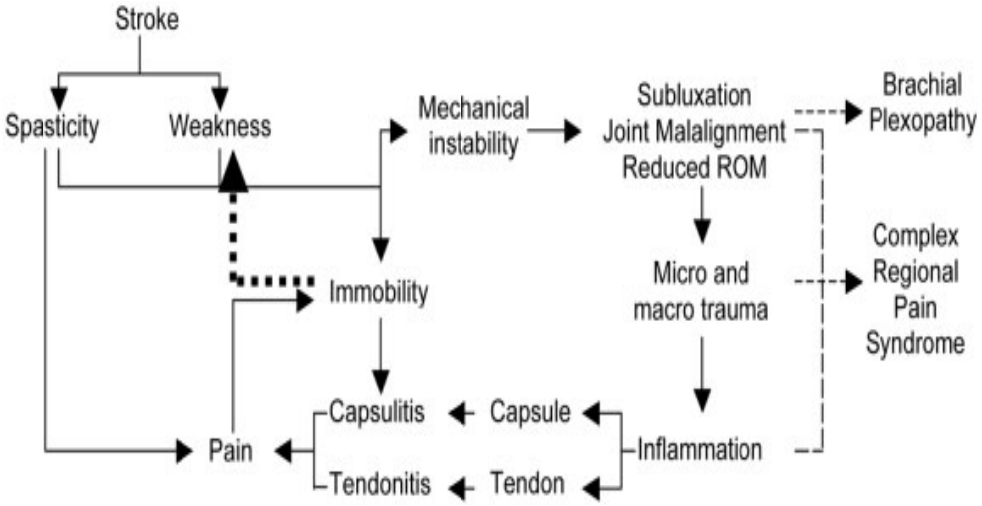
# Model of Shoulder Pain
(Sheffler L, Chae J. Muscle Nerve 2007;35:562-590)




# Model of post stroke shoulder pain
Niessen 2009

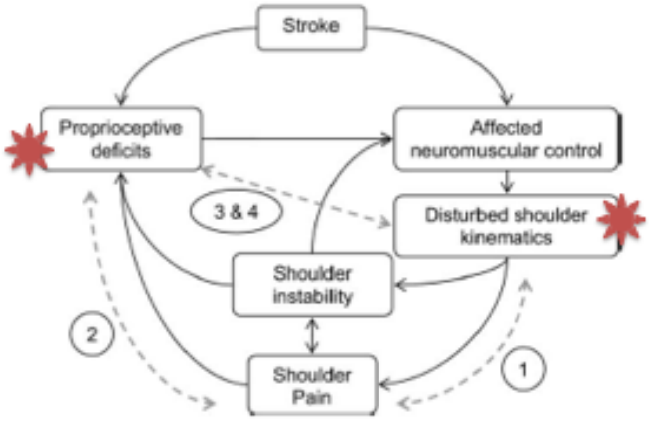



**Fig 5. Placement of the main findings in the model described in the introduction. Gray dotted lines represent a significant relation.**

# Foot Drop Stimulation Versus Ankle Foot Orthosis After Stroke : 30-Week Outcomes

Patricia M. Kluding, Kari Dunning, Michael W. O'Dell, Samuel S. Wu, Jivan Ginosian, Jody Feld and Keith McBride

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Original Contribution

# Foot Drop Stimulation Versus Ankle Foot Orthosis After Stroke

## 30-Week Outcomes

Patricia M. Kluding, PhD; Kari Dunning, PhD; Michael W. O'Dell, MD; Samuel S. Wu, PhD; Jivan Ginosian, MS; Jody Feld, DPT; Keith McBride, DPT

***Background and Purpose***—Drop foot after stroke may be addressed using an ankle foot orthosis (AFO) or a foot drop stimulator (FDS). The Functional Ambulation: Standard Treatment versus Electric Stimulation Therapy (FASTEST) trial was a multicenter, randomized, single-blinded trial comparing FDS and AFO for drop foot among people ≥3 months after stroke with gait speed ≤0.8 m/s.

***Methods***—Participants (n=197; 79 females and 118 males; 61.14±11.61 years of age; time after stroke 4.55±4.72 years) were randomized to 30 weeks of either FDS or a standard AFO. Eight dose-matched physical therapy sessions were provided to both groups during the first 6 weeks of the trial.

***Results***—There was significant improvement within both groups from baseline to 30 weeks in comfortable gait speed (95% confidence interval for mean change, 0.11–0.17 m/s for FDS and 0.12–0.18 m/s for AFO) and fast gait speed. However, no significant differences in gait speed were found in the between-group comparisons. Secondary outcomes (standard measures of body structure and function, activity, and participation) improved significantly in both groups, whereas user satisfaction was significantly higher in the FDS group than in the control group.

***Conclusions***—Using either an FDS or an AFO for 30 weeks yielded clinically and statistically significant improvements in gait speed and other functional outcomes. User satisfaction was higher in the FDS group. Although both groups did receive intervention, this large clinical trial provides evidence that FDS or AFO with initial physical therapy sessions can provide a significant and clinically meaningful benefit even years after stroke.

***Clinical Trial Registration Information***—URL: http://www.clinicaltrials.gov. Unique Identifier: NCT01138995. **(*Stroke*. 2013;44:00-00.)**

**Key Words:** electric stimulation ■ foot drop stimulation ■ gait ■ orthosis ■ rehabilitation ■ stroke



Stroke is one of the most significant causes of disability in adults. Damage to the motor cortex or corticospinal tract often results in contralateral hemiplegia with significant persistent distal weakness. Patients with this pattern of weakness are often unable to actively dorsiflex the foot during the swing phase of gait, which is referred to as drop foot. This gait impairment can result in compensatory movement patterns, slowed gait velocity, limited functional mobility, and increased risk of falls.[1–3]

The traditional treatment for persistent drop foot is an ankle foot orthosis (AFO) that holds the foot in a neutral position. The most common type of AFO is a solid plastic brace, although it may be made of metal or composite materials, with any number of modifications, including an articulated or hinged ankle joint. In general, AFOs have been found to support ankle dorsiflexion during swing phase and improve knee stability in early stance phase in individuals with drop foot.[2,4] However, there are several significant disadvantages of AFOs such as limited ankle mobility that may contribute to the development of contracture[4,5] and difficulty with standing from a chair,[6] along with discomfort and unfavorable aesthetics.[7]

An alternative to the more traditional AFO is the use of functional electric stimulation. Foot drop stimulators (FDS) use functional electric stimulation to stimulate the common peroneal nerve, activating the muscles that dorsiflex the foot during the swing phase of gait. The effect of FDS or an AFO on gait can be measured in several ways, but conflicting terms have previously been used in the literature.[8,9] We

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**The online-only Data Supplement is available with this article at http://stroke.ahajournals.org/lookup/suppl/doi:10.1161/STROKEAHA.111.000334/-/DC1.**

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***Stroke*** **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.111.000334**

have attempted to provide clear operational definitions that describe the scope and timing of comparison,[10] as illustrated in Figure 1. The immediate effect refers to changes in gait that occur when initially wearing the device. A training effect above and beyond the immediate effect may occur as the patient uses the orthosis or FDS over time. The therapeutic effect refers to improvements in walking seen even without wearing an orthosis or FDS and may result from changes in neural plasticity, peripheral strength, cardiopulmonary system, or other systems. The total effect refers to the changes in gait that occur over time, and encompasses both the immediate and training effects.

The results of past FDS studies in stroke have had generally positive results on these different effects, using quasi-experimental and within-subject study designs.[11–13] Increased gait speed has been found consistently in patients with stroke comparing no orthotic with use of both FDS[9,14–16] and AFO.[17,18] Small, short-term studies using a within-subjects comparison of AFO and FDS in stroke found that both devices increased gait speed after 8 weeks.[11,13] The only long-term study of FDS on gait speed found a pattern of significant improvement even at 11 months in participants with a nonprogressive disorder (ie, stroke).[8] The only randomized controlled trial on AFO use in stroke found no significant improvement in gait speed after 3 months.[19] To date, no randomized controlled trials have directly compared surface FDS with AFO in people with drop foot after stroke. However, a randomized controlled trial that compared an implantable peroneal nerve stimulator showed significantly increased gait speed compared with the AFO/control group.[20]

The Functional Ambulation: Standard Treatment versus Electric Stimulation Therapy (FASTEST) trial was designed to compare FDS and AFO for drop foot among people ≥3 months after stroke, with a gait speed ≤0.8 m/s. This was a multicenter, randomized controlled, single-blinded trial. We hypothesized that after 30 weeks, participants randomized to the FDS group would demonstrate greater improvement in gait speed than participants randomized to the AFO group. This hypothesis was based on the anticipated total device effects, encompassing both the immediate and training effects, from the results of previous studies showing positive long-term effects of FDS on gait speed.[8,20] Other comparisons illustrated in Figure 1 were also assessed.

## Methods

A detailed description of the trial design and the methods have been published previously,[10] with a brief summary provided here. Participants ≥3 months after stroke with gait speed ≤0.8 m/s were randomized to 30 weeks of wearing either a surface FDS (treatment group) or a standard AFO (control group). At 30 weeks, the control group crossed over to receive an FDS and was followed for an additional 12 weeks, whereas the original treatment group continued to use their FDS. This article reports on the primary and secondary outcomes at 30 weeks, before crossover.

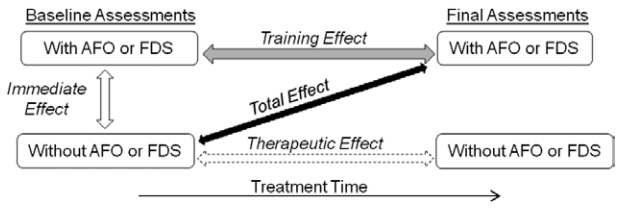


**Figure 1.** Illustration of comparisons of effect of ankle foot orthosis (AFO) or foot drop stimulator (FDS) on gait.

### Participant Screening and Randomization

Participants were recruited at 11 clinical sites across the United States (see the online-only Data Supplement). Each site obtained Institutional Review Board approval, and informed consent was obtained before any study procedures.

Inclusion and exclusion criteria are presented in Table 1. The screening process included assessment by an independent orthotist and a physical therapist to verify that each participant demonstrated drop foot requiring an AFO, and to determine whether his or her current AFO was appropriate based on best practice points as described by a consensus document published by the International Society for Prosthetics and Orthotics,[21] as well as Medicare reimbursement guidelines. If the participant did not have an AFO, a new custom-made AFO was prescribed by the site team and paid for by the sponsor. If the current AFO needed modification, those modifications were prescribed by the site team and paid for by the sponsor. The specific type of AFO (eg, solid ankle, hinged, etc) prescribed for each participant was left to the discretion of the study team of each site. This process ensured that all subjects had an appropriate AFO when needed during the study, and occurred before randomization.

Once study eligibility was confirmed, random group assignment was performed by the sponsor using a web-based application prepared by the study statistician (S.W.). Covariate adaptive randomization[22] was used to ensure balanced group allocation at each site for age and time after stroke and known demographic confounders, within 4 subgroups: 3 to 6 months after stroke, >6 months after stroke, <65 years of age, and ≥65 years of age or Medicare beneficiary. For each new participant, the Web-based application determined imbalance corresponding to its covariate characteristics based on cumulative distribution of assignments up to that point. If there was assignment imbalance, the subject was allocated to the under-represented group with a $P$ value of 2/3; otherwise, the subject was randomized with equal probability.

### Interventions

During the first 6 weeks of the study, both groups received 8 dose-matched sessions of physical therapy (PT) led by a licensed physical therapist who had received training and competency assessment in the use of FDS. Regardless of group assignment, the first 2 to 4 therapy visits focused on education on device use (AFO or FDS), initial gait training, and an individualized home exercise program. The remaining sessions of PT focused on gait training with the assigned device.

#### *FDS Group*

The FDS used in this study was the NESS L300 Foot Drop System, manufactured by Bioness Inc. (Valencia, CA). The L300 comprised a functional stimulation cuff with integrated stimulation unit and electrodes, a control unit, and an in-shoe pressure sensor. The unit is initially configured by a clinician using a handheld computer interface. The pressure sensor detects heel off and initial contact events during gait. It transmits wireless signals to the stimulation cuff, which initiates/pauses the stimulation of deep and superficial branches of the peroneal nerve via 2 surface electrodes. The foot dorsiflexors and evertors are, therefore, activated to ensure foot clearance during the swing phase and prevent excessive ankle inversion during early stance, respectively.

Standardized protocols derived by the sponsor from >5 years of market experience were used by all sites for initial fitting of the FDS, gait training, wearing schedule, home exercise program, and participant education. Written skin care guidelines were reviewed and issued to the participant during the initial fitting and reviewed throughout the training period.

#### *AFO Group*

Education on use, care, gait training, home exercise program, and maintenance of the AFO was provided, along with a wearing

**Table 1. Eligibility Criteria**

| Inclusion Criteria | Exclusion Criteria |
|---|---|
| At least 1 stroke ≥3 mo before study enrollment, resulting in drop foot | Fixed ankle contracture at ≥5 degrees of plantar flexion in the hemiplegic leg with the knee extended |
| Ankle dorsiflexion response with test stimulation in sitting and standing, and adequate ankle and knee stability during gait with test stimulation | Pain in the affected leg, rated ≥4 on a 10-point visual analog scale |
| Medically stable | Participating in PT, OT, new exercise program, or any other interventional clinical research studies without the sponsor's approval |
| Score ≥24 on the Mini Mental State Examination (MMSE), or have a competent caregiver if <24. | Botulinum toxin to the hemiplegic leg or arm within the past 6 wk or planned during the course of the study |
| Age ≥18 y or older | Expectation of a significant change in oral medications for spasticity |
| Able to walk ≥10 meters with a maximum of 1 person assist | Complete lower extremity hemisensory loss |
| Self-selected gait speed ≤0.80 m/s without orthotic effect | Use of any FDS device for foot drop for an accumulative >3 h within the last 6 mo before study enrollment |
| | Any electric or metallic implant; significant swelling/edema in the lower leg; chronic skin problems or cancerous lesion in close proximity to the site of FDS stimulation; pregnant or plan on becoming pregnant; unstable seizure disorder; orthopedic conditions that would affect ambulation; major untreated depression |

FDS indicates foot drop stimulator; PT, physical therapy; and OT, occupational therapy.

schedule when needed (eg, new AFO). It is impossible to implement a sham control treatment for FDS because the participant can feel the stimulation and see their foot move. Moreover, some form of drop foot intervention is necessary for safe walking. Therefore, control participants received surface sensory stimulation with a transcutaneous electric nerve stimulation (TENS) device at each PT visit during the first 2 weeks. TENS intensity was set at the lowest stimulation level that yielded a sensory response without motor response, at a frequency of 100 pps and duration of 200 μsec.

## Outcomes

Repeated outcome measures were obtained at baseline and after 6, 12, and 30 weeks. For baseline and 30-week sessions, including testing both with and without the device, see Figure 1. Well visit follow-ups were performed at weeks 16, 20, and 24, which included fall questionnaires and skin/AE assessment only.

Outcome testing was performed by physical therapists blinded to group assignment. The therapists all received training and passed an on-site competency test for consistency in outcomes assessment. To maintain blinding, a large piece of vinyl fabric was secured over the lower leg and shoe on the involved lower extremity to conceal the device and pressure sensor. All subjects wore an FDS control unit on their belt, regardless of group assignment.

### *Primary Outcome*

Comfortable and fast walking speed were assessed was assessed with a 10-meter walk test.[23] Walking speed has been shown to be an important predictor of community ambulation, functional status, and survival.[24–26] The most commonly used assistive device at the time of assessment was used and documented, along with documentation of the amount of assistance provided.

### *Secondary Outcomes*

Additional outcome measures were included, encompassing the breadth of the International Classification of Function model.[27] These included a measure of body structure and function (lower extremity Fugl-Meyer), several activity measures to assess functional mobility (Timed up and go), walking endurance (6-minute walk test [6MWT]), and balance (Berg balance scale; Functional reach test), and a participation-level measure (Stroke Impact Scale). All outcome measures are valid and reliable in people with stroke.[23,28–36]

Step activity monitors were worn on the uninvolved leg during all waking hours for 7 consecutive days in weeks 6 and 24 to quantify the amount of walking at home and in the community (StepWatch by Orthocare Innovations, LLC).[37]

A user satisfaction survey[16] was completed at week 12 (after completion of PT sessions) and again at week 30 in both groups. This 12-item survey had a total range of scores from 0 to 24, with a higher number indicating greater satisfaction with the device.

### *Adverse Events and Falls*

The cumulative frequency and severity of adverse events (AEs), number of events per subject, and percentage of subjects experiencing an AE were reported from randomization to the 30-week visit. Fall incidence was obtained by self-report from participants and their caregivers retrospectively 6 months before baseline and at each study visit during the 30-week intervention period. Circumstances regarding each fall were collected, including any injury or medical attention received.

## Statistical Analysis

### *Sample Size and Power Analysis*

The original power analysis for this study resulted in the plan to enroll 176 eligible participants, allowing for a 25% dropout rate, which would result in 132 participants who would complete the study. This was estimated to provide 80% power to detect a clinically meaningful (0.1 m/s)[38] difference in walking speed change between groups using a 2-sample *t* test with a 2-sided 0.05 level. After the first planned interim analysis (September 2011), the enrollment goal was increased to 206. This increase allowed for (1) the addition of a primary hypothesis for a subgroup of persons with initially severe gait (<0.4 m/sec gait speed), and (2) the reduction of the risk of type II errors on several secondary outcomes. As a result of favorable trends in outcomes for participants with severe gait impairment, a hypothesis was added that participants in this subgroup randomized to the FDS group would demonstrate greater improvement in gait speed than those randomized to the AFO group. The sponsor elected to close enrollment at 197 participants.

### *Data Management and Quality*

A secure Web-based electronic data capture system (Medidata Rave) was used for clinical data collection and management. Third party monitors performed regular visits at each site to review and verify all study data in source documents.

### Data Analysis

Differences in demographic and baseline variables between groups were analyzed using $t$ test or $\chi^2$ test. Variables found to be significantly different between groups were used as covariates in the final analyses, in addition to the prespecified covariates of study site and whether a new AFO prescription was provided at study entry.

The primary intent-to-treat analysis involved 2 tests: 1 for the entire sample and the other for the severe subgroup. The study-wide error rate was controlled at the 0.05 level by applying the Hochberg step-up procedure.[39] Each statistical test was based on the Fisher combination of 2 $P$ values: 1 from before and the other from after the first interim analysis. Both $P$ values were derived from a linear model investigating whether the groups differ in walking speed improvement from baseline to 30 weeks, after controlling for the aforementioned covariates.

Outcomes for participants who could not complete the 30-week evaluation were imputed by a regression model that takes into account participant dropout bias (described in protocol article).[10] In addition, Wilcoxon rank-sum tests were conducted to compare secondary outcomes between the 2 groups. For simplicity, only the completers were analyzed, and there were no adjustment for covariates. However, the family-wise error rate for all secondary hypotheses testing was controlled at 0.05 level based on Holm step-down procedure,[40] which rejects a hypothesis only if its $P$ value and each of the smaller $P$ values are less than their corresponding critical values.

## Results

### Recruitment, Screening, and Randomization

More than 1200 potential subjects were screened by phone, via chart review, or in person. After initial screening, 389 subjects signed informed consent and participated in further in-person screening (Figure 2). A total of 197 participants were enrolled and randomized.

### Participant Characteristics

Participant characteristics at baseline with between-group comparisons are presented in the online-only Data Supplement. The only significant differences between groups were in categories of sex (greater percentage of females in the treatment group) and stroke type (greater percentage of ischemic stroke in treatment group). Both were used as covariates in all subsequent analyses. It is notable that 118 of 197 (60%) participants received a new or modified AFO at study entry. A description of type of AFO at each site is provided in the online-only Data Supplement.

### Primary Outcome: Gait Speed

At 30 weeks, both comfortable and fast gait speed improved significantly within both the FDS and AFO groups for total effect, as well as training and therapeutic effect ($P$<0.001 for all). In addition, the immediate effect was also significant within groups ($P$<0.001). The specific change values are presented in Table 2 for the entire sample and in the online-only Data Supplement for the severe subgroup. However, no significant differences were found between groups for comfortable gait speed improvement for either the entire sample (0.15±0.14 vs 0.14±0.16; $P$=0.78 with Fisher combination test) or in the severe subgroup (0.11±0.14 vs 0.11±0.11; $P$=0.16 with Fisher combination test). Figure 3 illustrates the trajectory of change of the entire sample for comfortable gait speed between groups over time, for both the training effect and the therapeutic effect. No sex-based or racial/ethnic-based differences were present for the primary outcome.

### Secondary Outcomes

All outcome measures had similar patterns of change, with significant improvements noted within both groups but no significant between-group differences. Figure 4 illustrates comparisons for total orthotic effect, immediate orthotic effect, training effect, and therapeutic effect for several of the gait outcomes. Specific values for these changes in the entire sample and the severe subgroup are presented in the online-only Data Supplement. No between-group differences were noted in the number of steps per day, as measured with the step activity monitors at week 6 (1891 steps per day in control group; 2092 steps per day in treatment group) or week 30 (2069 steps per day in control group; 2369 steps per day in treatment group).

### User Satisfaction

The total user satisfaction survey score measured at week 12 (after completion of PT sessions) was significantly higher in the treatment group than the control group (21.9±2.4 versus 19.0±4.4; 95% confidence interval of mean difference, 1.71–3.87; $P$<0.001), and these differences persisted at week 30 (21.8±2.9 versus 19.1±4.0; 95% confidence interval, 1.64–3.74). Analysis of scores for individual items is presented in the online-only Data Supplement.

### Safety/AEs

Twenty serious AEs were reported, but none were related to the study or the device. The frequency and severity of AEs are summarized in the online-only Data Supplement. The total number of AEs was higher ($P$<0.01) in the treatment group: 82 FDS participants reported a total of 219 AEs, 130 (59%) of them related to device/procedure compared with 61 AFO participants who reported a total of 147 AEs, 50 (34%) of them related to device/procedure. However, nearly all of the related AEs were of mild severity (92% for FDS and 96% for AFO). Anticipated skin irritation issues accounted for 51 (40%) of study-related AEs in the treatment group. The number of participants who fell in the 2 groups during the study period was not significantly different, with a greater number of falls experienced in the control group.

## Discussion

FASTEST is the largest randomized controlled trial comparing FDS and AFOs in persons with stroke to date. The hypothesis that participants randomized to the FDS group would demonstrate greater improvement in gait speed than participants randomized to the AFO group was not supported. Rather, the AFO and FDS groups both made statistically and clinically significant gains in gait speed and other outcomes across all domains of the International Classification of Function model. The observed gains were likely because of a composite effect of the devices, motor learning, and the PT intervention provided at the start of the study.

There may be several reasons why there were no between-group differences in the total effect or other comparisons

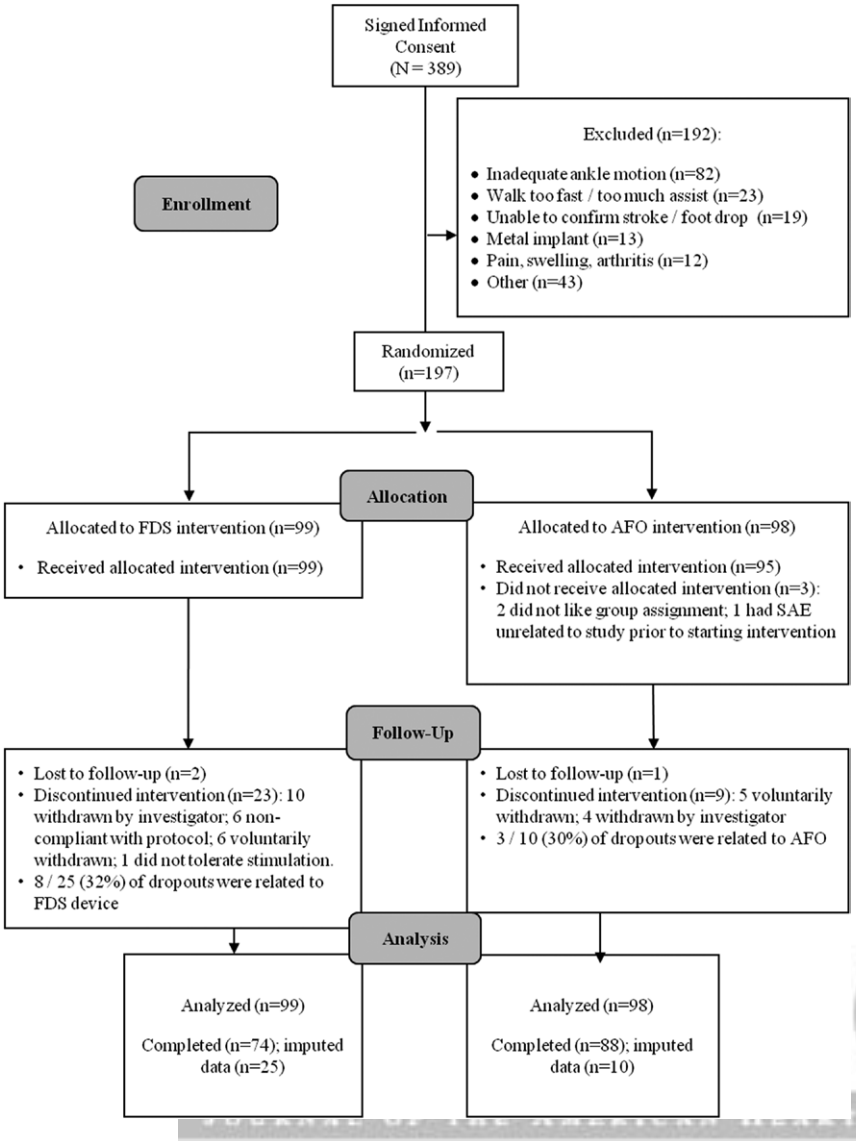


**Figure 2.** Consolidated standards of reporting trials (CONSORT) diagram. AFO indicates ankle foot orthotic; FDS, foot drop stimulator; and SAE, serious adverse event.

contrary to our hypotheses. To protect against selection bias favoring FDS, all participants were evaluated before randomization to ensure that their current AFO was safe and effective. The fact that more than half (60%) of participants enrolled in this study required a new or modified AFO was unexpected because the participants all had drop foot, were community dwelling, and had completed their PT before enrollment. Nonetheless, a majority did not have an AFO that met minimal standards for fitness and safety. Therefore, many individuals in the standard care control group received either a new or modified brace before randomization in addition to receiving the PT intervention. This might have contributed to the unexpected improvements in gait and other outcomes in this group.

Participants in both groups received 8 PT sessions over the first 6 weeks focused on gait training and an individualized home exercise program. As evidenced in Figure 3, the impact of PT may have been particularly prominent in the first 12 weeks after randomization. The PT sessions were essential for initial instruction and gait training with the FDS to maximize effectiveness and safety of gait as well as for monitoring for compliance and skin care. The beneficial impact of PT in persons with chronic stroke is well known.[41–43] Although potentially blunting differences between groups, our results support the value of PT as part of the initial deployment and management of either FDS or AFO for foot drop in patients with stroke.

The control/AFO group also received TENS during the PT treatment sessions in an attempt to provide sensory nerve stimulation as a sham treatment compared with the motor and sensory stimulation experienced with FDS. It is possible that TENS itself contributed to increased gait speed in the control group. Although a systematic review and meta-analysis stated there was insufficient evidence to make conclusions regarding the effectiveness of TENS,[9] several studies have shown increased gait speed after the use of TENS to the lower extremity combined with gait training in people with chronic stroke.[44–48]

It is notable that the immediate effect was statically significant for both devices. This finding speaks to the immediate impact of both devices and the degree of limitation that foot drop entails in persons after stroke and is consistent with

**Table 2. Change in Outcomes by Treatment Group**

| | | Overall (n=197) | Control (n=98) | Treatment (n=99) | *P* Value for Between Groups |
|---|---|---|---|---|---|
| Change in comfortable gait speed, m/s | Long-term device effect | 0.15±0.15* | 0.15±0.14* | 0.14±0.16* | 0.749 |
| | Immediate device effect | 0.08±0.11* | 0.09±0.12* | 0.07±0.10* | 0.180 |
| | Training effect | 0.07±0.11* | 0.06±0.11* | 0.08±0.12* | 0.379 |
| | Therapeutic effect | 0.10±0.14* | 0.09±0.14* | 0.10±0.14* | 0.460 |
| Change in fast gait speed, m/s | Long-term device effect | 0.15±0.17* | 0.17±0.18* | 0.13±0.16* | 0.125 |
| | Immediate device effect | 0.07±0.13* | 0.09±0.15* | 0.05±0.11* | 0.018 |
| | Training effect | 0.08±0.14* | 0.07±0.15* | 0.08±0.14* | 0.711 |
| | Therapeutic effect | 0.05±0.14* | 0.05±0.14* | 0.06±0.13* | 0.466 |
| Change in 6-min walk distance, m | Long-term device effect | 44.7±56.9* | 48.6±51.1* | 40.9±62.1* | 0.341 |
| | Immediate device effect | 22.5±41.2* | 25.8±42.3* | 19.3±39.9* | 0.276 |
| | Training effect | 22.2±44.4* | 22.9±42.5* | 21.5±46.3* | 0.834 |
| | Therapeutic effect | 13.7±46.1* | 11.9±41.9* | 15.6±50.1* | 0.576 |
| Change in Timed up and go (TUG) test, s | Long-term device effect | −5.16±17.66* | −4.38±21.37* | −5.93±13.06* | 0.539 |
| | Immediate device effect | −3.22±13.01* | −3.19±14.34* | −3.26±11.61* | 0.970 |
| | Training effect | −1.93±13.64* | −1.19±15.52 | −2.67±11.51* | 0.447 |
| | Therapeutic effect | −1.27±11.95 | −0.01±13.12 | −2.52±10.58* | 0.140 |
| Change in Berg Balance Scale score | Long-term device effect | 2.86±5.46* | 3.75±4.62* | 1.97±6.08* | 0.022 |
| | Immediate device effect | 1.51±4.07* | 2.12±4.21* | 0.92±3.86* | 0.039 |
| | Training effect | 1.34±4.79* | 1.64±4.25* | 1.06±5.27* | 0.397 |
| | Therapeutic effect | 1.85±4.87* | 2.05±4.57* | 1.65±5.16* | 0.564 |
| Change in functional reach distance, inches | Long-term device effect | 1.10±6.67* | 1.09±6.30 | 1.12±7.05 | 0.969 |
| | Immediate device effect | 0.61±6.43 | 0.83±5.40 | 0.39±7.32 | 0.631 |
| | Training effect | 0.49±6.16 | 0.25±6.48 | 0.73±5.84 | 0.586 |
| | Therapeutic effect | 0.15±7.03 | 0.28±6.84 | 0.03±7.25 | 0.800 |
| Change in Fugl-Meyer Lower Extremity score | Long-term device effect | 0.71±3.42* | 1.04±3.26* | 0.38±3.56 | 0.178 |
| | Immediate device effect | 0.37±2.97 | 0.58±3.31 | 0.16±2.59 | 0.323 |
| | Training effect | 0.34±3.22 | 0.46±3.60 | 0.22±2.81 | 0.607 |
| Change in Stroke Impact Scale (SIS) participation scores | Long-term device effect | 7.79±17.83* | 7.09±17.24* | 8.48±18.47* | 0.587 |
| | Immediate device effect | 1.56±14.86 | 1.51±14.81 | 1.62±14.99 | 0.960 |
| | Training effect | 6.23±16.19* | 5.59±17.85* | 6.86±14.41* | 0.581 |
| Change in SIS mobility scores | Long-term device effect | 5.18±14.78* | 3.19±14.30* | 7.14±15.04* | 0.061 |
| | Immediate device effect | −1.27±11.17 | −2.63±11.77* | 0.08±10.42 | 0.088 |
| | Training effect | 6.45±13.51* | 5.83±13.26* | 7.06±13.79* | 0.523 |

*$P<0.05$ for within group comparison.

other studies that have examined this effect with FDS.[14,16] It is expected that learning to walk with a new device occurs over time, as has been shown with other studies of FDS without a control or comparison group.[8,13,15,16] In the context of this trial, we defined a total effect of device use over time as distinct from a therapeutic effect that reflects change in walking without any device. However, these factors are not mutually exclusive. The therapeutic effect of functional electric stimulation was confirmed with a meta-analysis examining the results of 5 studies on gait speed in patients with stroke,[9] along with more recent studies.[8]

Although AFOs are commonly used to address foot drop after stroke, there is a surprisingly small amount of quality research to support the use of AFOs in neuromuscular disorders as noted by a recent review of literature.[49] The single randomized controlled trial that has been published on this topic did not find clinical or significant improvements in gait speed when comparing a standard polypropylene AFO (set in 5° of dorsiflexion) with a placebo AFO that allowed normal range of motion.[19] However, only 50% of patients in that study complied with wearing the AFO, which seems to confirm the issues related to prestudy AFO use in our study.

With regard to AFO alone, there is little to no data on the biological basis for effectiveness in persons with stroke. Kinematic studies have demonstrated the biomechanical advantage at the ankle, knee, and hip by passively supporting dorsiflexion during the swing phase of gait with an AFO.[2,4] We did observe a significantly improved total device effect for the Berg balance scale in the control AFO group compared with the FDS group. However, the magnitude of

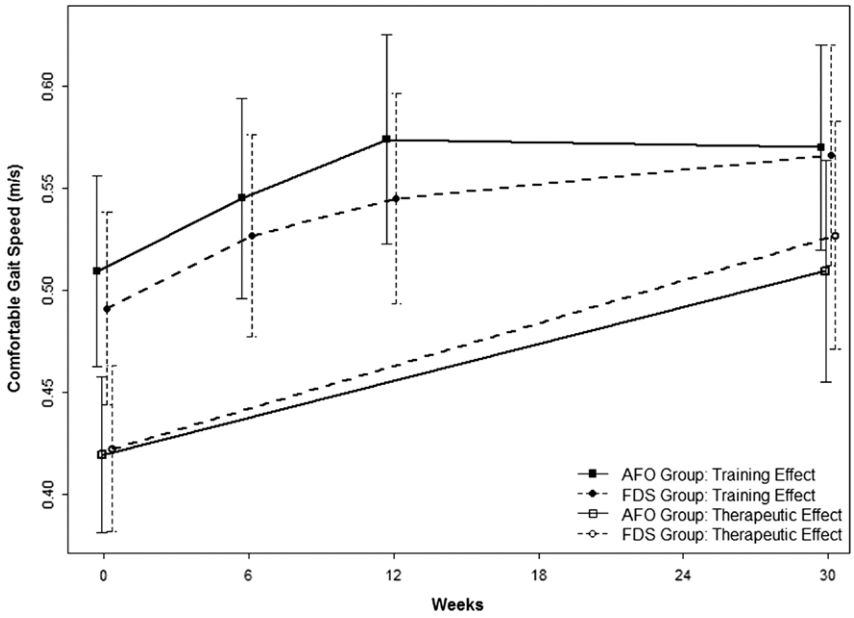


**Figure 3.** Trajectory of change in outcome measures week 0 to 30, illustrating the training effect (solid square and circle) and the therapeutic effect (open square and circle). AFO indicates ankle foot orthotic; and FDS, foot drop stimulator.

change was below the minimal detectable change (beyond measurement variation) in older adults[50] and less than the smallest real difference in people with chronic stroke.[51] The AFO may have mechanical attributes that are amenable for better performance of this test, especially in single limb stance activities, but the lack of difference in falls between groups indicates that this solo finding may have little clinical significance. The biological basis of the therapeutic effect observed in the AFO group in this study could be increased in peripheral muscle strength (unlikely given the relative immobilization of the ankle), neural plasticity, or improved cardiopulmonary conditioning. However, our study is not designed to distinguish these or other mechanisms. It should be noted that the 2005 Stroke Rehabilitation Guidelines from the American Heart Association make clear that AFOs should not replace functional exercise directed at regaining muscle strength and control, which suggests limited therapeutic benefit.[52]

With regard to FDS, there is a more extensive examination of the underlying biological effect.[53] Peroneal nerve stimulation has been found to alter surface electromyographical activity,[54] enhance cortical excitability,[55] and, in the upper extremity, alter activity on functional magnetic resonance imaging.[56] The latter study, and others,[57] also suggest that the combination of voluntary muscle contraction and functional electrical stimulation may be more effective in activating the cortex. Although no similar data exist for AFO, the relatively immobilizing effect of an ankle brace would be theoretically less desirable than the movement allowed with FDS use. In our study, a greater number of AEs were reported in the treatment group. Skin irritation from the FDS electrodes was an anticipated factor that has been previously reported,[58] but the majority of the AEs in both groups were of mild severity.

Although participants in both groups in our study had equivalent improvement in functional outcomes, there was a significant difference in the user satisfaction scores. This is consistent with multiple previously published studies with subjective reports or surveys favoring FDS over AFO.[11,12,58–60] Our user satisfaction survey was identical to that previously used by Hausdorff and Ring,[16] but that specific survey has not been previously used to compare FDS and AFO. Poor compliance with AFOs has been reported in people with foot drop,[19,61] and may have been a factor leading to the lack of adequate use of AFOs in many of the participants at enrollment into the study. Because an impressive improvement was seen with both devices, and nearly all AEs were mild and expected, the issue of compliance may be the single most importance factor in the functional improvements expected over long-term use of a device for foot drop. Although the number of steps per day assessed at 2 points during the trial was similar between groups, this study was not long enough to show the impact of compliance over a long term. An economic comparison of long-term use of AFO versus FDS would also be valuable but was outside the scope of this trial.

The average age of participants in this study was 61 years, which is comparable to the age of other large clinical trials in stroke rehabilitation,[62,63] and an average of 4.5 years after stroke. However, the average age for people hospitalized for stroke is 70 years, and the incidence of stroke increases with age.[64] Age is well known as a predictive factor of mortality and initial recovery,[65] although less is known about the influence of age on rehabilitation in the chronic phase of stroke.

A wide range of outcome measures were used in this trial, with substantive efforts toward standardization and blinding of assessments. However, other outcome measures may also be meaningful in comparing FDS with AFO based on previous research, including obstacle avoidance,[66] ankle dorsiflexion strength,[67] and cortical pathways used for muscle activation.[53] Furthermore, the development of a validated measure of user satisfaction is important to adequately capture the factors that lead to long-term compliance and the subjective experience of the individual with drop foot from stroke.

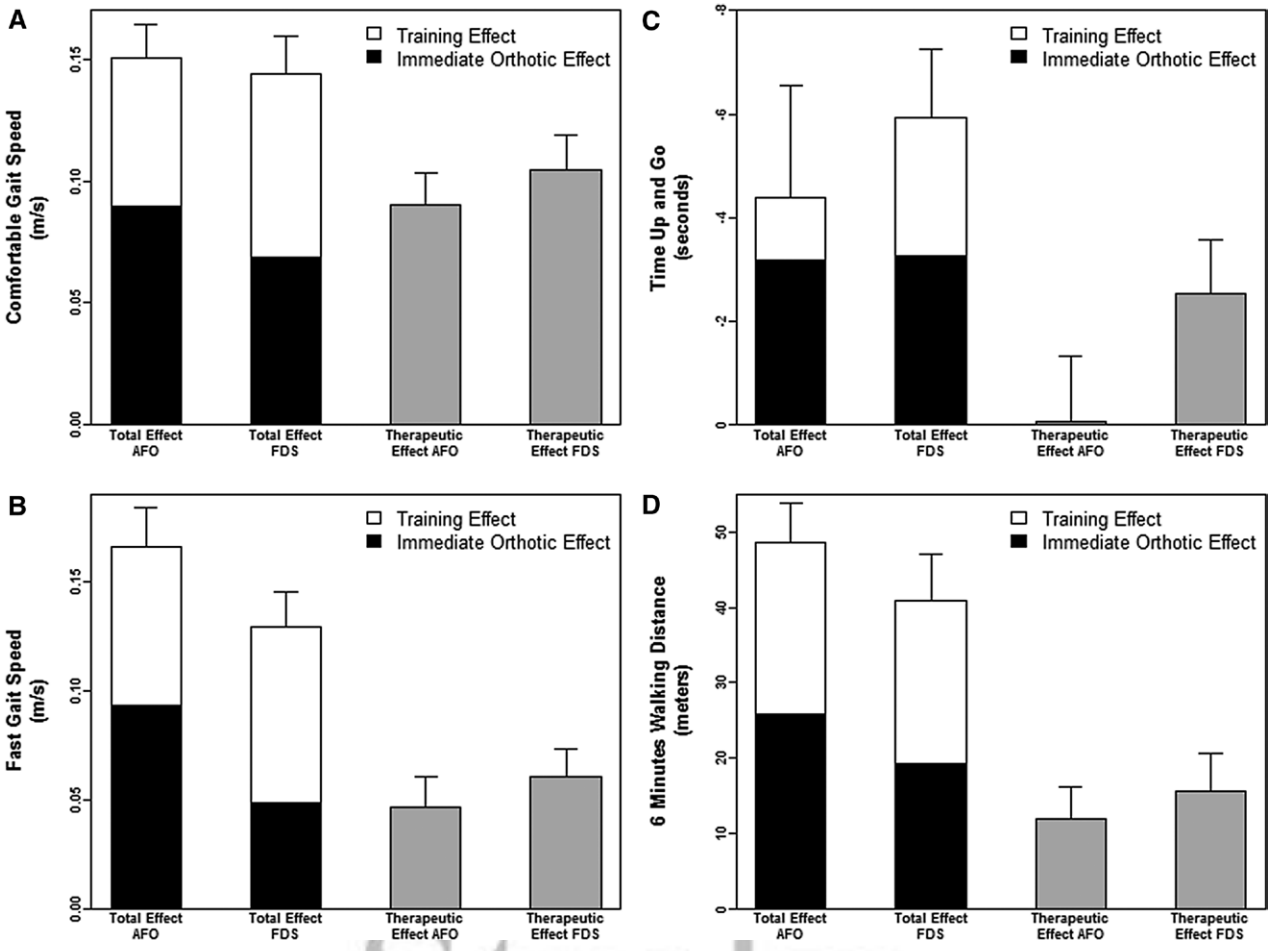


**Figure 4.** Illustration of total effect (immediate orthotic effect plus training effect) and therapeutic effect in ankle foot orthotic (AFO) and foot drop stimulator (FDS) groups for several outcome measures at 30 weeks: change scores for comfortable gait speed (**A**), fast gait speed (**B**), timed up and go (**C**), 6-minute walk distance (**D**).

## Conclusion

We found that an AFO or an FDS used for 30 weeks after stroke had similar effects on gait speed. Still, with effect sizes ranging from 0.93 to 1.00, the FASTEST trial provides encouraging evidence that rehabilitation interventions for drop foot can have a positive impact even many years after stroke. These clinically relevant improvements in gait speed and other functional outcomes have important implications for healthcare reform and insurance coverage policy.

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## Disclosures

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**SUPPLEMENTAL MATERIAL**

LIST OF SUPPLEMENTAL TABLES:



Supplemental Table 1: Study Sites and Number of Participants.

| | **City and State** | **Number of participants enrolled and randomized** |
|---|---|---|
| University of Kansas Medical Center | Kansas City KS | 31 |
| Brooks Rehabilitation Hospital | Jacksonville FL | 24 |
| Sharp Rehabilitation Center | San Diego CA | 24 |
| Weill Cornell Medical College | New York NY | 23 |
| University of Cincinnati | Cincinnati OH | 20 |
| University of Utah Medical Center | Salt Lake City UT | 16 |
| National Rehab Hospital | Washington DC | 15 |
| Rancho Los Amigos National Rehab Center | Downey CA | 15 |
| Univ of Texas Southwestern Medical Center | Dallas TX | 14 |
| Long Beach Memorial Medical Center | Long Beach CA | 8 |
| St. Charles Hospital & Rehabilitation | Port Jefferson NY | 7 |
| **Total number of subjects** | | **197** |

Supplemental Table 2: Comparison of Baseline Variables for All Participants (N=197).

| Variable | | Overall (N=197) | Control (N=98) | Treatment (N=99) | P-value |
|---|---|---|---|---|---|
| Age | | 61.14±11.61 | 61.58±10.98 | 60.71±12.24 | 0.598 |
| Gender | Female | 79 | 31 (39.2%) | 48 (60.8%) | **0.016** |
| | Male | 118 | 67 (56.8%) | 51 (43.2%) | |
| Ethnic | Caucasian | 113 | 59 (52.2%) | 54 (47.8%) | 0.692 |
| | African-American | 47 | 21 (44.7%) | 26 (55.3%) | |
| | Asian | 11 | 6 (54.5%) | 5 (45.5%) | |
| | Hispanic | 16 | 6 (37.5%) | 10 (62.5%) | |
| | Other | 10 | 6 (60.0%) | 4 (40.0%) | |
| Education | Did not complete high school | 15 | 8 (53.3%) | 7 (46.7%) | 0.930 |
| | High school graduate | 48 | 25 (52.1%) | 23 (47.9%) | |
| | College/trade school | 93 | 45 (48.4%) | 48 (51.6%) | |
| | Post-graduate school | 14 | 8 (57.1%) | 6 (42.9%) | |
| | Graduate School | 27 | 12 (44.4%) | 15 (55.6%) | |
| Time Post Stroke | 3-6mo | 17 | 8 (47.1%) | 9 (52.9%) | 0.817 |
| | ≥6mo | 180 | 90 (50.0%) | 90 (50.0%) | |
| | Years from stroke to randomization | 4.55±4.72 | 4.34±4.1 | 4.77±5.29 | 0.523 |
| Stroke Side | Left | 104 | 58 (55.8%) | 46 (44.2%) | 0.074 |
| | Right | 93 | 40 (43.0%) | 53 (57.0%) | |
| Vascular Distribution | MCA | 81 | 36 (44.4%) | 45 (55.6%) | 0.416 |
| | ACA | 6 | 2 (33.3%) | 4 (66.7%) | |
| | PCA | 4 | 1 (25.0%) | 3 (75.0%) | |
| | Basal Ganglia | 32 | 20 (62.5%) | 12 (37.5%) | |
| | Cerebellum | 2 | 1 (50.0%) | 1 (50.0%) | |
| | Brainstem | 11 | 8 (72.7%) | 3 (27.3%) | |
| | Unknown | 37 | 19 (51.4%) | 18 (48.6%) | |
| | Other | 24 | 11 (45.8%) | 13 (54.2%) | |
| Stroke Location | N/A | 20 | 8 (40.0%) | 12 (60.0%) | 0.062 |
| | Cortical | 83 | 35 (42.2%) | 48 (57.8%) | |
| | Subcortical | 94 | 55 (58.5%) | 39 (41.5%) | |
| Stroke Type | N/A | 6 | 2 (33.3%) | 4 (66.7%) | **0.021** |
| | Hemorrhagic | 46 | 31 (67.4%) | 15 (32.6%) | |
| | Ischemic | 145 | 65 (44.8%) | 80 (55.2%) | |
| Hemisensory | Intact | 90 | 44 (48.9%) | 46 (51.1%) | 0.825 |
| | Partial | 107 | 54 (50.5%) | 53 (49.5%) | |

| | | | | | |
|---|---|---|---|---|---|
| Baseline AFO | New AFO prescription | 81 | 38 (46.9%) | 43 (53.1%) | 0.506 |
| | Modified AFO prescription | 37 | 24 (64.8%) | 13 (35.1%) | |
| | No change | 79 | 36 (45.6%) | 43 (54.4%) | |
| Outcome Measures | Comfortable walking speed (m/s) | 0.42±0.20 | 0.42±0.19 | 0.42±0.21 | 0.918 |
| | Fast walking speed (m/s) | 0.56±0.30 | 0.55±0.28 | 0.56±0.32 | 0.877 |
| | Fugl-Meyer lower extremity score | 20.00±4.89 | 19.86±4.60 | 20.14±5.18 | 0.685 |
| | Timed Up and Go (s) | 33.27±25.27 | 32.19±22.61 | 34.34±27.73 | 0.551 |
| | 6 min walk distance (m) | 151.65±82.40 | 151.23±74.36 | 152.07±90.04 | 0.944 |
| | Berg Balance Score | 40.20±9.85 | 40.09±10.68 | 40.31±9.00 | 0.875 |
| | Functional reach (cm) | 21.85±8.27 | 22.11±7.93 | 21.60±8.64 | 0.670 |
| | SIS ADL | 68.31±17.85 | 67.58±17.99 | 69.04±17.77 | 0.566 |
| | SIS Mobility | 73.47±17.86 | 74.59±18.31 | 72.36±17.42 | 0.381 |
| | SIS iADL | 21.52±26.15 | 21.38±26.79 | 21.67±25.63 | 0.938 |
| | SIS Participation | 56.96±23.40 | 55.22±22.64 | 58.67±24.12 | 0.302 |
| Walking Status | Household Ambulators (gait speed 0-0.4 m/s) | 88 | 44 (44.9%) | 44 (44.4%) | 0.949 |
| | Limited Community Ambulator (gait speed 0.4-0.8 m/s) | 109 | 54 (55.1%) | 55 (55.6%) | |
| Functional Ambulation Classification (FAC) Score | 2 (Dependent for Physical Assistance - Level II) | 3 | 1 (1.0%) | 2 (2.0%) | 0.812 |
| | 3 (Dependent for Physical Assistance - Level I) | 21 | 10 (10.2%) | 11 (11.1%) | |
| | 4 (Dependent for Supervision) | 31 | 14 (14.3%) | 17 (17.2%) | |
| | 5 (Independent, Level surfaces only) | 61 | 34 (34.7%) | 27 (27.3%) | |
| | 6 (Independent) | 81 | 39 (39.8%) | 42 (42.4%) | |
| Occupation | Not Working | 82 | 45 (45.9%) | 37 (37.4%) | 0.224 |
| | Employed | 115 | 53 (54.1%) | 62 (62.6%) | |

MCA = middle cerebral artery; ACA = anterior cerebral artery; PCA = posterior cerebral artery; AFO = ankle foot orthotic; SIS = stroke impact scale; ADL = activities of daily living; iADL = instrumental activities of daily living; * P-values were from Chi-square or two sample t-test for categorical or continuous variables, respectively.

Supplemental Table 3: Change in Outcomes by Treatment Group, in Participants with Initial Severe Impairment (N=88).

| | | Overall (N=88) | Control (N=44) | Treatment (N=44) | P value for between groups |
|---|---|---|---|---|---|
| Change in Comfortable Gait Speed (m/s) | Long-term device effect | 0.11±0.13* | 0.11±0.14* | 0.11±0.11* | 0.954 |
| | Immediate device effect | 0.06±0.10* | 0.06±0.11* | 0.06±0.08* | 0.670 |
| | Training effect | 0.05±0.08* | 0.05±0.09* | 0.06±0.08* | 0.567 |
| | Therapeutic effect | 0.04±0.08* | 0.03±0.08* | 0.05±0.08* | 0.236 |
| Change in Fast Gait Speed (m/s) | Long-term device effect | 0.12±0.15* | 0.12±0.17* | 0.12±0.13* | 0.999 |
| | Immediate device effect | 0.07±0.11* | 0.08±0.13* | 0.07±0.10* | 0.676 |
| | Training effect | 0.05±0.10* | 0.05±0.11* | 0.06±0.09* | 0.639 |
| | Therapeutic effect | 0.03±0.09* | 0.01±0.10 | 0.04±0.07* | 0.081 |
| Change in 6 minute walk distance (m) | Long-term device effect | 31.2±43.5* | 34.8±48.2* | 27.7±38.4* | 0.444 |
| | Immediate device effect | 19.6±31.2* | 21.2±38.1* | 17.9±22.8* | 0.628 |
| | Training effect | 11.7±31.3* | 13.6±33.7* | 9.7±28.9* | 0.563 |
| | Therapeutic effect | 2.6±27.2 | -3.0±29.7 | 8.2±23.5* | 0.054 |
| Change in Timed up and go (TUG) (s) | Long-term device effect | -8.91±24.62* | -6.02±31.80 | -11.80±14.05* | 0.274 |
| | Immediate device effect | -5.90±18.52* | -5.60±20.27 | -6.21±16.81* | 0.879 |
| | Training effect | -3.01±18.42 | -0.42±22.48 | -5.59±12.95* | 0.190 |
| | Therapeutic effect | -1.38±17.48 | 1.20±19.16 | -3.96±15.42 | 0.167 |
| Change in Berg Balance Scale score | Long-term device effect | 3.65±5.61* | 5.03±4.94* | 2.28±5.95* | 0.021 |
| | Immediate device effect | 1.95±4.33* | 3.42±4.08* | 0.48±4.11 | 0.001 |
| | Training effect | 1.70±4.42* | 1.61±4.49* | 1.80±4.39* | 0.841 |
| | Therapeutic effect | 1.97±5.44* | 2.63±4.78* | 1.30±6.02 | 0.256 |
| Change in Functional Reach distance (inches) | Long-term device effect | 1.10±6.41 | 1.34±5.71 | 0.86±7.10 | 0.725 |
| | Immediate device effect | 1.20±6.28 | 1.58±5.25 | 0.82±7.21 | 0.572 |
| | Training effect | -0.10±5.79 | -0.24±6.12 | 0.04±5.50 | 0.822 |
| | Therapeutic effect | 0.30±6.31 | 0.82±5.71 | -0.21±6.87 | 0.449 |

| | | | | | |
|---|---|---|---|---|---|
| Change in Fugl-Meyer lower extremity score | Long-term device effect | 0.59±3.52 | 1.11±3.56* | 0.07±3.43 | 0.164 |
| | Immediate device effect | 0.28±3.05 | 0.89±3.31 | -0.32±2.67 | 0.064 |
| | Training effect | 0.31±3.16 | 0.23±3.54 | 0.39±2.76 | 0.815 |
| Change in SIS (Stroke Impact Scale) participation scores | Long-term device effect | 7.61±16.76* | 5.12±16.48* | 10.10±16.86* | 0.164 |
| | Immediate device effect | 1.81±14.67 | 0.07±15.32 | 3.54±13.95 | 0.270 |
| | Training effect | 5.81±16.66* | 5.05±17.79 | 6.56±15.62* | 0.672 |
| Change in SIS mobility scores | Long-term device effect | 5.00±15.16* | 3.18±16.68 | 6.82±13.42* | 0.263 |
| | Immediate device effect | -2.37±11.41 | -3.35±12.58 | -1.38±10.16 | 0.423 |
| | Training effect | 7.37±12.33* | 6.53±13.82* | 8.20±10.73* | 0.528 |

Supplemental Table 4: Ankle Foot Orthotic (AFO) Type at Each Site.

| Site | All | Articulated (N=126) | Non-articulated (N=55) | Pre-fabricated or other (N=14) | P-value from Chi-squared test |
|---|---|---|---|---|---|
| University of Kansas Medical Center | 31 | 25 (80.6%) | 4 (12.9%) | 2 (6.5%) | <0.0001 |
| Brooks Rehabilitation Hospital | 24 | 13 (54.2%) | 9 (37.5%) | 2 (8.3%) | |
| Sharp Rehabilitation Center | 24 | 23 (95.8%) | 1 (4.2%) | | |
| Weill Cornell Medical College | 23 | 10 (43.5%) | 8 (34.8%) | 5 (21.7%) | |
| University of Cincinnati | 20 | 7 (35.0%) | 13 (65.0%) | | |
| University of Utah Medical Center | 16 | 14 (87.5%) | 1 (6.3%) | 1 (6.3%) | |
| National Rehabilitation Hospital | 15 | 3 (20.0%) | 9 (60.0%) | 3 (20.0%) | |
| Rancho Los Amigos NRC | 15 | 12 (85.7%) | 2 (14.3%) | | |
| UT Southwestern Medical Center | 14 | 8 (61.5%) | 5 (38.5%) | | |
| Long Beach Memorial Med. Center | 8 | 5 (62.5%) | 3 (37.5%) | | |
| St. Charles Hospital & Rehab | 7 | 6 (85.7%) | | 1 (14.3%) | |
| **Variable** | | **Mean±SD** | **Mean±SD** | **Mean±SD** | **ANOVA P-value** |
| Comfortable Gait Speed Change from Baseline to 30-weeks | | 0.16±0.15 | 0.12±0.14 | 0.09±0.13 | 0.063 |

Supplemental Table 5: User Satisfaction Survey Results.

| Item description and score assignments | | Control | Treatment | P-value |
|---|---|---|---|---|
| 1. How do you feel about continuing with use of the brace? | Unenthusiastic (0) | 23 (25.3%) | 3 (3.5%) | <0.0001 |
| | Indifferent (1) | 27 (29.7%) | 6 (7.1%) | |
| | Enthusiastic (2) | 41 (45.1%) | 76 (89.4%) | |
| 2. How would you rate the brace against other aids to assist your gait? | Less Useful (0) | 8 (8.9%) | 1 (1.2%) | <0.0001 |
| | As Useful (1) | 32 (35.6%) | 11 (12.9%) | |
| | More Useful (2) | 50 (55.6%) | 73 (85.9%) | |
| 3. How would you describe your walking ability since using the brace? | Worse (0) | 3 (3.3%) | 1 (1.2%) | 0.057 |
| | Same (1) | 13 (14.3%) | 4 (4.7%) | |
| | Better (2) | 75 (82.4%) | 80 (94.1%) | |
| 4. How often did you need help to adjust the brace (e.g., positioning the brace to achieve accurate movement)? | Almost every time (0) | 6 (6.7%) | 8 (9.4%) | 0.473 |
| | Occasionally (1) | 13 (14.6%) | 17 (20.0%) | |
| | Rarely (2) | 70 (78.7%) | 60 (70.6%) | |
| 5. How would you describe using the brace all day long? | Inconvenient (0) | 18 (19.8%) | 5 (5.9%) | 0.024 |
| | Convenient (1) | 42 (46.2%) | 45 (52.9%) | |
| | Very convenient (2) | 31 (34.1%) | 35 (41.2%) | |
| 6. How would you rate your confidence in performing tasks that require walking with the brace? | Less confidence (0) | 2 (2.2%) | 1 (1.2%) | 0.047 |
| | No difference (1) | 16 (17.6%) | 5 (5.9%) | |
| | More confident (2) | 73 (80.2%) | 79 (92.9%) | |
| 7. Do you find the use of the brace safe? | No (0) | 1 (1.1%) | | 0.332 |
| | Yes (2) | 90 (98.9%) | 85 (100.0%) | |
| 8. Do you feel greater confidence in walking on inclines and/or uneven ground while using the brace? | No (0) | 20 (22.0%) | 8 (9.4%) | 0.023 |
| | Yes (2) | 71 (78.0%) | 77 (90.6%) | |

| | | | | |
|---|---|---|---|---|
| 9. Do you feel comfortable wearing the brace in social situations? | No (0) | 18 (19.8%) | 7 (8.2%) | 0.028 |
| | Yes (2) | 73 (80.2%) | 78 (91.8%) | |
| 10. Have you increased your physical activities since using the brace? | No (0) | 18 (19.8%) | 8 (9.4%) | 0.053 |
| | Yes (2) | 73 (80.2%) | 77 (90.6%) | |
| 11. Is the brace something you would use everyday, all day? | No (0) | 25 (27.5%) | 6 (7.1%) | 0.0004 |
| | Yes (2) | 66 (72.5%) | 79 (92.9%) | |
| 12. Would you recommend a person with your condition to use the brace? | No (0) | 7 (7.7%) | 1 (1.2%) | 0.038 |
| | Yes (2) | 84 (92.3%) | 84 (98.8%) | |
| | | 19.1±4.4 | 21.9±2.4 | <0.001 |
| Total score at week 12 | | 19.1±4.0 | 21.8±2.9 | <0.001 |
| Total score at week 30 | | 23 (25.3%) | 3 (3.5%) | <0.0001 |

Supplemental Table 6: Summary of Adverse Events and Falls.

| Serious Adverse Events | | All | Control | Treatment |
|---|---|---|---|---|
| Related to device/procedure | | 0 | 0 (0%) | 0 (0%) |
| Unrelated to device/procedure | | 20 | 6 (30%) | 14 (70%) |
| Adverse Events | | | | |
| All Adverse Events | | 366 | 147 (40%) | 219 (60%) |
| Related to device/procedure – *of total* | | 180 | 50 (28%) | 130 (72%) |
| Severity | Mild | 167 | 48/50 (96%) | 119/130 (92%) |
| | Moderate | 13 | 2/50 (4%) | 11/130 (8%) |
| | Severe | 0 | 0 (0%) | 0 (0%) |
| | Anticipated | 175 | 49/50 (98%) | 126/130 (97%) |
| Falls | | | | |
| Number of falls | | 77 | 43 (56%) | 34 (44%) |
| Number of fallers | | 57 | 29 (51%) | 28 (49%) |

# A study of lower-limb mechanics during stair-climbing

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# A Study of Lower-Limb Mechanics during Stair-Climbing*

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ABSTRACT: **The motions, forces, and moments at the major joints of the lower limbs of ten men ascending and descending stairs were analyzed using an optoelectronic system, a force-plate, and electromyography. The mean values for the maximum sagittal-plane motions of the hip, knee, and ankle were 42, 88, and 27 degrees, respectively. The mean maximum net flexion-extension moments were: at the hip, 123.9 newton-meters going up and 112.5 newton-meters going down stairs; at the knee, 57.1 newton-meters going up and 146.6 newton-meters going down stairs; and at the ankle, 137.2 newton-meters going up and 107.5 newton-meters going down stairs. When going up and down stairs large moments are present about weight-bearing joints, but descending movements produce the largest moments. The magnitudes of these moments are considerably higher than those produced during level walking.**

CLINICAL RELEVANCE: **The findings in this study indicate that the forces generated and the functional requirements during stair-climbing should be considered when establishing design criteria for prosthetic devices for weight-bearing joints and when advising patients about their activities.**

Going up and down stairs is a common activity of daily living. From a mechanical viewpoint, it is quite different from level walking. The differences are reflected by changes in the ranges of motion of the different joints during gait, and changes in the phasic muscle activities and in the maximum joint forces and moments. An understanding of the mechanics of stair-climbing is an important step toward greater knowledge of the function of the lower extremities and the pathogenesis of lower-extremity disorders. This information is also needed to improve patient management and to develop criteria for the design of safe joint replacements for the lower extremity.

Kinematic studies have shown that a larger range of knee motion is required during stair-climbing than during level walking[5,7,11]. Using electrogoniometers, Laubenthal et al. observed that about 83 degrees of knee flexion is required to go up and down stairs. Hoffman et al. reported a similar range of sagittal knee motion during stair-climbing in a group of fifty subjects and found that approximately 12 degrees' more knee flexion is required during stair-climbing than during level walking.

Observations of phasic muscle activity[1,6,12,14] have indicated that there are major differences in the activities of the muscles during stair-climbing as opposed to level walking. These differences in activity are mainly in the muscles responsible for vertical movement of the body. Climbing up stairs, the differences are reflected by changes in the contractions of the soleus, quadriceps femoris, hamstrings, and gluteus maximus during the support phase; going down stairs, the differences are reflected by changes in the contractions of the soleus and quadriceps femoris muscles[6,14]. The duration of the activity of the flexor muscles of the knee has been observed to be small compared with the activity of the extensor muscles of the knee, both ascending and descending stairs[12]. Furthermore, the knee extensor muscles are required to generate larger forces during stair-climbing than during level walking. Morrison and Paul confirmed this observation using data derived by means of electromyography, a force-plate, and high-speed moving pictures of three subjects ascending and descending stairs. The information obtained was used to calculate maximum joint forces at the knee, which were found to be 12 to 25 per cent higher than those during level walking. Using an analytical model, Townsend and

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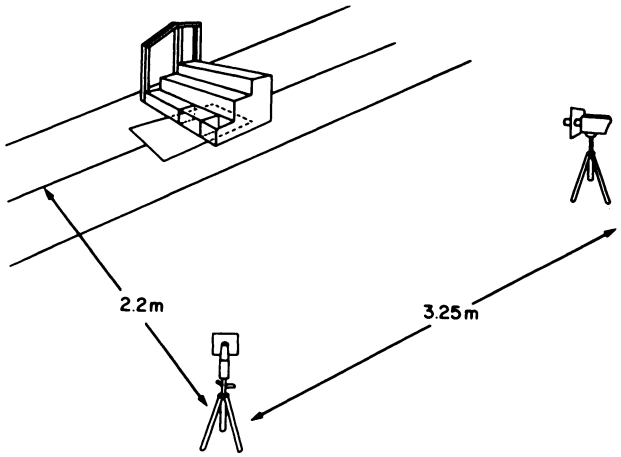


FIG. 1

Camera positions in relation to the staircase and walkway. Note the force-plate and the segment of the first step cut out with a free section resting on the force-plate so that foot-floor and foot-first step reaction forces could be measured.

Tsai[13] observed that a wide range of limb configurations is mechanically feasible during the ascent and descent of stairs. Thus, there is a potential for significant variations in the way different individuals climb stairs.

None of the currently available studies has provided a comprehensive set of data on lower-limb mechanics in normal subjects during stair-climbing. Either the subject populations were small or only a limited number of parameters were studied. The purpose of this study was to analyze the mechanics of the lower limb in ten normal subjects going up and down stairs so that common patterns of motion, forces, and phasic muscle activity could be identified and separated from individual variations.

## Materials and Methods

The study was performed on ten men with a mean age of twenty-eight years (range, twenty to thirty-four years). Their weights ranged from fifty-nine to eighty-three kilograms, with a mean of seventy-one kilograms, and their heights ranged from 165 to 193 centimeters with a mean of 179 centimeters. None of the subjects had had previous diseases or injuries of the locomotor system, and no abnormalities were found by examination.

A homogeneous group of test subjects was selected to reduce differences in measurements due to age or body type, since correlations of this type were not among the objectives of the investigation. The subject population was probably more vigorous than an older or disabled group and therefore had joint loads that were larger than those occurring in patients who are likely to have joint replacements.

The instrumentation included a two-camera optoelectronic digitizer (Selspot), light-emitting diodes, a multicomponent force-plate (Kistler), a chart recorder with electromyographic signal conditioning, a minicomputer (PDP 11/40), and a staircase.

The acquisition and processing of the optical and ground-reaction force data were computerized. Eight channels of analog signals from the force-plate were digitized at a rate of 200 samples per second. Simultaneously, the digital signals from each camera were acquired at a frame rate of seventy-five samples per second. Each camera provided two coordinates in the camera reference frame. The three-dimensional positions of the light-emitting diodes were located from the two sets of coordinates using a modified photogrammetric method[2]. A calibration grid containing twenty-nine calibration points was used to provide a reference system for scaling, correction for distortion, and measurement of position. The photogrammetric technique was found previously to be well suited for use with optoelectronic data-acquisition equipment[2]. Using this technique, the system was found to have a resolution of one part in 500.

The two cameras of the optoelectronic digitizer were located on one side of the stairs and were placed symmetrically relative to the force-plate. So placed, they were 2.20 meters from the center line passing along the walkway through the center of the force-plate, and were separated from one another by a distance of 3.25 meters (Fig. 1). This placement was chosen to give three-dimensional views with an adequate viewing range (2.5 meters) as well as to maintain a minimum camera-to-subject distance.

The kinematic parameters for the three major joints (hip, knee, and ankle) were calculated from the three-dimensional positions of six points on each lower

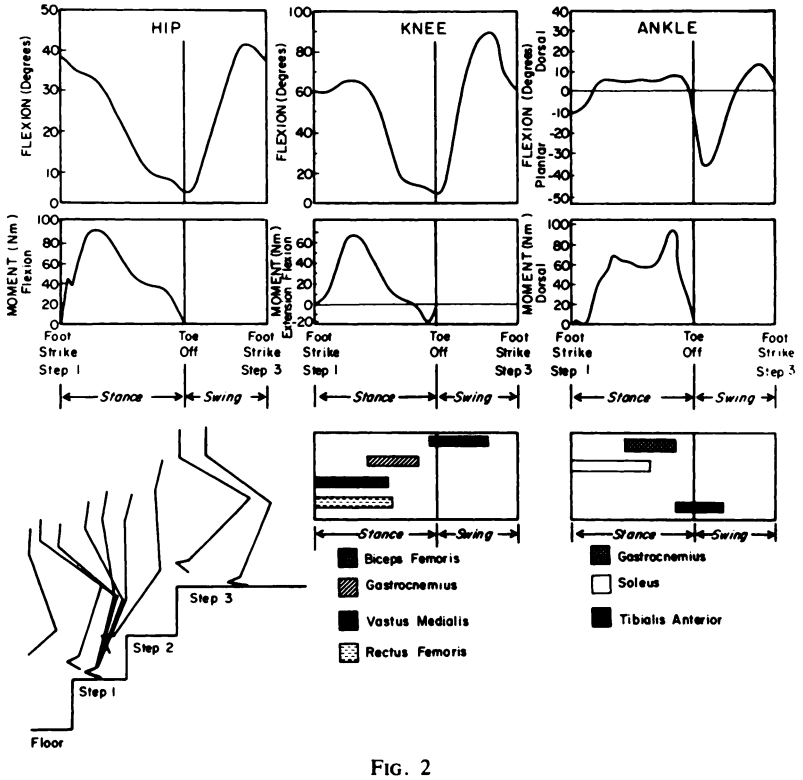


FIG. 2

Sagittal-plane flexion-extension movements of the hip and knee and plantar flexion-dorsiflexion movements of the ankle; moments about these joints; and phasic activities of the knee and ankle muscles in one limb of a subject ascending from Step 1 to Step 3. (The hip muscles were not studied.)

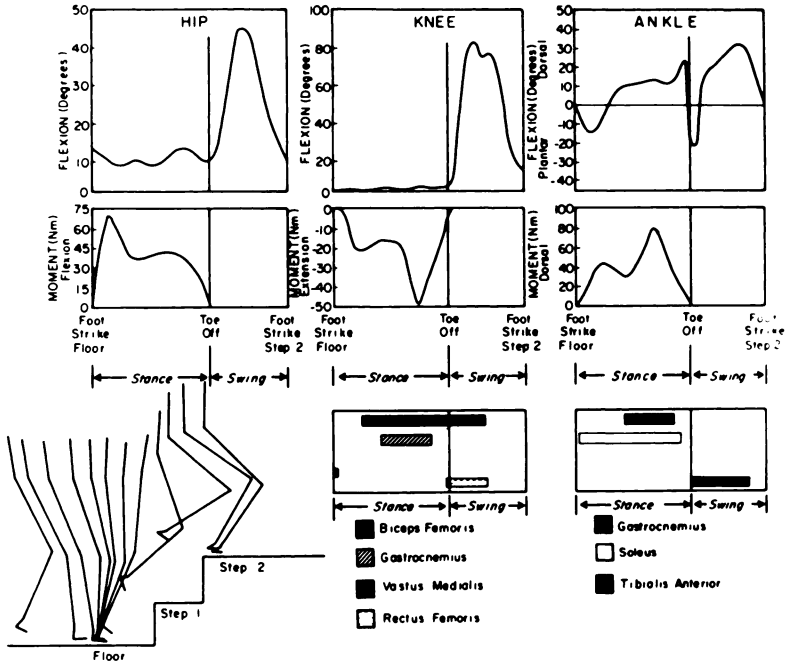


FIG. 3

Sagittal-plane flexion-extension movements of the hip and knee and plantar flexion-dorsiflexion movements of the ankle; moments about these joints; and phasic activities of the knee and ankle muscles in a subject ascending from the floor to Step 2. (The hip muscles were not studied.)

extremity. The points were located by placing light-emitting diodes at the following locations: in the region of the anterior superior iliac spine, over the greater trochanter, over the center of the lateral joint line at the knee, at the lateral malleolus, over the lateral aspect of the calcaneus, and at the base of the fifth metatarsal. Angular joint motions at the hip, knee, and ankle were determined by calculating the angles between vectors defined by the three-dimensional coordinates of the light-emitting diodes located on adjacent limb segments.

The foot-ground reaction force obtained from the force-plate and the instantaneous positions of the hip, knee, and ankle joints were used to compute the net external moment about each joint center throughout stance phase. The moment was calculated by taking the cross product of a vector defining the position of the joint center and of the vector defining the foot-ground reaction force. (Taking the vector cross product is an operation performed on two vectors that yields a third vector perpendicular to the plane defined by the first two vectors. The magnitude of the third vector is equal to the product of the magnitude of the first two vectors and of the sine of the angle between them.) The net vectors of the joint reaction moments were then resolved into component vectors that were aligned along the axes of flexion-extension, abduction-adduction, and internal-external rotation.

The test staircase was composed of three steps, each 25.5 centimeters deep and fifty-eight centimeters wide, with a step height of twenty-one centimeters (standard dimension for an outside staircase). A handrail was placed on the left side. The slope of the staircase was 38 degrees. Outdoor-staircase dimensions were selected because they specify a greater step height and slope than do inside-staircase dimensions, and it was assumed that on these stairs higher physiological demands would be produced. A section of the first step of the staircase was cut out so that this section would rest on the force-plate and permit direct measurement of the foot-stair reaction forces (Fig. 1). It was also possible to measure foot-floor reaction forces directly in front of the first step, using the same force-plate.

Prior to each observation, the subject was instrumented with the diodes already described. The positions of the joint centers of the hip, knee, and ankle in the frontal plane were estimated relative to the diodes placed over the greater trochanter, the lateral joint line of the knee, and the lateral malleolus. The hip-joint center was estimated to be 1.5 to two centimeters distal to the mid-point of a line from the anterior superior iliac spine to the pubic symphysis. The knee-joint center was located in the frontal plane by identifying the mid-point of a line between the peripheral margins of the medial and lateral tibial plateaus at the level of the joint surfaces. The ankle-joint center was estimated to be at the mid-point of a line from the tip of the medial malleolus to the tip of the lateral malleolus.

Bipolar surface electrodes were placed over the rectus femoris, the vastus medialis, the biceps femoris, the medial head of the gastrocnemius, the lateral head of the soleus, and the tibialis anterior muscles. The amplifiers were adjusted following test contractions of each muscle.

Measurements were made while the subjects were ascending and descending the staircase, and observations were recorded while the subjects did and did not use the handrail during the following gait sequences: (1) as the limb moved up from foot-strike on the first step (Step 1) to foot-strike on the third step (Step 3); (2) as the limb moved up from foot-strike on the floor to foot-strike on the second step (Step 2); (3) as the limb moved down from toe-off from Step 3 to toe-off from Step 1; and (4) as the limb moved down from toe-off from Step 2 to toe-off from the floor.

The moments were calculated during the support phase on the first step and on the floor because in these positions the largest inertial contributions were expected.

## Results

The data on limb function were separated into those for ascending and those for descending movements, and into those for movements of the limb going up and down from step to step and going from floor to step and from step to floor. Movements and moments in the sagittal plane were described separately from those in the frontal and horizontal planes since movement in the sagittal plane is the primary movement. The sagittal-plane projection of a stick-figure representation of one limb, along with the flexion-extension motions, the moments tending to produce flexion-extension, and the patterns of phasic muscle activity at the knee and ankle of each subject were recorded. Typical ascending (Step 1 to Step 3 and floor to Step 2) and descending (Step 3 to Step 1 and Step 2 to floor to Step 2) patterns were identified (Figs. 2 through 5).

### *Sagittal-Plane Movements and Moments*

#### *Ascending — Step 1 to Step 3*

The movements of a single limb ascending from Step 1 to Step 3 are illustrated in Figure 2. When the foot strikes Step 1, the hip and knee joints are flexed and the ankle joint is plantar flexed. As the limb moves from foot-strike to mid-stance, the hip and knee joints extend and the ankle joint dorsiflexes slightly. While the hip and knee joints are extending from the flexed positions that were present at foot-strike, there is an external moment at both joints tend-

ing to produce flexion. The knee extensors (vastus medialis and rectus femoris) are active from the time of foot-strike through mid-stance and balance the flexion moment at the knee. Thus, the external flexion moment at the knee is in a direction opposite to the extension movement of the knee, and the extensor muscles are acting both to balance the external flexion moment and to extend the knee. At the ankle joint both the motion and the moment are in the direction of dorsiflexion and the plantar flexors act to balance the dorsiflexion moment. The soleus muscle is active from foot-strike to mid-stance, while the gastrocnemius is active from mid-stance to just before toe-off.

plantar flexion. No muscle activity was observed between mid-swing and foot-strike during ascent from Step 1 to Step 3.

*Ascending — Floor to Step 2*

The movements of a single limb ascending from the floor to Step 2 are shown in Figure 3. These differ from the movements when ascending from Step 1 to Step 3. At the outset, as the foot strikes the floor prior to lifting of the opposite limb up to Step 1, the hip and knee are near full extension and the ankle is plantar flexed. Then, as the limb moves from mid-stance to toe-off, the hip and knee remain

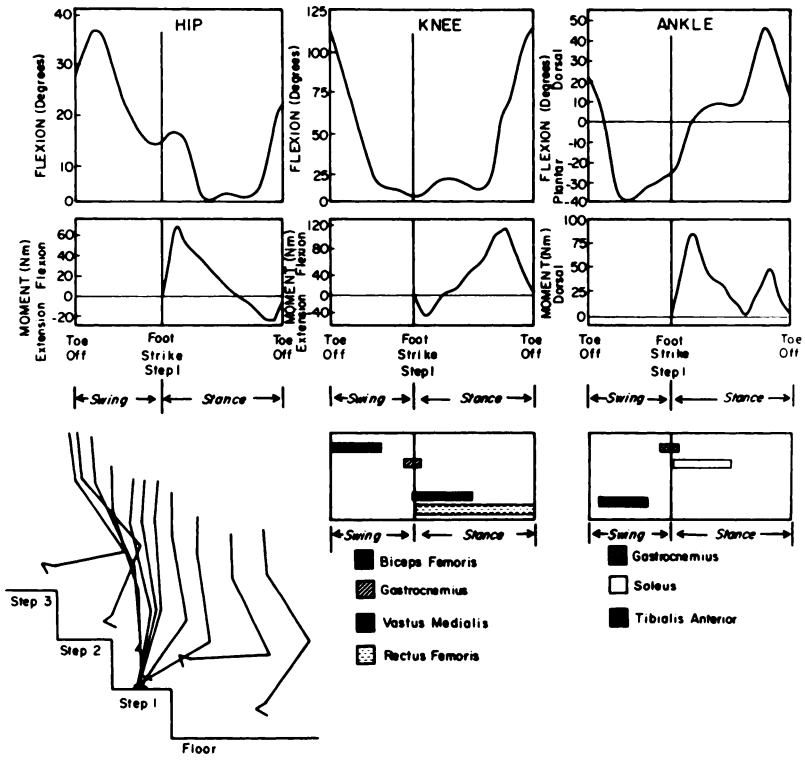


FIG. 4

Sagittal-plane flexion-extension movements of the hip and knee and plantar flexion-dorsiflexion movements of the ankle; moments about these joints; and phasic activities of the knee and ankle muscles in one limb of a subject descending from Step 3 to Step 1. (The hip muscles were not studied.)

As the limb moves from mid-stance toward toe-off, the hip and knee continue to extend and the ankle plantar flexes during toe-off. At the same time the moment at the hip joint decreases but continues to be in the direction of flexion, and the external moment at the knee changes to extension, the same direction as the movement. The biceps femoris becomes active just before toe-off and remains active through mid-swing until the knee attains maximum flexion. The dorsiflexion moment at the ankle joint reaches a maximum just before toe-off. The tibialis anterior becomes active just before toe-off and remains active until mid-swing phase. From mid-swing to foot-strike on Step 3, the hip joint and knee joint move from a position of maximum flexion toward extension, while the ankle joint moves from a position of maximum dorsiflexion toward nearly fully extended and most of the upward movement results from dorsiflexion of the ankle. The external moment at the hip tends to produce hip flexion throughout the entire stance phase. The external moment at the knee tends to extend the joint, but the knee flexors (biceps femoris and gastrocnemius) are active starting after heel-strike and continuing through all or most of the rest of stance phase. The moment at the ankle, which tends to dorsiflex the joint, reaches a maximum before toe-off, but the soleus remains active from foot-strike until just prior to toe-off, when it ceases to be active. During swing phase the hip and knee reach a position of maximum flexion and then begin to move toward extension shortly before foot-strike. The ankle changes abruptly from dorsiflexion before toe-off to plantar flexion right after toe-off. It then dorsiflexes

TABLE I

MEAN OF THE MAXIMUM VALUES OF SAGITTAL-PLANE MOTION (FLEXION)*
(IN DEGREES)

| | Stance | | | | Swing | | | |
|---|---|---|---|---|---|---|---|---|
| | Up | | Down | | Up | | Down | |
| | Step 1 to Step 3 | Floor to Step 2 | Step 3 to Step 1 | Step 2 to Floor | Step 1 to Step 3 | Floor to Step 2 | Step 3 to Step 1 | Step 2 to Floor |
| Hip | 33.8 (6.9) | 7.7 (4.6) | 13.4 (7.0) | 13.2 (6.9) | 40.8 (8.7) | 41.9 (9.9) | 23.0 (10.5) | 28.2 (12.9) |
| Knee | 52.5 (5.2) | 20.55 (6.8) | 68.9 (13.3) | 28.9 (16.0) | 73.4 (12.4) | 83.3 (5.2) | 81.6 (11.3) | 87.9 (4.4) |
| Ankle† | 13.6 (8.6) | 10.0 (7.6) | 24.7 (8.9) | 27.0 (11.4) | −25.3 (11.5) | −25.1 (10.0) | −25.6 (5.3) | −23.2 (4.0) |

* Standard deviation is in parentheses.

† At the ankle joint a positive value indicates dorsiflexion and a negative value indicates plantar flexion.

until mid-swing and finally plantar flexes to neutral prior to foot-strike on Step 2. The biceps femoris and rectus femoris are active during swing from toe-off through mid-swing, while the tibialis anterior is active during the first 80 per cent of swing phase.

*Descending — Step 3 to Step 1*

The movements of a single limb descending from Step 3 to Step 1 are illustrated in Figure 4. At toe-off from Step 3, the hip and knee are flexed and the ankle is dorsiflexed to a maximum or nearly so. During swing phase, hip and knee flexion decreases and the ankle moves into plantar flexion. The biceps femoris is the only active knee muscle at the start of swing and remains active through mid-swing. The tibialis anterior is active during mid-swing and the gastrocnemius becomes active just prior to foot-strike on Step 1. When the foot strikes Step 1, the hip joint is only slightly flexed, the knee is near full extension, and the ankle is plantar flexed. Then, as the limb moves toward mid-stance on Step 1, the hip joint extends and a simultaneous external hip-flexion moment is present, which must be offset by contraction of the extensor muscles of the hip. (Recordings of the hip muscles were not made in this study.) At the knee there is an external extension moment just after foot-strike, which persists while the knee is flexing slightly. The knee extensors are active from foot-strike throughout the major portion of stance phase on Step 1. The dorsiflexion moment at the ankle reaches a

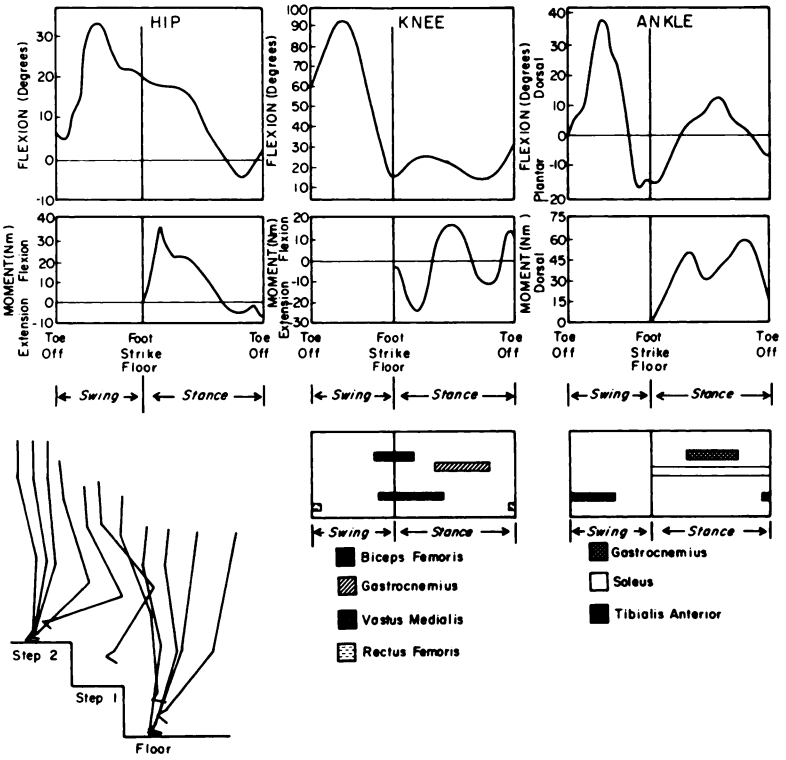


FIG. 5

Sagittal-plane flexion-extension movements of the hip and knee and plantar flexion-dorsiflexion movements of the ankle; moments about these joints; and phasic activities of the knee and ankle muscles in one limb of a subject descending from Step 2 to the floor. (The hip muscles were not studied.)

TABLE II

MEAN OF THE MAXIMUM NET JOINT-REACTION MOMENTS (FLEXION-EXTENSION)*
(IN NEWTON-METERS)

| | Up | | | | Down | | | |
|---|---|---|---|---|---|---|---|---|
| | Step 1 to Step 3 | | Floor to Step 2 | | Step 3 to Step 1 | | Step 2 to Floor | |
| | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail |
| Hip | 123.9 (33.6) | 107.4 (27.0) | 54.1 (22.2) | 51.0 (19.4) | 112.5 (43.1) | 99.2 (26.7) | 66.5 (22.0) | 75.0 (20.8) |
| Knee | 54.2 (17.2) | 52.4 (14.1) | −57.1 (15.1) | −44.7 (20.0) | 146.6 (48.0) | 139.1 (45.0) | −42.9 (10.0) | −59.6 (26.0) |
| Ankle | 101.8 (38.0) | 108.6 (44.0) | 137.2 (34.0) | 108.1 (40.0) | 107.5 (32.0) | 104.3 (18.0) | 75.5 (12.0) | 88.5 (29.0) |

* A positive value indicates flexion at the hip and knee and dorsiflexion at the ankle. Negative values indicate extension at the hip and knee and plantar flexion at the ankle. Standard deviation is in parentheses.

maximum while the ankle is moving toward dorsiflexion. Thus, the plantar flexors, which are active until mid-stance, balance the dorsiflexion external moment that is present while the ankle is moving from plantar flexion to dorsiflexion. As the limb moves from mid-stance to toe-off, the hip remains near full extension and the moment at the hip changes toward extension. Prior to toe-off, the knee begins to flex as the external moment tending to flex the knee reaches a maximum and decreases prior to toe-off. Therefore, prior to toe-off, knee flexion is under the control of the knee extensors (rectus femoris) as they act to balance a large external flexion moment at the knee that develops just before toe-off. Maximum dorsiflexion at the ankle occurs just prior to toe-off and is associated with a rise in dorsiflexion moment.

*Descending — Step 2 to Floor*

During descent from Step 2 to the floor, the limb leaves Step 2 and during swing phase moves toward the floor, while the hip and knee flex and the ankle moves into plantar flexion (Fig. 5). During swing phase, the rectus femoris and tibialis anterior are active at toe-off and during the first part of swing, while the vastus medialis and gastrocnemius become active near the end of this phase. At foot-strike on the floor, the hip is still moderately flexed, the knee is nearly fully extended, and the ankle is plantar flexed. Then, as the limb on the floor moves toward mid-stance, the hip extends, the knee flexes slightly, and the ankle dorsiflexes. At mid-stance the external moment at the hip tends to flex the joint, and the external knee moment changes from extension to flexion. After mid-stance the hip and knee moments change back to extension. Both knee flexors (biceps femoris) and extensors (vastus medialis) are active at foot-strike, and the vastus medialis remains active until mid-stance. The gastrocnemius becomes active during mid-stance as the ankle joint moves from dorsiflexion at mid-stance to plantar flexion at toe-off. Dorsiflexion of the ankle increases during stance phase to reach a maximum during mid-stance and then changes to plantar flexion just prior to toe-off. The soleus is active throughout stance phase.

*Maximum Ranges of Flexion-Extension Motion and Flexion-Extension Moments*

The maximum ranges of movement and the maximum external moments at the hip, knee, and ankle while ascending stairs were compared with those while descending stairs (Tables I through IV).

*Motions*

At the hip, the most flexion occurred during swing phase while ascending (41.9 degrees), and at the knee the

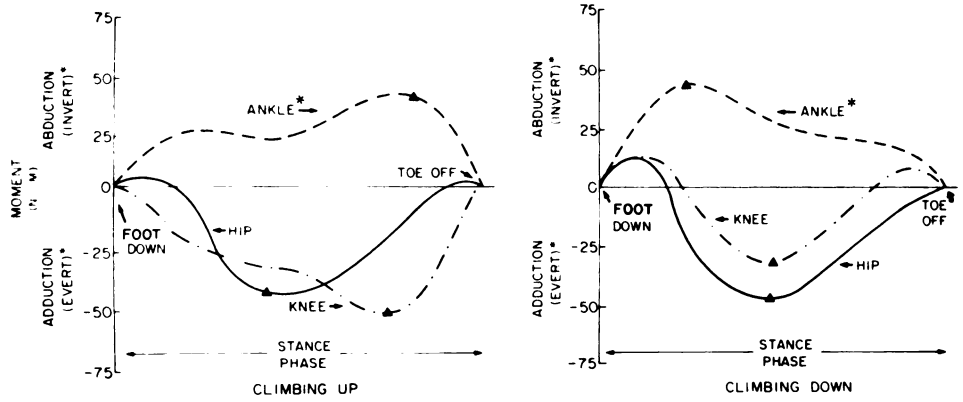


FIG. 6

Typical patterns of abduction-adduction moments at the hip and knee and inversion-eversion moments (*) at the ankle joints. The patterns going up and down from step to step and between floor and step were similar.

TABLE III

MEAN OF MAXIMUM EXTERNAL MOMENTS (ABDUCTION-ADDUCTION)*
(IN NEWTON-METERS)

| | Up | | | | Down | | | |
|---|---|---|---|---|---|---|---|---|
| | Step 1 to Step 3 | | Floor to Step 2 | | Step 3 to Step 1 | | Step 2 to Floor | |
| | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail |
| Hip | −37.0 (18.7) | −36.5 (20.3) | −60.7 (28.3) | −58.4 (28.5) | −40.1 (23.3) | −33.4 (12.1) | −86.0 (31.5) | −63.9 (23.5) |
| Knee | −33.0 (17.0) | −28.2 (9.0) | −32.5 (21.0) | −39.4 (18.0) | −23.6 (16.0) | −27.2 (11.0) | −59.5 (37.0) | −38.5 (18.0) |
| Ankle | 42.8 (33.0) | 39.2 (9.0) | 22.6 (9.0) | 19.4 (13.0) | 44.5 (14.0) | 47.5 (17.5) | 31.3 (28.0) | 17.8 (8.0) |

* A positive value indicates abduction at the hip and knee and inversion at the ankle. A negative value indicates adduction at the hip and knee and eversion at the ankle. Standard deviation is in parentheses.

most flexion occurred during swing phase while descending the stairs (87.9 degrees) (Table I). However, there was no significant difference between the amounts of swing-phase hip and knee flexion while ascending and descending stairs.

On the other hand, at the knee there was a significant difference between the amounts of stance-phase flexion during floor-to-step and during step-to-step ascending and descending movements. Thus, during the stance phase while descending, the knee flexed more than twice as much going from step to step (68.9 degrees) as it did going from step to floor (28.9 degrees).

At the ankle joint during swing phase the motion patterns while ascending and descending stairs were similar. During stance phase, on the other hand, dorsiflexion was less while ascending from floor to step (10 degrees) than while descending from step to step (24.7 degrees). The most dorsiflexion (27 degrees) was observed during mid-stance phase while descending from step to step.

*Moments*

At the hip, the maximum flexion moment (123.9 newton-meters) during ascent was observed while the limb was ascending from Step 1 to Step 3 (Table II), and this moment was reduced by a factor of slightly more than two while the limb was ascending from the floor to Step 2. Step-to-step descent produced a moment at the hip approximately twice that produced by descending from Step 2 to the floor (112.5 compared with 66.5 newton-meters).

At the knee, the maximum flexion moment (146.6 newton-meters) occurred during step-to-step descent. This moment was nearly three times that produced at the knee joint by other activities. Thus, the most stressful activity for the knee joint appears to be step-to-step descent.

At the ankle, both going up and going down stairs tended to produce dorsiflexion-plantar flexion moments that were not significantly different. The activity that produced the largest moment (137.2 newton-meters) at the ankle during stance phase was ascending from floor to step. Using the handrail in the usual fashion had no statistically significant influence on the magnitude of any of the flexion-extension moments observed in this investigation.

*Frontal-Plane and Horizontal-Plane Moments*

The abduction-adduction and internal-external rotation moments at the hip and knee and the inversion-eversion and internal-external rotation moments at the ankle were analyzed in a similar manner to that described for the flexion-extension moments of these joints. The typical patterns for going up and down from step to step and between floor and step were similar (Figs. 6 and 7).

*Abduction-Adduction and Inversion-Eversion Moments*

At the hip, the abduction-adduction moment tended to adduct the joint throughout the entire stance phase. The maximum adduction moment of 86.0 newton-meters was observed during descent from Step 2 to the floor (Table III). The adduction moments observed while descending from Step 3 to Step 1 were about half as large as the moments recorded while descending from Step 2 to the floor. At the knee, the maximum adduction moment occurred when descending from Step 2 to the floor (59.5 newton-meters). At the ankle there was an inverting moment throughout the entire stance phase which was maximum (47.5 newton-meters) during descent from Step 3 to Step 1.

*Internal-External Moments*

The internal-external moments were quite low (less than twenty newton-meters) at all joints during every activity studied (Table IV). The patterns of the internal-external rotation moments were also quite variable. The most common finding (Fig. 7) was an internal rotation moment at the hip and ankle and an external rotation moment at the knee during the stance phase of the activities studied.

## Discussion

The net moments at the hip, knee, and ankle were found to be of sufficient magnitude to require that they be considered in any analysis of the mechanics of the lower limb during stair-climbing, and in the design of implants for joint reconstruction. It appears from our results that the

TABLE IV

MEAN OF MAXIMUM EXTERNAL MOMENTS (INTERNAL-EXTERNAL ROTATION)*
(IN NEWTON-METERS)

| | Up | | | | Down | | | |
|---|---|---|---|---|---|---|---|---|
| | Step 1 to Step 3 | | Floor to Step 2 | | Step 3 to Step 1 | | Step 2 to Floor | |
| | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail | No Handrail | Handrail |
| Hip | 14.7 (5.5) | 13.4 (6.1) | 11.7 (3.8) | 10.3 (3.0) | 15.6 (6.1) | 12.0 (3.2) | 18.0 (7.7) | 15.1 (5.4) |
| Knee | −6.8 (3.0) | −6.4 (3.0) | −7.8 (3.7) | −6.3 (2.0) | −15.1 (9.1) | −15.5 (5.3) | −15.0 (9.0) | −14.3 (8.0) |
| Ankle | 9.1 (6.0) | 9.2 (4.3) | 13.2 (4.8) | 11.2 (5.0) | 10.9 (4.0) | 12.0 (5.0) | 19.7 (8.0) | 13.6 (2.0) |

* A positive value indicates internal rotation and a negative value indicates external rotation. Standard deviation is in parentheses.

flexion-extension moments correlate with the activity of the major flexor-extensor muscle groups, since the net external moments must be balanced primarily by muscle forces.

There is often antagonistic and synergistic muscle activity across a joint prohibiting direct calculation of muscle forces without additional assumptions defining some type of optimization criteria. However, the magnitude of the moment can be used as a relative indicator of the magnitude of the muscle forces across a joint.

Similarly, the contact forces in the joints are directly proportional to the net reaction moments about the joints. Thus, an activity that produces a large external moment will probably produce a large contact force in a joint. It is useful to re-examine the results with these relations in mind.

The ankle joint was subjected to relatively large dorsiflexion moments while both ascending and descending stairs, which necessitated comparable muscle forces in the plantar-flexor muscle group. These dorsiflexion moments were similar in magnitude to those observed during level walking[3,8]. However, the inversion moments while descending or ascending from one step to another were larger in magnitude than those observed during level walking.

At the knee, the flexion moments while descending stairs were the largest and necessitated a large force in the knee extensor muscles to offset them. This flexion moment was about three times greater than the flexion moment generated during level walking. If one assumes that the joint force at the knee is proportional to the external moments at this joint, then the magnitude of the knee-joint contact force generated while descending stairs could be more than six times body weight. The large external moment about the knee while descending stairs occurred when the knee was at about 50 degrees of flexion, whereas during level walking the largest moment occurs when the knee is near full extension[3,8]. Thus, on stairs the knee-joint surface probably sustains a resultant contact force that is different in both direction and magnitude from that occurring during level walking. It should be noted that the flexion-extension moment at the knee when the foot struck the floor while descending from Step 2 was about 50 per cent less than the moment when descending from one step to another, because when stepping down to the floor both feet descend to the same level rather than the swing-phase limb going to the next step below. A patient can reduce the joint forces significantly if both limbs are brought down to the same step while descending from one step to the next.

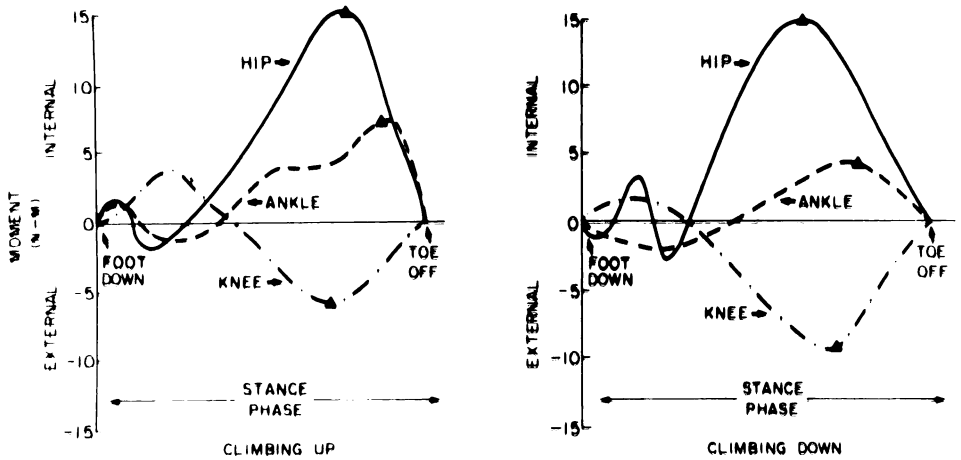


FIG. 7

Typical patterns of internal-external moments at the hip, knee, and ankle joints. The patterns going up and down from step to step and between floor and step were similar.

Many patients descend stairs in this fashion because of the pain associated with the large force generated by placing one foot on every other step as they go down the stairs.

As at the knee joint, the flexion-extension moments at the hip were also found to be larger while descending from one step to another than from one step to the floor. The step-to-step flexion moments were about one and a half times greater than those observed during level walking, whereas the moments while ascending from one step to another were of about the same magnitude as those during level walking. The hip was flexed between 30 and 40 degrees when the largest moments were generated. Thus, the resultant load on the femoral head may have a large force component that is perpendicular to the frontal plane. This component of the load may be an important consideration in the design of the femoral stem of a total hip replacement, since this component could generate tensile stresses on the anterior surface of the femoral stem[4].

## Conclusions

The results of this study show that going up and down stairs results in high joint moments. The highest moment usually occurs while descending stairs. The magnitudes of the flexion-extension moments at the hip and knee are greater during stair-climbing than during level walking[3,8]. The largest increase in moment going up and down stairs compared with level walking occurs at the knee joint. The moments at the ankle going up and down stairs do not show any significant increase over level walking. In the development of prosthetic devices for the lower extremity, functional activities such as stair-climbing should be considered among the design criteria.

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# The "Good" Limb Makes the "Bad" Limb Worse: Experience-Dependent Interhemispheric Disruption of Functional Outcome After Cortical Infarcts in Rats

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Following stroke-like lesions to the sensorimotor cortex in rats, experience with the ipsi-to-lesion (ipsilesional), "nonparetic", forelimb worsens deficits in the contralesional, "paretic", forelimb. We tested whether the maladaptive effects of experience with the nonparetic limb are mediated through callosal connections and the contralesional sensorimotor cortex. Adult male rats with proficiency in skilled reaching with their dominant (for reaching) forelimb received ischemic bilateral sensorimotor cortex lesions, or unilateral lesions, with or without callosal transections. After assessing dominant forelimb function (the paretic forelimb in rats with unilateral lesions), animals were trained with their nonparetic/nondominant forelimb or underwent control procedures for 15 days. Animals were then tested with their paretic/dominant forelimb. In animals with unilateral lesions only, nonparetic forelimb training worsened subsequent performance with the paretic forelimb, as found previously. This effect was not found in animals with both callosal transections and unilateral lesions. After bilateral lesions, training the nondominant limb did not worsen function of the dominant limb compared with controls. Thus, the maladaptive effects of training the nonparetic limb on paretic forelimb function depend upon the contralesional cortex and transcallosal projections. This suggests that this experience-dependent disruption of functional recovery is mediated through interhemispheric connections of the sensorimotor cortex.

*Keywords:* corpus callosum, motor skill, unilateral, nonparetic, recovery

Stroke affects approximately 795,000 Americans annually and accounts for one of every 18 deaths in the United States (Lloyd-Jones et al., 2009). A prevalent problem after stroke is loss of function in the hand and arm contralateral to the side of injury (the "paretic" side). As a result, stroke survivors begin to rely on the ipsilesional side, despite the presence of mild impairment in this side. A now established treatment approach for upper arm impairments is constraint-induced movement therapy (CIMT; Mark, Taub, & Morris, 2006; Taub, Uswatte, Mark, & Morris, 2003), where use of the paretic arm is encouraged through restraint of the nonparetic hand for most waking hours. This is intended to counteract the effects of learned nonuse of the paretic arm, which is thought to result from repeated experience with its incompetence. Data from clinical trials indicate that this therapy can significantly improve upper arm deficits (Wolf et al., 2006; Park, Wolf, Blanton, Winstein, & Nichols-Larsen, 2008). Frequently, however, stroke survivors learn to use their nonparetic side in order to carry out daily tasks (e.g., Dobkin, 2006). Though learning how to compensate with this body side may convey immediate functional benefits, its long-term neural and behavioral consequences are not well understood.

Unilateral sensorimotor cortex (SMC) damage in the caudal forelimb representation area in rats results in sensory and motor impairments in the contralesional forelimb and a compensatory reliance on the ipsilesional forelimb, mimicking some aspects of upper extremity impairment and learned nonuse in human stroke (e.g., Allred & Jones, 2004; Bury & Jones, 2002; Hsu & Jones, 2005; Luke, Allred, & Jones, 2004). We use the terms "nonparetic" and "paretic" to refer to the two limbs in this animal model to be consistent with the clinical terminology used in reference to the weakness and partial loss of motor function found after unilateral cerebral stroke.

Recently, we found that rats trained with their nonparetic forelimb early after unilateral SMC damage have worsened motor function, decreased responsiveness to rehabilitative training of the paretic forelimb and a reduced peri-lesion neuronal activation of FosB/ΔFosB compared to rats without nonparetic forelimb training (Allred & Jones, 2008; Allred, Maldonado, Hsu, & Jones, 2005). This may indicate plasticity-inhibiting effects of the nonparetic forelimb on the remaining cortex of the injured hemisphere, and that learning to compensate with the nonparetic body side can limit neural recovery mechanisms of the paretic limb. However, the mechanisms underlying this effect are entirely unknown.

Following unilateral brain injury, there are abnormalities in interhemispheric activity that are associated with reduced functional outcome. For example, after visual cortex lesions in cats, there is increased activity in contralesional regions. Visual neglect to stimuli presented in the contralesional field can be

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reduced with transient lesions of the contralateral cortex (Rushmore, Valero-Cabre, Lonber, Hilgetag, & Payne, 2006; Ward & Cohen, 2004). Interhemispheric inhibition (as measured using a paired-pulse transcranial magnetic stimulation protocol) from the contralesional to the lesion hemisphere is increased following stroke in humans (Duque et al., 2005; Murase, Duque, Mazzocchio, & Cohen, 2004; see also Perez & Cohen, 2009), and this is correlated with deficits in motor performance (Murase et al., 2004). In functional MRI studies, better functional outcome tends to correspond with more normal lateralized cortical activity during hand movements (reviewed in Cramer, 2008). It seems logical to think that experience with the ipsilesional body side may also further disrupt interhemispheric activity and contribute to worsened recovery.

The present studies were designed to test whether the maladaptive effects of nonparetic forelimb experience are mediated by the contralesional SMC and intercortical connections. If so, then reduction in these intercortical connections, which can be induced with partial transections of the corpus callosum (Bury et al., 2000), should mitigate the maladaptive effect of nonparetic forelimb experience on functional recovery of the paretic forelimb. Furthermore, the effect should not be found in animals with bilateral SMC lesions.

## Method

### Subjects

Forty-four, 6- to 7-month-old adult male Long-Evans rats were housed in pairs on a 12:12 light/dark cycle in standard laboratory cages. Animals were provided with standardized housing supplementation (a 10.5-cm diameter polyvinyl chloride pipe, small wooden objects, and cardboard paper rolls). At the beginning of each experiment, animals were fed a restricted diet of 16–19 g/day/rat standard rat chow so that they were motivated to perform the skilled reaching task. Animal protocols were approved by the University of Texas Institutional Animal Care and Use Committee.

### Experimental Designs

**Experiment 1.** This study was designed to test whether the nonparetic forelimb training effects on paretic forelimb function depend upon callosal projections of the SMC. If so, then the impairing effects of nonparetic forelimb training on skilled motor behavior in the paretic limb should be present in animals with an intact corpus callosum, but absent or reduced if callosal connections of the SMC are partially severed by transection (CCX). Rats were divided into four groups based on postoperative contralesional forelimb reaching deficits: two groups received control procedures (Cont, $n = 7$; CCX_Cont, $n = 10$) and two groups received training of the nonparetic, ipsilesional limb (NonParT, $n = 7$; CCX_NonParT, $n = 9$).

**Experiment 2.** This study was designed to test whether the worsening of paretic limb function by training the other limb depends upon the contralesional cortex. If so, then the effect should not be found in animals with bilateral SMC damage. It was hypothesized that animals with bilateral SMC damage trained with their nondominant (for the task) forelimb (Bilat_ND, $n = 6$) would perform at a similar level with their dominant (for the task) forelimb compared to animals receiving control procedures (Bilat_Cont, $n = 5$). Figure 1 outlines the experimental designs.

### Ischemic SMC Lesions

All animals were given unilateral SMC lesions opposite their dominant-for-reaching forelimb (Experiment 1) or in both hemispheres (Experiment 2) using the endothelin-1 (ET-1) method, which results in localized transient ischemia (Fuxe et al., 1997; Adkins, Voorhies, & Jones, 2004). Animals were anesthetized with i.p. injections of ketamine (100 mg/kg) and xylazine (10 mg/kg) and maintained under a surgical plane of anesthesia throughout the procedure, with ketamine boosters when necessary. A craniectomy was made by connecting four drill holes (A/P: −1.0, +2.0, M/L: 2.0, 4.5) and dura was removed just prior to topical application of 3.0 μl (Experiment 1) or 4.0 μl (Experiment 2) of ET-1 (80 pmol, American Peptide, Inc.). (Animals in Experiment 2 received a greater amount of ET-1 to ensure that the lesions were large enough to adequately test the importance of the nondominant SMC in dominant forelimb recovery.) Animals were then left undisturbed for 10 minutes before suturing. Buprenor-

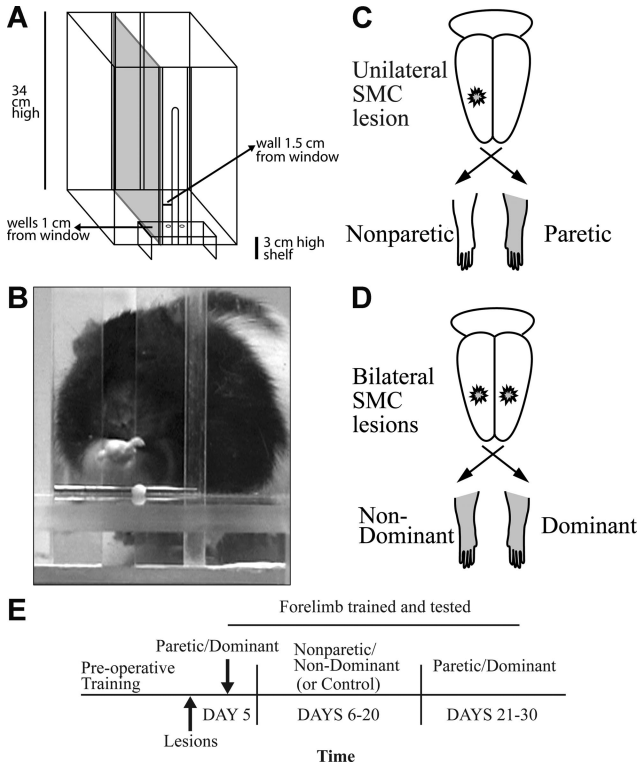


*Figure 1.* Experimental Design. A. Schematic of the reaching chamber. The inner chamber wall and pellet placement are adjusted to train the left versus right limb. This chamber is configured for reaching with the right forelimb. B. A rat aiming and reaching for a banana flavored food pellet. C. Unilateral SMC lesion and corresponding nonparetic (ipsilesional) and paretic (contralesional) forelimbs. Transections of the corpus callosum are not depicted. D. Bilateral SMC lesions and corresponding non-dominant- and dominant-for-reaching forelimbs. E. Time line of experimental procedures. In Experiment 1, transections were given at the same time as ischemic lesions.

phine (.05 mg/kg), an analgesic, was administered subcutaneously postsurgery when the animal began to arise from anesthesia. Behavioral assessment of reaching ability began 5 days after surgeries. For animals with corpus callosum transections (see below), ET-1 was applied to the cortical surface directly following the completion of the transection. Animals in Experiment 2 received bilateral craniectomies, and ET-1 was applied to one cortex and then immediately to the other cortex, with the first side chosen randomly.

## Corpus Callosum Transections

Transections were made using methods that focus callosal lesions in the region of the interhemispheric projections of the SMC (Bury et al., 2000; Bury & Jones, 2002). As a control, midline skull between A/P −2.0 to +1.5 mm relative to Bregma was thinned and removed in all animals in Experiment 1. To prevent mechanical damage, the electrode was not lowered into the brain of Cont or NonParT animals. For those animals receiving a transection (CCX_Cont, CCX_NonParT), the dura and sagittal sinus were pushed to the side and a size 00 ethyl cyanocrylate coated insect pin with an exposed tip was lowered 4.7 mm into the brain at −1.5 mm posterior to Bregma. The side of approach was opposite the SMC lesion. After lowering, 0.7 mV of anodal current was passed through the electrode while it was moved rostrally to Bregma over 9 seconds. The electrode was then raised 0.7 mm and current was again passed while the electrode was moved rostrally 1 mm anterior to Bregma over 6 seconds.

## Single Pellet Retrieval Task

Reach training was carried out as previously described (Allred & Jones, 2008; Hsu & Jones, 2005; Maldonado, Allred, Felthauser, & Jones, 2005), adapted from Whishaw and others (Miklyaeva & Whishaw, 1996; Whishaw, 1992; Whishaw, Pellis, & Gorny, 1992). Briefly, for shaping, animals were placed in a Plexiglas reaching chamber with their cage mate for 10 minutes. Forty-five-mg banana-flavored pellets (Bioserve, Inc.) were dropped into the chamber and placed on a 3-cm high shelf located outside of the reaching chamber (see Figure 1). On each subsequent day animals were placed into the reaching chamber alone for 10 minutes and permitted to reach for pellets on the shelf. Once a limb of preference was established (15 of 20 reach attempts made with same forelimb), this was considered the dominant-for-reaching limb and, the next day the task was configured so that they could only successfully reach pellets with the dominant forelimb. A Plexiglas wall was placed ipsilaterally to the reaching limb and pellets were placed in a shallow well 1 cm from the center window for 30 trials or 10 minutes, whichever came first. To discourage tongue use, a 2-mm diameter rod (a drill bit) was adhered to the platform where it made contact with the reaching chamber. Preoperatively, animals were trained to a proficient level (≥50% success/reach attempt).

On each trial, animals were given up to five reach attempts to obtain a single pellet. Trials concluded when the pellet was knocked from its well or greater than five reach attempts took place (failures), the pellet was retrieved but dropped inside the chamber before consumption (drop), or the pellet was retrieved and taken directly to the mouth (success). Postoperative nondominant (nonparetic in Experiment 1) limb training was for 60 trials/day for 15 days. Postoperative performance was calculated based on % successful retrievals (success + drops/total reach attempts). Reach training focused on the paretic/dominant forelimb was used to assay the initial effects of the lesions and the effects of experience with the other limb (see Figure 1). Preoperatively and during the postoperative paretic/dominant forelimb assessment and training periods, animals received up to 30 trials per day for either 9 days (Experiment 1) or 13 days (Experiment 2).

Reach training focused on the nonparetic/nondominant forelimb was used as an experimental manipulation. Postoperative reach training of the nondominant (nonparetic in Experiment 1) side was for 60 trials/day for 15 days. (The larger number of trials in this phase was intended to ensure its robustness as an experimental manipulation, which may vary with training intensity; Allred & Jones, 2008). Animals in control conditions were placed in a reaching chamber, without an inner wall, and given banana-flavored food pellets on the cage floor at approximately the same rate as trained animals.

## Schallert Cylinder Test

To test forelimb use asymmetries, the Schallert cylinder test (Schallert, Kozlowski, Humm, & Cocke, 1997) was used preoperatively and postoperatively. Animals were placed into a 19 cm diameter Plexiglas cylinder for approximately 2 minutes to encourage upright postural support behaviors. Use of the forelimbs (ipsilateral, contralateral, or bilateral) on the cylinder walls was recorded from slow motion playbacks of videotape. Percent use of the nondominant forelimb was calculated based on noumber of nondominant touches/sum of all touches.

## Histology and Lesion Evaluation

At the conclusion of each experiment, animals were overdosed with sodium pentobarbital (100 mg/kg) and perfused transcardially with 0.1 M phosphate buffer and 4% paraformaldehyde in the same buffer. Brains were removed and sliced coronally with a vibratome into 50 μm thick sections collected in six alternating sets. Sliced brains were stored in cryoprotectant at 4 °C. One set of sections was immediately mounted onto gelatin-coated slides and stained with toluidine blue, a Nissl stain.

The volume of remaining cortex in the SMC region was measured by tracing seven 50 μm (between 2.2 mm anterior to and 0.80 mm posterior to Bregma) coronal Nissl stained sections using Neurolucida (Microbrightfield Inc.) perimeter tracing software at 17× magnification. Moving caudally, the first section containing the head of the caudate was chosen and subsequent sections were 600 μm apart. Volume was obtained by applying the formula: $\Sigma A$*section distance where $\Sigma A$ is the total area summed across all sections (Gundersen et al., 1988).

## Statistical Analyses

All statistical analyses were carried out using SPSS statistical software (SPSS, Inc) with *a priori* planned comparisons. We chose to perform planned comparisons rather than an omnibus analysis of variance (ANOVA) comparing all four groups as literature has shown that a priori designs are a more powerful approach to test

specific planned (prior to the experiment) comparisons (e.g., Kuehne, 1993; Benton, 1989; DuRapau, 1988). The comparisons were designed to test whether reaching performance in the dominant/paretic forelimb testing period was: 1) affected by prior nonparetic limb training after unilateral lesions alone (Cont vs. NonParT) and 2) affected by prior nonparetic limb training after unilateral lesions with callosal transections (CCX_Cont vs. CCX_NonParT), and 3) affected by prior nondominant limb training after bilateral lesions (Bilat_Cont vs. Bilat_ND). This analysis plan tests the effects of the primary behavioral manipulation (training the nondominant/nonparetic limb) by only comparing groups with similar injuries to one another, which avoids potential complications in the interpretations related to differences in injury extent.

Performance during the nonparetic forelimb training period was also compared between NonParT and CCX_NonParT. Behavioral analyses were performed with repeated-measures ANOVAs and student's *t* tests. Volume analyses were performed with one-way ANOVAs or paired sample *t* tests. All data are expressed as means ± SEM. Effects were considered significant at $p < .05$. Three rats in the CCX_Cont group (Experiment 1) had particularly large damage resulting from the callosal transection procedure. Excluding these animals did not change statistical outcome on behavioral measures and therefore they remained in the study, however they are considered separately, as described below.

## Results

### Corpus Callosum Transections Mitigate the Negative Impact of Nonparetic Forelimb Experience

After unilateral SMC lesions (Experiment 1), there was no difference in acquisition of the skilled reaching task with the nonparetic forelimb in rats with or without callosal transections, $F(1, 15) < 1.0$, $p > .05$, Figure 2A. Consistent with previous findings, this nonparetic forelimb training led rats with SMC lesions to perform significantly worse than Cont rats when later trained with their paretic forelimb, $F(1, 12) = 5.82$, $p < .05$, Figure 2B. A significant group by day interaction effect was also found, $F(8, 96) = 2.13$, $p < .05$ and subsequent post hoc analyses for day revealed significant differences on Days 4 through 8 ($F$'s $> 5.0$, $p$'s $< .05$) and Day 9 ($F = 4.74$, $p = .05$). However, in rats with both unilateral lesions and transections of the corpus callosum, there was no significant effect of the nonparetic/ipsilesional forelimb training on the paretic forelimb, $F(1, 17) = 1.44$, $p > .05$; CCX_Cont versus CCX_NonParT, though there was a tendency for CCX_Cont rats to perform better than CCX_NonParT rats, Figure 2C. These results cannot be explained by differences in reaching activity of the paretic forelimb. There was no significant group or group by day interaction for the number of reach attempts made with the paretic limb over the days of training this limb.

Transections did not result in significant deficits in reaching behavior in the paretic forelimb. As measured on Day 5 postlesion, rats without transections had a 57.1 ± 7.83% reduction from preoperative performance levels whereas rats with both unilateral lesions and callosal transections had a 53.3 ± 7.21% drop from preoperative baseline (Figures 2B-C). CCX_NonParT rats tended to perform better than NonParT rats with their paretic forelimb, however this effect failed to reach significance, $F(1, 14) = 1.25$, $p > .05$.

### Lack of Nondominant Forelimb Training Effects After Bilateral SMC Lesions

Nondominant forelimb training after bilateral lesions did not worsen subsequent performance with the dominant forelimb compared to control rats, $F(1, 9) < 1.0$, $p > .05$ (Figure 3). Animals with bilateral lesions also did not differ in the number of reach

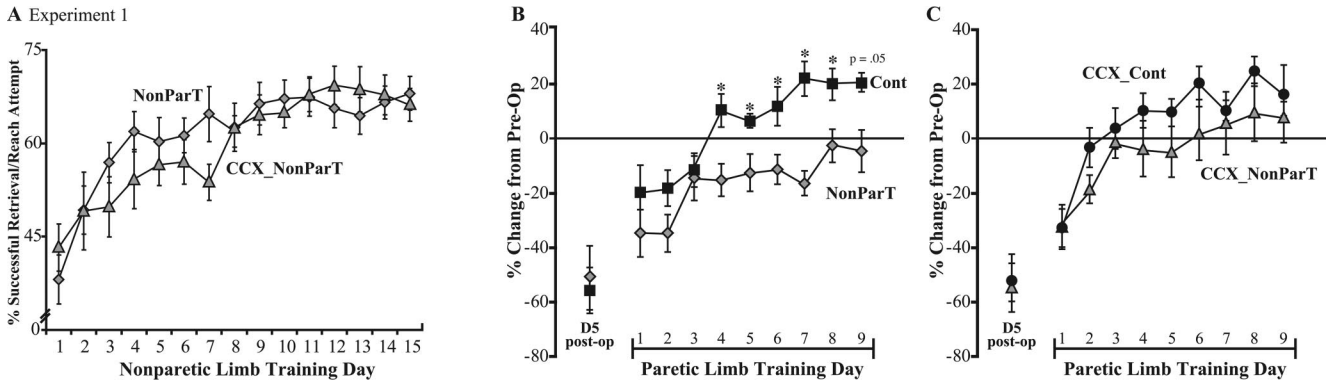


*Figure 2.* Nonparetic forelimb training worsens performance of the paretic forelimb in rats without corpus callosum transections. A. Performance during the period of training of the nonparetic, ipsilesional, limb (NonParT) after unilateral SMC lesions in rats with or without CCX. There was no significant difference in acquisition of the skilled reaching task with the nonparetic forelimb between these two groups. The first day of nonparetic limb training was 6 days after lesions. B. After training the nonparetic limb, NonParT rats had major deficits in the paretic, contralesional forelimb compared to control animals. C. In contrast, in rats with unilateral SMC lesions and callosum transections performance with the paretic forelimb was not significantly affected by prior nonparetic forelimb training. Day 1 of the paretic limb training period in B and C corresponds to 22 days after the lesions. Data in panels B and C were calculated as %[(preoperative-postoperative)/preoperative] successful retrievals per reach attempt. Data in all figures are means ± SEM. $^{*}p < .05$.

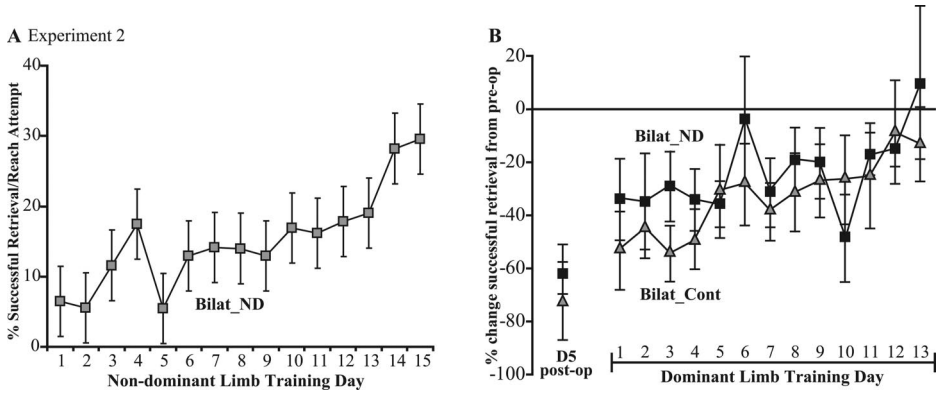


*Figure 3.* Nondominant forelimb training in rats with bilateral SMC lesions does not worsen performance of the dominant forelimb. A. Nondominant limb learning curve of rats with bilateral lesions (Experiment 2). Rats that had learned the task with the dominant limb were learning it for the first time with the nondominant limb after the lesion. The first training day was 6 days postlesion. B. Rats with bilateral lesions had a similar rate of reacquisition of the skilled reaching task with their dominant forelimb regardless of whether they received earlier postlesion training with the nondominant forelimb (Bilat_ND) or earlier control procedures (Bilat_Cont). Panel B shows %[(preoperative-postoperative)/preoperative] successful retrievals per reach attempt. The first dominant limb training day in panel B was 22 days after lesions. Note the differences in scales in comparison to Figure 2.

attempts made with the dominant limb during this training period (Bilat_ND = 37.27 ± 0.85; Bilat_Cont = 35.67 ± 1.36, means ± SEM).

In addition to the lesion-induced deficits in the dominant limb, rats with bilateral lesions tended to perform poorly during the nondominant forelimb training period (Figure 3A) compared to animals in Experiment 1 (as expected because, unlike Experiment 1, this limb was contralateral to a SMC lesion).

## Training the Nondominant/Nonparetic Forelimb Led to its Perseverative Use

Rats with nonparetic forelimb training attempted to use this limb more than controls during the subsequent paretic limb training period (even though the apparatus was configured to only permit successful retrievals with the paretic limb). However, perseverative reaching with the nonparetic forelimb cannot explain the worsening of function of the paretic forelimb in NonParT compared to controls, because this perseverance effect was also found in rats with callosal transections and bilateral SMC lesions.

In Experiment 1, all animals with nonparetic forelimb training made futile reaches with this forelimb ($M = 20.10 \pm 2.87$ reaches) on the first day of the paretic forelimb training period. This compares with only one reach attempt made by only one animal in the Cont group. This effect did not vary significantly as a result of corpus callosum transections. All animals in the CCX_NonParT group made reach attempts with the nonparetic limb on the first day of the switch in sides (23.67 ± 4.65 reaches). In contrast, 4 of 10 rats in the CCX_Cont group made nonparetic reach attempts (1.9 ± 0.95 attempts on Day 1 averaged over all animals in this group). The number of reaches with the nonparetic forelimb in these groups significantly declined over days of paretic forelimb training and, by Day 4, the NonParT groups were no longer significantly different from Cont in this measure.

Consistent with data from Experiment 1, animals trained with their nondominant forelimb after bilateral lesions in Experiment 2 also made significantly more reaches with this limb ($M = 22.0 \pm 4.51$ reaches) on the first day of dominant limb training compared to Bilat_Cont animals ($M = 6.6 \pm 1.78$ reaches, $p < .05$). Reach attempt number with this limb significantly declined over days of training, though in contrast to Experiment 1, animals in both groups ($n = 6$, Bilat_ND; $n = 3$, Bilat_Cont) were still making reach attempts with this limb on Day 13 of dominant limb training (Bilat_ND = 7.2 ± 2.03; Bilat_Cont = 2 ± 0.95).

This perseverance effect was not linked with success levels on the skilled reaching task during the earlier nonparetic/nondominant forelimb training period. There were no significant correlations in either experiment between nonparetic/nondominant forelimb success levels and the number of reach attempts made with this forelimb during the subsequent paretic/dominant training period ($r$'s $< .5$, $p$'s $> .05$). Furthermore, there was no relationship between the severity of the perseverance and paretic/dominant forelimb performance in any group (NonParT, CCX_NonParT, or Bilat_ND, $r$'s $< .6$, $p$'s $> .05$).

## Unilateral, but Not Bilateral, Lesions Resulted in Postural Support Asymmetries

Consistent with previous findings (Allred & Jones, 2004; Allred & Jones, 2008; Barth, Jones, & Schallert, 1990), unilateral, but not bilateral, SMC lesions increased reliance on one forelimb (i.e., the nonparetic forelimb in Experiment 1) as measured on the Schallert cylinder test. Before the unilateral lesions, rats used the to-be-nonparetic forelimb solely for 36.47 ± 2.12% of wall touches and after the lesions they used it for 57.92 ± 2.68% (calculated as %ipsilesional/[ipsi + contra + bilateral]). There were no significant differences in the initial postlesion effects in rats with transections versus no transections (57.54 ± 3.81% and 58.44 ± 3.94%, respectively). Also consistent with previous findings (Allred et al., 2005), training the nonparetic forelimb tended to increase reliance on this forelimb compared with controls (61.43 ± 3.16 vs. 55.12 ± 3.67%), however, this failed to reach signifi-

cance, $F(1, 12) = 3.61$, $p = .11$. This same tendency was not found in animals with transections (CCX_NonParT = 53.73 ± 5.69%; CCX_Cont = 54.23 ± 6.39%) and it was also not found in animals with bilateral lesions (Bilat_ND = 41.38 ± 5.03%; Bilat_Cont = 40.0 ± 2.36%).

## Differences in Injuries Do Not Explain Differential Effects of Nonparetic/Nondominant Limb Training

**Sensorimotor cortex lesions.** SMC lesions produced damage to the forelimb representation area in the region between approximately 2.7 mm rostral and 0.8 mm caudal to bregma (see Figure 4). Lesions also frequently resulted in some superficial white matter damage directly below the lesion (72% of animals in Experiment 1; 55% of animals in Experiment 2). The striatum was considered damaged if the lesions penetrated the white matter under the lesions. With this criterion, striatal damage was incurred in approximately one third of the rats (38% of animals in Experiment 1; 36% of animals in Experiment 2). However, more than superficial striatal damage was not found in any animal.

Placement and extent of SMC lesions were similar between groups within experiments. All animals with unilateral lesions had a significantly smaller dominant hemisphere (due to the lesion) compared to the nondominant hemisphere, NonParT, $t(6) = -4.15$, $p < .01$; Cont, $t(6) = -4.74$, $p < .01$; CCX_NonParT, $t(8) = -5.03$, $p < .01$; CCX_Cont, $t(9) = -4.93$, $p < .01$. There was no difference between groups in either experiment in volume of remaining SMC of the dominant hemisphere (see Table 1) though animals with bilateral lesions in Experiment 2 tended to have larger lesions than animals with the unilateral lesions in Experiment 1 (as was intended due to the larger amount of endothelin-1 used in Experiment 2).

**Callosal transections.** All transections resulted in some damage to the corpus callosum between A/P + 1.2 and −0.3 relative to Bregma. Most transections also produced damage to the septal nucleus ($n = 8$, CCX_NonParT; $n = 9$, CCX_Cont; See Figure 4). In four brains, complete dorsal to ventral transections were not found in any single coronal plane, but major superficial damage was evident in several coronal planes between A/P 0.7 and −0.3 mm relative to Bregma. These variations in callosal injury characteristics were not clearly linked to differences in reaching performance with either limb.

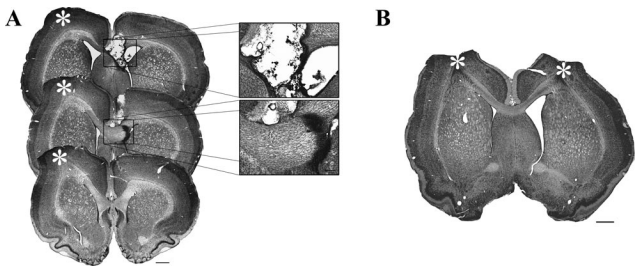


*Figure 4.* Representative lesions and callosal transections. A. Experiment 1, representative lesion and corpus callosum transection. B. Representative lesion from Experiment 2. Scale bars for low magnification images in panels A and B are 1 mm. Scale bar in inset is 250 μm. * indicates SMC damage.

Table 1
*Sensorimotor Cortex Volume ($mm^3$)*

| Experiment 1 | Lesion/dominant hemisphere | No-lesion/nondominant hemisphere |
|---|---|---|
| Cont | 84.51 (2.68) | 95.43 (1.61) |
| NonParT | 82.11 (3.28) | 95.61 (1.11) |
| CCX_Cont | 82.53 (1.22) | 88.63 (1.79)* |
| CCX_NonParT | 83.52 (2.07) | 94.38 (1.36) |
| **Experiment 2** | **Lesion/dominant hemisphere** | **Lesion/nondominant hemisphere** |
| Bilat_Cont | 80.85 (2.63) | 81.96 (5.25) |
| Bilat_ND | 76.61 (1.29) | 81.30 (5.94) |

*Note.* Data are means ± (*SE*).
* $p < .05$, significantly different from CCX_NonParT.

Callosal transections were made using a side of approach opposite the SMC lesions and, as intended, there was no damage from the electrode track evident in the cortex of the dominant hemisphere (the side of the SMC lesions) in any animal. However, despite being matched for initial impairment levels and despite similarity in white matter damage, in histological analysis, the CCX_Cont group was found to have significantly more cortical tissue loss in the side of the transection procedure compared to the CCX_NonParT group, $F(1, 18) = 6.31$, $p < .05$. This is unlikely to contribute to the results because the matched groups had similar deficits in the paretic forelimb as measured 5 days postoperatively (CCX_NonParT = 54.26 ± 9.54% drop from preoperative levels; CCX_Cont = 52.20 ± 10.79% drop from preoperative levels) as a result of matching groups for initial impairment levels. Furthermore, the transected groups were not different on the first day of paretic forelimb training (see Figure 2C). Finally, in secondary analyses of the behavioral results, excluding animals with larger cortical tissue loss on the side of the transection approach did not change statistical outcome on behavioral measures. For example, when three animals with the largest cortical damage in the CCX_Cont group were excluded such that, in the remaining animals, the cortical volume (91.78 ± 1.15 $mm^3$) was similar to that of CCX_NonParT (see Table 1), this resulted in little effect on mean values of reaching success during the paretic limb training period. In addition, the subgroup of CCX_Cont (excluding the three animals with the largest cortical damage) performed at 17.43 ± 6.38% of preoperative levels on paretic limb training Days 7–9, which was similar to the inclusive group and to CCX_NonParT (Figure 2C). The subgroup of CCX_Cont continued to be nonsignificant compared with CCX_NonParT ($F(1, 14) < 1.0$, $p = .85$). Furthermore, there was no correlation between transection-associated cortical injury and paretic limb performance in either group ($r$'s $< .5$, $p$'s $> .05$). Thus, there is no clear relationship between this secondary cortical damage and the attenuation of the nonparetic (NonParT) limb training effects by CCX.

## Discussion

The present results add more support to the finding that learning a skilled motor task with the nonparetic forelimb worsens performance and relearning with the paretic forelimb (Allred et al., 2005; Allred & Jones, 2008). This maladaptive effect was absent in

animals with transections of the corpus callosum. Furthermore, the effect was not reproduced in rats with bilateral lesions of the sensorimotor cortex that underwent equivalent sequential training of the two limbs. These data suggest an involvement of both the SMC of the contralesional hemisphere and of transcallosal projections in the maladaptive effects of nonparetic forelimb training on the function of the paretic forelimb.

This present study identifies a circuit that is required for the maladaptive effects of training the nonparetic limb, but the mechanisms remain to be uncovered. Reorganization in peri-lesion cortex is thought to be important for recovery of the paretic side after unilateral damage in both humans and animal models (e.g., Kleim, Barbay, & Nudo, 1998; Cramer, 2008). Previously, we found that nonparetic forelimb training disrupts the perilesion neuronal expression of FosB/ΔFosB resulting from paretic limb training (Allred & Jones, 2008). ΔFosB is a cumulatively expressed transcription factor involved in instigating structural plasticity (McClung et al., 2004) that is likely to be sensitive to the repetitive practice involved in reacquisition of the skilled reaching task with the paretic limb. Thus, it is possible that activity-dependent plasticity in perilesion cortex is disrupted by experience with the nonparetic forelimb.

Several other lines of evidence suggest that one hemisphere and body side can constrain activity and plasticity in the other, even in intact animals. Unilateral deprivation of sensory input in one arm (Floel et al., 2004), forelimb (O'Bryant, Bernier, & Jones, 2007), eye (Iny, Heynen, Sklar, & Bear, 2006), or whisker pad (Li et al., 2005) enhances somotosensory and motor abilities and experience-dependent plasticity (Glazewski et al., 2007) of the nondeprived side. Lidocaine inactivation of primary motor cortex in rats results in an expansion of the motor map in the contralateral hemisphere (Maggiolini, Viaro, & Franchi, 2008). It may be that this "normal" constraint is exaggerated after unilateral lesions and further exaggerated by experience with the nonparetic limb, and that these effects can be attenuated when transcallosal connections are severed. Unilateral lesions or transient inactivation of cortex are known to alter activity in the other hemisphere (Li, Rema, & Ebner, 2005; Clarey, Tweedale, & Calford, 1996). For example, there is increased excitability in the homotopic contralesional cortex (e.g., Que, Schiene, Witte, & Zilles, 1999; Witte, Bidmon, Schiene, Redecker, & Hagemann, 2000; Witte & Stoll, 1997). Furthermore, in humans, an abnormal inhibitory drive from the contralesional motor cortex to the damaged hemisphere is found in stroke patients preceding voluntary paretic hand movement (Murase et al., 2004).

Individuals with severe hemiplegic cerebral palsy develop an increase in ipsilateral corticospinal projections from the "intact" hemisphere, which Eyre and colleagues (2007) conclude may be competitively displacing contralateral projections from the infarcted cortex thereby making impairments worse. This raises the possibility that the experience with the nonparetic limb confiscates circuits that might otherwise mediate recovery of the paretic limb. If so, the role of callosal fibers in such a confiscation, and the timing of their involvement remains to be established. Training one forelimb in rats results in increases in synapses (Luke et al., 2004) and dendrites in the hemisphere opposite the trained limb (Greenough, Larson, & Withers, 1985; Bury & Jones, 2002; Allred & Jones, 2004) and involves mechanisms similar to long-term potentiation (LTP; Rioult-Pedotti, Friedman, & Donoghue, 2000; Monfils & Teskey, 2004). The LTP-like changes have been found to be specific to the contra-to-training hemisphere (Rioult-Petdotti et al., 2000; Monfils & Teskey, 2004). Furthermore, after unilateral SMC lesions, the training-related neuroplastic effects are enhanced in the cortex opposite the lesions (Bury & Jones, 2002; Jones, 1999; Jones, Chu, Grande, & Gregory, 1999; Luke et al., 2004) and this is linked with an increased capacity to learn a motor skills task with the nonparetic forelimb (Allred & Jones, 2004; Bury & Jones, 2002; Hsu & Jones, 2006). However, some bilateral dendritic growth of layer II/III pyramidal neurons has been observed after unilateral training in intact animals (Greenough, Withers, & Larson, 1985). If such ipsi-to-training neural plasticity occurs in perilesion cortex, this might reduce the ability to create further changes by training the paretic forelimb.

Rats trained with the nonparetic limb also perseverated in the attempt to use this forelimb after switching to the paretic forelimb, despite the task configuration making reaches with this limb futile. However, this effect cannot be responsible for exacerbating deficits in the paretic limb as it was seen in all animals with nonparetic/nondominant training, including those with corpus callosum transections and bilateral lesions. Furthermore in correlation analyses, it was not associated with deficits in paretic forelimb skilled reaching performance. This suggests that use of the nonparetic forelimb *while the paretic forelimb is being used* is not necessarily maladaptive. This adds to earlier findings that training rats to reach with both forelimbs after unilateral lesions does not worsen paretic forelimb function (Allred & Jones, 2008). Furthermore, the task learned with the nonparetic forelimb does not have to be one that was originally performed with the paretic side. In rats naïve to the reaching task prior to the lesions, postlesion training of the nonparetic forelimb results in a pronounced deficit in learning this task later with the paretic forelimb compared with controls (Allred et al., 2005). This may indicate that establishment of preinjury dominance for the task is not a critical factor in this effect.

While all rats did show some motor recovery with training of the paretic forelimb, the rate of relearning was slowed greatly after nonparetic limb training compared to control rats. It is possible that longer training of the paretic forelimb could overcome the maladaptive effects of prior nonparetic forelimb experience and learned disuse of the paretic forelimb. Furthermore, though the effect was not significant, there was a tendency for rats to perform worse with the paretic limb after nonparetic limb training even in callosally transected rats. This could be because the transection approach reduces, rather than eliminates, callosal fibers, but it might also indicate that there are at least some noncallosally mediated effects of this behavioral manipulation. Additionally, CCX_NonParT had a tendency to perform better with their paretic forelimb compared to NonParT rats. This effect may have failed to attain significance because the transections used in this study were partial and most likely did not completely obliterate interhemipsheric communication.

Following unilateral SMC lesions, even in the absence of any training, animals begin to rely more on their nonparetic forelimb. Compensatory reliance on this side for postural support is evident in home cage observations (Jones & Schallert, 1992) and it may be that these self-taught behaviors are also limiting recovery of the paretic forelimb. By training them in a skilled motor task that is not performed in the home cage environment, we may be exaggerating these effects. Furthermore, the nonparetic limb training effects

have been found to generalize to nonreaching behaviors, including coordinated forelimb placement in a grid walking task and postural support in upright exploratory movements (Allred & Jones, 2008). Motor skill training with the nonparetic limb in animals with unilateral lesions may therefore induce a greater use of this limb, at a cost of disuse of the paretic forelimb. However, in the present study, this postural support effect failed to reach significance, in contrast with previous findings. A more sensitive measurement of forelimb use for postural support may reveal greater experience-dependent effects in asymmetrical forelimb use for postural support behaviors.

The present results provide further support that learning new ways of using the nonparetic limb to compensate for impairments can be detrimental to recovery of function with the paretic forelimb (Allred & Jones, 2008; Allred et al., 2005), and probably exacerbate learned nonuse or learned bad use (Taub et al., 2003; Alaverdashvili, Foroud, Lim, & Whishaw, 2008). The exact mechanism(s) mediating this effect are still unknown; however, data from this study point to interhemispheric involvement and disruptive behavioral experience on recovery. The maladaptive effects of experience with the nonparetic body side may need to be overcome with treatment approaches, such as CIMT (Taub et al., 2003) and facilitating stimulation of the perilesion cortex (Adkins-Muir & Jones, 2003; Plow, Carey, Nudo, & Pascual-Leone, 2009). However, the optimal application of these strategies might be improved with a better understanding of the exact neural mechanisms of the present phenomenon, including the time periods in which intercortical interference is high. Though the present results indicate that the effect is mediated by intercortical connections, further investigation is needed to isolate the time period of their involvement as well as to understand exactly *how* they are disrupting the function of the paretic forelimb. A better understanding of the phenomenon seems likely to illuminate processes involved in neglect and learned nonuse.

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# Neurorehabilitation splinting: Theory and principles of clinical use

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**Abstract**. The use of splints in neurorehabilitation is common, with splints being used to meet varied clinical aims. This paper explores the use of splints after stroke and examines the rationale underpinning current use. It covers the use of splints to reduce spasticity, prevent contracture and improve activity. As well as presenting the theoretical rationale underpinning splinting as an intervention, it examines the current evidence from randomised trials testing the theories. In summary, there is strong evidence that wearing a splint all night has no additional effect in reducing spasticity over usual therapy or in preventing contracture, whether the wrist is splinted in neutral or in maximum extension. It is not surprising that splinting has not shown an effect on activity, given that there was little effect on the impairments that it was directed towards. In conclusion, it is now time to re-focus on improving muscle performance in order to enable activity rather than preparing the patient for function by affecting abnormal reflex activity.

## 1. Introduction

The aim of this paper is to trace the use of splints in neurorehabilitation and to examine the rationale underpinning current use. With the terms splint, brace, and orthosis being used interchangeably, we have chosen to use the term splint since it is widely accepted. We will cover the use of splints to reduce spasticity, prevent contracture and improve activity after stroke. The emphasis will be on the theoretical rationale underpinning splinting as an intervention and the current evidence from systematic reviews and randomised trials.

### *1.1. History of use of splints*

Using splints in neurorehabilitation is not a new concept, and yet clinicians are often not aware of the history and theories beyond their own experiences. Understanding the premise on which our clinical decision making is based strengthens the foundation of our clinical practice.

The definition of terms provides a foundation from which to work. A splint is defined as being a removable device designed for the support of weak or ineffective joints or muscles [1]. Text books cite that the purpose of splints are variously to increase function, prevent deformity, correct deformity, substitute for lost motion, protect healing structures, maintain range of motion, stabilise joints, restrict motion, allow tissue remodelling, improve muscle balance, control inflammation, protect normal structures, decrease pain, strengthen weak muscles, reduce spasticity, and increase patient independence.

The earliest application of splints in neurorehabilitation can be traced back to the late 1500s when metal splints were used to manage contracted joints. Splinting today has become an accepted and integral part of neurorehabilitation, from the time of admission to long after formal rehabilitation has ended. However, there is much inconsistency in the way splints are used.

### *1.2. Current use of splints*

A splint offers a therapeutic means of maintaining specific positions of a limb. Splints may be static; not

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allowing motion, or dynamic. Static splints are able to immobilize a joint in any position that the therapist chooses, thus providing a means of either rigidly supporting body structures or applying a prolonged stretch to muscles or skin [1–6]. The American Society of Hand Therapists [7] classify splints as: mobilization, immobilisation and restrictive. This classification is based on the key functions of the splint. Mobilisation splints are designed to mobilize joints, muscles and/or skin, while immobilisation splints aim to immobilise. Restrictive splints limit a specific aspect of movement for a specific purpose, as in the case of tenodesis splints.

The use of splints in neurorehabilitation has historically developed from the clinical experiences of therapists [8]. In general, however, therapists apply a splint to achieve one or more of the following aims:

- To decrease spasticity [4,6,8–10].
- To prevent or reduce contractures. The wrist and fingers assume a 'relaxed' position of flexion following acquired brain impairment, which contributes to the formation of a contracture. The splint is thought to act as an opposing force against the flexion contracture by providing a sustained stretch [2,4,11,12]
- To improve activity at a joint. For example, positioning a flexed wrist in more extension may place the fingers at a better position for active movement [4,6,13]. Static splinting in a functional position is usually considered to maintain correct joint alignment and increase the patient's ability to use their hand while more controlled movement is being regained [2,4,12].
- To protect joint integrity by immobilising the joint which is believed to decrease mechanical irritation caused by overstretching of a joint. Overstretching is thought to occur due to decreased proprioception within the joint following acquired brain impairment [4,9].
- To reduce pain [6,9,10].

There has been much debate about the mechanism by which splinting appears to be effective [14,15]. Originally, development and subsequent research regarding splinting was driven primarily by the theory that splints inhibit reflexive contraction of muscles (the neurophysiological rationale). With increased knowledge about both the neurological and musculoskeletal systems, clinicians continued to use splints but theorized that splints position the limb into a biomechanically advantageous position. While both the neurophysiological and biomechanical approaches have their advocates and opponents, there is a lack of consensus about the design, wearing duration and wearing compliance of splints. In short, the use of splints for people during neurorehabilitation remains controversial.

It is likely that much of the controversy surrounding splinting in neurorehabilitation may be eliminated if therapists had a good working knowledge of the capabilities and limitations of each type of splint, as well as a theoretical understanding of the scientific evidence underpinning these clinical opinions. This paper will cover the use of splints to reduce spasticity, prevent contracture and improve activity. For each topic, the clinical construct for the use of splinting will be covered, followed by the theoretical rationale for splinting, and finally the practical evidence will be put forward. Splints that are static and removable will be investigated, ie, casting will not be included because casts are not removable on a daily basis. Clinical applications for adults after stroke will be highlighted because these individuals are the largest group with brain damage seen by therapists.

## 2. Splinting to decrease spasticity

### *2.1. Clinical construct*

Many commonly used splints in neurorehabilitation are applied with the aim that they will inhibit spasticity with an end result of improving activity. This of course depends on the premise that (a) a splint is able to inhibit spasticity, and (b) that inhibiting spasticity leads to greater activity. The points of contact of a splint are thought by some clinicians to impact on whether or not a splint inhibits or elicits spasticity. Such thoughts have stemmed from early publications (Rood [16]) where spasticity was thought to increase as a result of sensory stimulation of the palmar surface of the hand, which would then result in unwanted muscle contractions. Based on these assumptions, many therapists recommend splinting on the dorsal surface of the hand only. In addition to concerns that splinting the flexor surface of the hand will "trigger" spasticity, there is also a belief by some clinicians that certain positions "break" patterns of spasticity. Stemming from a single case study conducted in 1962 [17], some therapists believe that to inhibit spasticity the hand must be positioned with the wrist in neutral, and the fingers abducted and extended.

### 2.2. Theoretical rationale

In neurologically-normal people, passive stretch of relaxed muscles does not result in reflex muscle activity and the limb feels normal, ie, neither stiff nor exceptionally loose. The presence of exaggerated stretch reflexes in spastic patients means that passive stretch of relaxed muscles elicits reflex activity which results in increased resistance, ie, hypertonia. Hypertonia needs to be clearly distinguished from reflex hyperexcitability in patients with spasticity. The most widely accepted definition of spasticity is that of Lance [18] who described it as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome". Furthermore, the specificity of this definition was reiterated [19] with the added rider that "spasticity does not include impaired voluntary movement and an abnormal posture". Therefore, the primary feature of spasticity is the exaggeration of stretch reflexes. Some of the confusion about clinically identifying spasticity has likely arisen because the clinical measurement of spasticity involves gauging the resistance of the limbs to passive movement (ie hypertonia) [20]. This procedure does not allow different causes of an increase in resistance to be identified.

It is now well recognised that factors other than reflex hyperexcitability may produce an increase in resistance to passive movement [21–23], the most common being muscle contracture. As a consequence of this, unless stretch-evoked muscle activity can be demonstrated via EMG, an increased resistance to passive movement cannot automatically be attributed to reflex hyperexcitability. O'Dwyer et al. [20] found that 13 out of 24 (54%) people less than one year after stroke exhibited an increase in resistance to stretch, ie, hypertonia, but only 5 out of 24 (21%) exhibited stretch-related muscle activity, ie, spasticity. Perry [21] reported no stretch-related muscle activity in 10% of people who were labelled as spastic after stroke and Lin et al. [24] suggested that up to 30% of hypertonia after stroke may not be due to spasticity.

This places importance on the measurement tool used to quantify spasticity. It is now well recognized that the most commonly used tool – the Ashworth Scale – is deficient in quantifying spasticity because it cannot differentiate spasticity from contracture [25–28]. This is because it is mostly a scale assessing hypertonia. A study of the validity of the Ashworth Scales versus the Tardieu Scale found that the Ashworth Scale overestimated the incidence of spasticity in stroke 15% of the time [29]. In all of these cases, participants had a contracture, suggesting that the Ashworth Scale is confounded by contracture. The Tardieu Scale is a more valid measure of spasticity, probably because it takes into account the main factor to which the stretch reflex is known to be sensitive – the velocity of stretch. This velocity-dependence of the stretch reflex has been well established (eg, Thilmann et al. [30]).

Although splinting will reduce stimulation of hyperreflexia by immobilising the joint, this does not mean that it will reduce spasticity in the long term when the splint comes off. Importantly, the incidence of spasticity after stroke is quite low with O'Dwyer et al. [20] reporting it at 21%, Watkins et al. [31] reporting it at 27%, Sommerfeld et al. [32] and Welmer et al. [33] at 19%, Lundstrom et al. [34] at 17% and Wissel et al. [35] at 22%. It is interesting to note that most of these studies used the Modified Ashworth Scale to quantify spasticity which overestimates spasticity. Furthermore, studies investigating the link between spasticity and activity have found that the two are not correlated after stroke [20,36] and that when spasticity has been reduced it does not necessarily lead to better activity [37]. These findings suggest that routine intervention for reducing spasticity during rehabilitation after stroke, in particular splinting, is inappropriate.

In terms of tactile stimulation exacerbating spasticity, the primitive reflexes sometimes seen after brain injury (such as the palmer grasp reflex) are cutaneous reflexes whereas spasticity is a disorder of the proprioceptive reflexes. Furthermore, it is more common to observe grasp reflexes after traumatic brain injury rather than after stroke. Therefore, it is not likely that tactile stimulation from a volar hand splint will trigger spasticity.

### 2.3. Practical evidence

Is there evidence that spasticity can be reduced by splinting after stroke? There have been four randomized trials carried out examining the benefits of splinting to reduce spasticity after stroke.

Two randomized trials examined whether spasticity was reduced as a result of splinting. One high quality trial (PEDro score 8/10) compared splinting with the wrist in neutral overnight versus splinting with the wrist extended overnight versus a no splint intervention for 4 weeks in 63 stroke patients [38]. There was no difference in the Tardieu spasticity angle between the

wrist extended splint and no splint (mean difference 1 deg, 95% CI −2 to 5). One low quality trial (PEDro score 1/10) compared wearing a finger-spreader splint for 6 hours versus 12 hours versus 22 hours/day over 2 weeks in 9 stroke patients [39]. There was no difference in wrist stiffness (hypertonicity) between the different lengths of time wearing the splints (mean difference 0.00 Nm.rads, 95% CI –0.42 to 0.44).

Two randomized trials examined whether dorsal splinting reduced spasticity more than volar splinting. One moderate quality trial (PEDro score 4/10) compared splinting the hand in the functional position in either volar or dorsal splints for 2 hours in 10 stroke patients [40]. Both volar and dorsal splints resulted in an immediate statistically significant decrease in hypertonus. One low quality trial (PEDro score 2/10) compared splinting the hand in dorsal versus volar splints for 2 hours/day in 30 stroke patients [12]. There was no significant difference between the splints in decreasing hypertonus (mean difference 0.1 lb, 95% CI −1.4 to 1.5).

In summary, there is strong evidence that wearing a splint all night has no additional effect in reducing spasticity over usual therapy. Furthermore, even wearing the splint up to 22 hours per day did not affect spasticity. Results from studies also suggest that there is no difference between using a dorsal splint to a volar splint – this is not surprising since people after stroke rarely exhibit exaggerated cutaneous reflexes.

## 3. Splinting to decrease contracture

### *3.1. Clinical construct*

Neurological conditions are often accompanied by physiologic joint restriction and contractures [3]. Splints are used by clinicians with the aim of maintaining or lengthening soft tissues and maintaining joint integrity [41].

Submaximal range splinting is still used in neurorehabilitation, despite a lack of evidence underpinning its efficacy. The design is based on the reasoning that muscles splinted on full stretch or maximal range will increase hypertonicity. Although rarely seen any longer that a clinician would splint a (for example) wrist in flexion to address a flexion contracture, it does remain common practice to splint to decrease contracture at less than full available range. For instance, text books cite a position commonly referred to as “functional” as being the optimal position for handsplinting, that is, 20 to 30 degrees of wrist extension [4]. The use of the ‘functional’ position has not been supported scientifically and appears to contradict the beneficial effects of full stretch in animal studies. An unwillingness to provide full stretch when splinting has meant that a potentially effective treatment has not been offered to a patient by therapists. This rejection may deprive people following acquired brain impairment of an opportunity to apply stretch at its optimal muscle lengthening efficacy if, in fact, stretch to end of joint range is effective [42,43].

### *3.2. Theoretical rationale*

Contracture is the loss of joint range of motion. This is partially the result of a shortening of muscle length due to a decrease in the number of sarcomeres in series along the myofibrils [44]. It is accompanied by an increase in the resistance to passive stretch which is probably attributable to remodelling of muscle connective tissue [44]. The range of joint motion is reduced both by the shortening of the muscle fibres and by the loss of muscle compliance.

Contracture can be easily produced in experimental animals via immobilisation of muscles in shortened positions. Similar prolonged muscle shortening may arise in humans through immobilisation or muscle imbalance. Furthermore, in stroke patients with reduced range of elbow extension, O’Dwyer et al. [20] measured increased passive resistance during elbow extension that was independent of muscle activity. Contracture is therefore an important contributor to hypertonia and the potential to confuse this with spasticity is clear.

Recognition of the role of increased passive tissue stiffness is crucial in the measurement of muscle contracture. When assessing range of joint motion, it is important that the force applied is standardized and does not exceed the magnitude of force that is normally sufficient to stretch the muscles through the joint range. Even if a muscle has some contracture, it may still be possible to achieve a full range of joint motion if sufficient force is applied [45] and the increased stiffness wrongly be attributed to spasticity.

Animal studies have shown that positioning at-risk muscles in the lengthened position for prolonged periods of time, whether by casting or suspension, has resulted in the prevention of loss of sarcomeres in series [46]. This maintenance of sarcomeres was presumed to be accompanied by prevention of loss of muscle length and joint range of motion. Therefore, based on these principles, splinting at-risk muscles in a maxi-

mally lengthened position for considerable proportions of the day should have an effect on maintaining the length of muscles after stroke.

### 3.3. Practical evidence

Is there evidence that contracture can be prevented by splinting after stroke? There have been five randomized trials examining the benefits of splinting to prevent contracture after stroke.

Three randomized trials examined whether contracture was prevented/reduced as a result of splinting. One high quality trial (PEDro score 8/10) compared splinting with the wrist in neutral overnight versus splinting with the wrist extended overnight versus a no splint intervention for 4 weeks in 63 stroke patients [38]. There was no difference in range of motion of wrist and finger flexors between the wrist extended splint and no splint (mean difference −1 deg, 95% CI −4 to 2). Another high quality trial (PEDro score 8/10) compared hand splinting in the neutral position overnight with no splint for 4 weeks in 28 stroke patients who were also having daily upper limb stretches [47]. There was no difference in range of motion of wrist and finger flexors between the splint and the no splint group (mean difference 1 deg, 95% CI −4 to 6). A third high quality trial (PEDro score 8/10) compared wearing a splint with the wrist in neutral and no finger support 6 hours/day versus no splint for 13 weeks in 30 stroke patients [48]. There was no difference in the proportion of participants having a contracture (defined as <2/24 on Fugl Meyer Assessment joint range of motion subtest) between the splint and no splint groups (risk difference 27%, 95% CI −8 to 54).

One randomised trial examined splinting in the lower limb versus another intervention for prevention of contracture. This high quality trial (PEDro score 8/10) compared wearing a splint with the affected ankle at plantargrade 7 nights (12 hr) per week with standing on a tilt table for 30 min with the ankle at maximum dorsiflexion 5 times per week in 30 stroke patients over 4 weeks [49]. They found no difference in range of ankle dorsiflexion (mean difference 1 deg, 95% CI −5 to 7). Both groups did not develop a contracture; however, since there was no control group, the prevention of contracture may have been due to other factors.

A last randomized trial examined the immeditate effect of two different splints. This moderate quality randomized trial (PEDro score 4/10) compared splinting the hand in the functional position in either volar or dorsal splints for 2 hours in 10 stroke patients [40]. They found that either volar or dorsal splints resulted in an immediate statistically significant increase in passive range of wrist extension; however, point measures and measures of variability were not reported.

In summary, there is strong evidence that wearing hand splints all day or night additional to usual therapy after stroke has no effect in preventing contracture, whether the wrist is splinted in neutral or in maximum extension. Furthermore, it appears that there is no difference between using a splint to other means of contracture prevention.

## 4. Splinting to improve activity

### 4.1. Clinical construct

Splints are also used in neurorehabilitation to improve activity, be that by holding a joint in a position that assists in an activity, such as in the case of thermoplastic molded ankle-foot splint, or a thumb abduction splint which positions the thumb in an enhanced prehension position for grasp/release; or by compensating for weakness by providing external support or movement, such as in the case of a posterior leaf spring, ankle-foot splint, or newer technology advances such as electronic stimulation splints (BioNESS) or dynamic handsplints such as Saebo splint. Many neurorehabilitation protocols, particularly in the management of the upper limb after stroke, call for long-term splinting.

### 4.2. Theoretical rationale

Given that wearing a splint, particularly early in a rehabilitation training program, sends a message that an external positioning device is responsible for aligning a joint, it is likely that the patient may fail to integrate movement training from therapy into everyday movement training. Thus, the concern that early and/or continual splinting predisposes a patient to learned nonuse.

One of the disadvantages of using splinting is that it effectively immobilises the joint(s) and therefore discourages or even disallows muscle activity and therefore movement. For example, when wearing an ankle-foot splint, the ankle is immobilised at plantargrade and this results in a decrease in muscle activity in the dorsiflexors [50]. For this reason, it would seem inappropriate to prescribe splints during the early stages of rehabilitation, when the emphasis is on using the neural plasticity of the system to harness the potential for re-

covery of muscle strength. On the other hand, using an ankle-foot splint often increases confidence and results in faster walking with more symmetry [51,52], making it more appropriate for ongoing use once recovery has effectively reached a plateau. In this situation, there is not necessarily an expectation that activity will improve as a result of wearing the splint. For example, wearing an ankle-foot splint for 6 months resulted in deterioration when walking without the splint in stroke survivors.

### *4.3. Practical evidence*

Is there evidence that activity can be improved as a result of short-term splinting after stroke? There have been four randomized trials examining the effect of splinting on activity after stroke.

Few of these randomised trials investigated splinting where the main aim is to improve motor activity. One moderate quality trial (PEDro score 5/10) compared wearing an inflatable pressure splint with the shoulder in 90 degrees of flexion and maximum external rotation and the elbow fully extended versus a no splint intervention for 30 minutes/day over 3 weeks in 18 stroke patients [54]. In terms of activity, it found no difference in Fugl-Meyer Assessment scores (mean difference 0/57, 95% CI $-10$ to 10) between the splint and the no splint group.

Most randomized trials have investigated splinting where the main aim is to reduce impairments such as spasticity or contracture and the carry over to motor activity has been measured as a secondary outcome. One high quality trial (PEDro score 8/10) compared splinting with the wrist in neutral overnight versus splinting with the wrist extended overnight versus a no splint intervention for 4 weeks in 63 stroke patients [38] in order to prevent contracture. In terms of activity, it found no difference in Motor Assessment Scale scores for the upper limb between the wrist extended splint and no splint (mean difference 0.0/18 points, 95% CI $-0.4$ to 0.4). Another high quality trial (PEDro score 8/10) compared hand splinting in the neutral position overnight with no splint for 4 weeks in 28 stroke patients who were also having daily upper limb stretches [47] in order to prevent contracture. In terms of activity, it also found no difference in Motor Assessment Scale scores for the upper limb (mean difference 0.1/18 points, 95% CI $-2.3$ to 2.7) between the splint and the no splint group. A third high quality trial (PEDro score 8/10) compared wearing a splint with the affected ankle at plantargrade 7 nights (12 hours) per week with standing on a tilt table for 30 min with the ankle at maximum dorsiflexion 5 times per week in 30 stroke patients over 4 weeks [49] in order to prevent contracture. In terms of activity, it found no difference in Motor Assessment Scale scores for standing up from a chair (mean difference 0.5/6 points, 95% CI $-0.4$ to 1.4).

In summary, its not surprising that splinting had little effect on activity, given that there was little effect on the impairment (such as contracture or spasticity) that it was directed towards. A major obstacle to functional use of the hand is the inability to open the hand spontaneously, and the potential benefit of dynamic and newer technology splints on hand opening, activity and functional use has yet to be adequately studied in clinical trials.

## 5. Summary

As the rehabilitation of adults following stroke consumes substantial health resources, it is crucial that an evidence-based approach be adopted in response to controversies and clinical uncertainties, such as those abundant in the debate about splinting. Despite the widespread use of splints for adult stroke patients, surprisingly few randomized trials ($n = 5$), have examined the effect of splinting in this population. However, several of these are of high quality and can be used to guide clinical practice. Unfortunately, the evidence suggests that splinting as currently provided in neurorehabilitation is not effective in decreasing spasticity, preventing contracture or improving activity. The time may have come to reconsider our clinical practices and beliefs.

It is unsurprising that static splinting has little effect on activity, given that these splints are provided primarily to influence contracture development or decrease spasticity with an anticipated improvement in activity that should occur with less contracture and/or less spasticity. Static splinting has not been able to demonstrably reduce either spasticity or contracture and since it was shown to do neither, a subsequent effect on activity was also not detected. Studies to date have focused their attention on investigating the benefits of static splints and thus the potential benefit of dynamic and newer technology splints such as those that deliver electrical stimulation or EMG-triggered stimulation have yet to be adequately studied. While further research is needed on the many possible interventions for increasing activity, including splint use and how they contribute to maximizing functional use, future stud-

ies should use large sample sizes and simple, accurate, and reliable measures of relevance to the primary clinical reasons for applying splints which will ensure the clinical utility of study findings.

Examination of current evidence for splints to reduce spasticity and prevent contracture shows that clinical theories held by neurorehabilitation professionals require reconsideration. Clinicians are encouraged to re-focus on improving muscle performance in order to enable hand activity, rather than preparing the patient for function by affecting abnormal reflex activity.

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# Anticipatory Postural Adjustments: Translating Research Methodology to the Clinic

## Israel 2014

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## References


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## A New Look At Neurologic Shoulder Pain

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