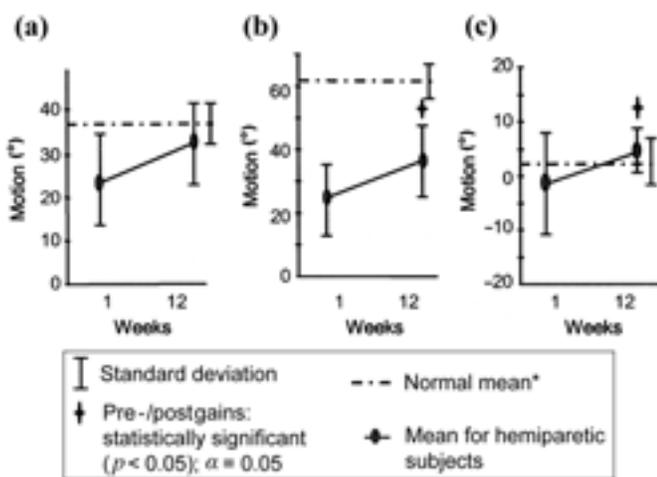
Table 2.

Comparison of 3-month treatment response.

Gait Component	NO-FNS (n = 7)			FNS-IM (n = 8)		
	Pretreatment (Mean ± SD)	Posttreatment (Mean ± SD)	Change	Pretreatment (Mean ± SD)	Posttreatment (Mean ± SD)	Change
Peak Swing Hip Flexion	31.3° ± 5.1°	37.8° ± 7.1°	6.5°, p = 0.07	24.7° ± 13.0°	31.3° ± 10.1°	6.6°, p = 0.06
Peak Swing Knee Flexion	31.0° ± 12.1°	32.4° ± 11.8°	1.4°, p = 0.84	23.5° ± 14.6°	33.5° ± 15.9°	10.0°, p = 0.02*
Mid-Swing Ankle Dorsiflexion	1.3° ± 7.3°	4.6° ± 6.6°	3.3°, p = 0.40	-1.3° ± 9.0°	5.0° ± 3.9°	6.3°, p = 0.04*

Note: All testing was performed with no activation of FNS.
 *Significant gains, p ≤ 0.05.
 SD = standard deviation

NO-FNS = no functional neuromuscular stimulation treatment
 FNS-IM = functional neuromuscular stimulation with intramuscular electrodes treatment

**Figure 6.**

Significant kinematic changes in swing phase following functional neuromuscular stimulation with intramuscular electrodes: (a) change in peak swing hip flexion, (b) gain in peak swing knee flexion, and (c) gain in mid-swing ankle dorsiflexion. *Source for normal mean values: Winter DA. The biomechanics and motor control of human gait. Waterloo, Ontario, Canada: University of Waterloo Press, Dana Porter Library; 1987. p. 121. Murray MP, Mollinger, LA, Gardner GM, Sepic SB. Kinematic and EMG patterns during slow, free, and fast walking. J Orth Res. 1984;2:272–80.

position. Counter to their intention to extend the hip and flex the knee, they either flex both joints or extend both joints. FNS-IM activated muscles counter to the abnormal synergies. In this way, FNS-IM provided the means to practice the limb movement of hip extension and knee flexion that is required for normal gait.

During the over-ground gait training portion of treatment, as well as during BWSTT, FNS-IM provided advantages in gait component practice that the NO-FNS group did not have. First, for those unable to do so, FNS-IM provided the means to practice initiating flexion of

the hip, knee, and ankle at separate times during swing phase. For many patients following stroke, initiating flexion of the involved hip, knee, and ankle at separate times is difficult; rather, flexion of all three joints is initiated simultaneously. In the normal swing phase of gait, initial flexion of the lower-limb joints occurs first at the knee, followed by the hip, and finally the ankle.

A second gait practice advantage was that FNS-IM provided the means to practice flexing the knee and ankle at the appropriate rate during swing phase. For normal swing phase, lower-limb joint flexion occurs at the fastest rate for knee flexion and at a comparatively slower rate for hip flexion (illustrated by the slopes for normal swing phase hip and knee flexion in **Figure 5(a)** and **(b)**). Following stroke, some patients have difficulty with flexing the knee more rapidly than the hip. For example, for the NO-FNS group after treatment, inspection and comparison of **Figure 2(a)** with **2(b)** show that the rate of hip versus knee flexion was very similar. One result of this impairment can be that there is no time to execute sufficient range of knee flexion before full hip flexion is completed, and then the limb can drag on the floor. In that event, the patient may adopt a compensatory strategy such as limb circumduction. For the FNS-IM group after treatment, inspection and comparison of **Figure 4(a)** with **4(b)** show that after treatment the rate of knee flexion was greater for knee flexion versus hip flexion, closer to the normal condition. A third gait practice advantage was that FNS-IM provided the means to practice a more normal extent of joint excursion for hip, knee, and ankle. Following stroke, patients have difficulty performing active limb flexion to the extent required during normal swing phase execution. FNS-IM provided practice of knee flexion and ankle dorsiflexion beyond what the subject was able to produce volitionally and up to the normal joint excursion needed for swing phase. Even though the FNS-IM gait

Table 3.

Percentage of swing phase at which three gait components were performed.

Timing Characteristics for Three Gait Components	NO-FNS (<i>n</i> = 7)			FNS-IM (<i>n</i> = 8)		
	Pretreatment (Mean ± SD)	Posttreatment (Mean ± SD)	Change	Pretreatment (Mean ± SD)	Posttreatment (Mean ± SD)	Change
Percentage of Swing Phase for Peak Swing Hip Flexion	61.4 ± 16.6	57.7 ± 11.9	-3.7, <i>p</i> = 0.09	79.7 ± 8.6	68.9 ± 12.6	-10.8, <i>p</i> = 0.01*
Percentage of Swing Phase for Peak Swing Knee Flexion	23.5 ± 2.7	28.5 ± 6.6	5.0, <i>p</i> = 0.13	31.7 ± 15.9	30.45 ± 13.8	-1.3, <i>p</i> = 0.58
Percentage of Swing Phase for Mid-Swing Ankle Dorsiflexion	43.4 ± 19.5	49.7 ± 21.4	6.3, <i>p</i> = 0.13	55.7 ± 10.0	53.9 ± 14.3	-1.8, <i>p</i> = 0.78

Note: All testing was performed with no activation of FNS.
*Significant gains, *p* ≤ 0.05.
SD = standard deviation

NO-FNS = no functional neuromuscular stimulation treatment
FNS-IM = functional neuromuscular stimulation with intramuscular electrodes treatment

Table 4.

Comparison of 3-month treatment response after 6-month follow-up.

Gait Components	NO-FNS (<i>n</i> = 7)			FNS-IM (<i>n</i> = 8)		
	Posttreatment (Mean ± SD)	Follow-Up (Mean ± SD)	Change	Posttreatment (Mean ± SD)	Follow-Up (Mean ± SD)	Change
Peak Swing Hip Flexion	37.8° ± 7.1°	33.2° ± 2.0°	-4.6°, <i>p</i> = 0.46	31.3° ± 10.1°	37.0° ± 10.3°	5.7°, <i>p</i> = 0.34
Peak Swing Knee Flexion	32.4° ± 11.8°	40.7° ± 9.3°	8.3°, <i>p</i> = 0.27	33.5° ± 15.9°	37.7° ± 11.0°	4.2°, <i>p</i> = 0.34
Mid-Swing Ankle Dorsiflexion	4.6° ± 6.6°	10.0° ± 5.1°	5.4°, <i>p</i> = 0.46	5.0° ± 3.9°	6.6° ± 5.0°	1.6°, <i>p</i> = 0.75

Note: All testing was performed with no activation of FNS.
SD = standard deviation

NO-FNS = no functional neuromuscular stimulation treatment
FNS-IM = functional neuromuscular stimulation with intramuscular electrodes treatment

practice dorsiflexion was not greater than normal, the results indicate that some subjects were volitionally hyperflexing the ankle by several degrees at posttreatment. This could have occurred because the ankle dorsiflexion during swing phase was a newly learned, consciously performed movement. Sometimes a newly acquired skilled movement is executed in a slightly exaggerated manner. Additionally, for some subjects, execution of the entire limb flexion in the sagittal plane was also a newly performed movement. Years of experience had taught them that attempted sagittal plane limb flexion would result in the limb dragging the floor. Some subjects may have overflexed the ankle during sagittal plane limb flexion as extra insurance that the limb would not drag the floor during execution of the newly acquired limb flexion movement pattern in the sagittal plane.

A fourth gait practice advantage was that FNS-IM could activate knee flexors and ankle dorsiflexors within a short time. For chosen-speed walking of adult men, the entire normal swing phase occurs within 430 ms. For that condition, swing phase knee flexion and ankle dorsiflexion are initiated within 25 ms [1,46]. Muscle activation latency in the lower limb is impaired in many patients following

stroke [47–48]. We used FNS-IM activation of muscles during gait practice to produce knee and ankle flexion within 25 ms. A fifth gait practice advantage was that, since FNS-IM could drive a more normal swing phase knee and ankle flexion pattern during gait practice, the subject was able to more normally utilize existing residual motor control. For example, the new practice capability of flexing the knee and ankle in the sagittal plane enabled the subject to practice existing volitional hip flexion in the sagittal plane. This could explain the significant posttreatment improvement in peak hip flexion timing for the FNS-IM group (even though the hip flexors were not treated with FNS-IM). With the resolution of the stiff-legged swing phase pattern, the hip was free to flex at the more normal, earlier time in swing phase. In these ways, the use of FNS-IM during over-ground gait training and BWSTT produced a swing phase movement pattern of the lower limb more closely approximating normal than was otherwise possible. Thus, the subject was able to practice, repetitively, the desired movement pattern for swing phase. Repetitive practice of the desired skilled movement is one prerequisite of successful motor learning [49–51].

One possible theory that can explain restoration of gait components for the FNS-IM group is that activity-dependent central nervous system (CNS) plasticity occurred. Basic science studies support the theory that repetitive practice of progressively more complex movements produces CNS structural and functional changes that then control the more skilled motor behavior. Compelling evidence exists for both normal animal models [52–55] and stroke animal models [54,56–60] to support activity-induced structural and functional CNS change. These animal model studies showed that treatment using complex motor tasks produced significantly greater gains in motor behavior compared with either gross movement practice or no specific movement training.

Evidence also exists to support the theory of activity-dependent CNS plasticity in humans following stroke. For example, Liepert reported that treatment produced associated motor gains and changes in CNS excitability [61–63]. Others found that increasing motor control was associated with an increase in ipsilesional cortex activity around the lesion and a reduction in the contralesional cortex activity [64–65]. More detailed case series studies showed that greater motor recovery in response to treatment was also associated with an increase in activity in the contralateral secondary somatosensory cortex and bilateral cerebellar hemispheres [66–67].

Human and animal studies regarding stroke rehabilitation provide substantial evidence that functional brain reorganization can occur in response to treatment after stroke. A number of variables may influence this process, and “it is not only the number of neurons left, but how they function and what connections they can make that will decide functional outcome” [68, p. 42]. Critical characteristics of rehabilitation are required for the influence of the function and connections of CNS neurons that produce motor recovery after stroke. These characteristics include retraining with movements that approximate normal movements as closely as possible, task-specificity [69–70], intensive practice (repetition) [49–51], focused attention [71–72], and variability in practice [73–74].

The treatment the FNS-IM group received may have provided those prerequisites for the activity-dependent CNS structural and functional changes necessary to enhance control of the complex swing phase, limb flexion pattern. By virtue of the unique modality capabilities described here, FNS-IM enabled the practice of a swing phase pattern more closely approximating normal. This more normal movement pattern was executed during the

specific task (over-ground walking) and repetitively practiced beyond volitional endurance capability (BWSTT + FNS-IM). The FNS stimulus was conducive to focusing attention on the task, through activation of afferent signals. Variability of practice was provided through gradation of FNS-IM assistance for movement, as well as gradation in assistive devices.

In prior work, Daly and colleagues presented evidence that FNS-IM exercise and gait training could produce gains in swing phase gait components in subjects with persistent gait deficits after stroke [4,11]. However, recovery of swing phase gait components required 6 to 18 months. In the current study, the FNS-IM group also received BWSTT. The combination of these treatment modalities could have contributed to the production of significant gains within 3 months. BWSTT + FNS-IM had several gait practice advantages in comparison to the over-ground FNS-IM gait training used in prior work. First, by virtue of the safety harness, fear of falling was mitigated and subjects were better able to attend to and attempt execution of new swing phase movements. Second, with removal of some body weight, subjects could better execute stance phase on the involved side, which then enabled a longer stride on the uninvolved side. The longer stride on the uninvolved side provided the possibility of practice of a more normal swing phase (hip extending and knee flexing). The potential therapeutic modality characteristics of BWSTT and FNS-IM together could have created additive advantages that enabled significant gains in motor control to occur within the shorter 3-month time frame versus the 6- to 18-month duration previously reported for similar results in response to FNS-IM alone [4,11].

Normal swing phase hip, knee, and ankle dorsiflexion are critical for gait safety and optimal energy expenditure during walking [2–3,9,75], partially because normal limb flexion contributes to the execution of a gait pattern that optimizes the center of mass (COM) pathway. That is, normal swing phase minimizes the magnitude of the excursion of the COM [2], in turn minimizing energy expenditure during walking. When a typical stroke, stiff-legged gait was simulated in normal individuals by immobilizing the knee, oxygen consumption during walking was significantly greater than normal walking with normal swing phase knee flexion (2 ml/kg/m vs. 1.5 ml/kg/m). In fact, gait deficits in hemiparetic individuals can elevate the energy cost of walking 1.5 to 2.0 times normal values [76–77]. Thus it is important for patients following stroke to achieve normal knee flexion, as well as normal hip and ankle dorsiflexion.

CONCLUSIONS

For subjects in the chronic phase following stroke, with gait deficits persisting 1 to 15 years, the group receiving FNS-IM demonstrated significant gains in volitionally performed swing phase gait components that were maintained for 6 months after the end of treatment. The NO-FNS group did not demonstrate gains in gait kinematics at either posttreatment or follow-up. The unique modality capabilities of FNS-IM enabled practice of muscle contractions and gait swing phase components that more closely approximated normal versus initial volitional capability.

The observed gains in swing phase gait components for the FNS-IM group were produced within 3 months. Prior treatment protocols of FNS-IM alone required 6 to 18 months of treatment to produce gains in gait kinematics. The shorter treatment response could have resulted from the advantages afforded by combining FNS-IM with BWSTT and exercise.

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