The Role of Technology in Task-Oriented Training in Persons with Subacute Stroke: A Randomized Controlled Trial

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This trial compares the effects of task-oriented physical therapy (PT) provided with and without the use of rehabilitation technology on locomotor recovery in 63 persons with subacute stroke. Participants in the experimental (EXP) group used a treadmill, a Kinetron isokinetic exerciser, and a limb-load monitor, whereas those in the control (CTL) group did not while engaging in PT 1 h per day, 5 days per week for 2 months. Locomotor recovery was assessed by clinical (gait speed, Fugl Meyer motor leg and arm subscores, the Balance Scale, the Timed Up and Go, and the Barthel ambulation subscore) and laboratory outcomes (gait kinematics and kinetics) pre- and posttherapy and 3 months later. Within groups, gait speed ($P < 0.01$) and all secondary measures improved posttherapy ($P < 0.01$–0.05), and improvements in clinical measures were maintained at follow-up, but there was no difference between groups ($P > 0.05$). When the groups were pooled, the increase in gait speed was associated ($r = 0.52$, $P = 0.003$) with an increase in ankle power generation of the affected leg. The results demonstrate that the efficacy of the task-oriented approach is not dependent on rehabilitation technology.

Key Words: *Subacute stroke—Task-oriented—Locomotor training—Physical therapy—Gait speed—Randomized controlled trial.*

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Each year about 35,000 Canadians will sustain a new stroke[1] and 60% to 80% of these individuals will initially lose the ability to walk independently.[2-4] At 3 months after stroke, one quarter to one third of stroke survivors require assistance or supervision while walking.[2,3,5] A number of studies since the 1950s have shown with clinical measures that most of the locomotor recovery occurs in the first 5 to 6 weeks after stroke[2-4,6-8] when rehabilitation, including physical therapy, is provided and presumed to augment "natural recovery."

Over the years, different physical therapy (PT) approaches[9-11] have been advocated to promote recovery, but clinical trials have failed to demonstrate a clear difference in efficacy.[12,13] In the past 20 years, however, the theoretical bases of motor learning and motor behavior,[14-16] studies of locomotor recovery in animal models,[17] and results of studies evaluating locomotor recovery in acute stroke survivors have promoted the task-oriented approach.[7,12,18-20] Importantly, task-oriented training has been related to the reorganization of the brain after an infarct. For example, Nudo and others[21] elegantly demonstrated with electrophysiological maps of the cortex in monkeys recovering from discrete cerebral lesions that task-oriented, timely practice of increasing difficulty modulates the plasticity of the brain after a lesion. In humans, transcranial magnetic stimulations have been used to map out practice-related changes in the brains of persons with stroke who practiced specific movements of the affected hand,[22] whereas Sullivan and others[23] documented activity-dependent cortical reorganization using fMRI mapping of the brains of persons with stroke who practiced walking on a treadmill 1 h a week for 4 to 10 weeks. The fMRI changes were correlated with faster over-ground walking speeds and more precise voluntary control of the tibialis anterior muscle.

When targeting locomotor recovery, one can question whether a generalized approach to PT is appropriate when the salient component in the disturbed motor control underlying the walking disability of a person poststroke may be paresis, hyperactive stretch reflexes, excessive coactivation,[24-27] increased stiffness,[28-30] or a combination of these components. The choice of therapy can be guided by knowledge of the type of disturbed motor control,[24,31,32] but this requires that the patient be able to walk and the availability of a gait laboratory. It can be assumed that the motor control impairments that affect the ability to walk will impact the capacity to produce the propulsive force[33] that is created by the generation of power by the ankle plantarflexors at the end of the stance phase (push-off), aided by the hip extensors at the beginning of the stance phase and the hip flexors at swing phase initiation (pull-off).[34-36]

Walking speed after stroke is generally slower and associated with a deficient plantarflexor power burst (A2).[34,35,37] To compensate for the loss of push-off power, persons after stroke tend to augment the contribution of the hip flexor power burst (H3) in early swing.[34,35] Olney and her colleagues[34,36,38,39] advocated the importance of training the ankle plantarflexors and hip flexors in a task-specific manner to promote walking speed, and 2 recent studies noted that chronic stroke survivors who participated in a PT program involving the accomplishment of a circuit of locomotor-related tasks,[40] or muscle strengthening and physical conditioning,[36] walked significantly faster posttherapy, owing, at least in part, to an improved A2 power burst.

The present study is the sequel to a randomized controlled pilot study carried out by our group.[7] The pilot study found that persons who had sustained a stroke due to middle cerebral artery infarct and who received 5 weeks of task-oriented physical therapy[18] beginning about 1 week after stroke onset walked faster than those receiving conventional therapy. The benefits of the approach, as measured by gait speed at 6 weeks poststroke, disappeared by 3 months after onset.

The present randomized controlled trial was planned to evaluate the task-oriented PT approach, developed for the pilot study,[18] under conditions of usual clinical practice in rehabilitation centers. The study was initially designed to determine the efficacy of a technology-driven task-oriented approach as compared to conventional therapy. Thus, in the experimental (EXP) group, the objective was to offer optimal training conditions with technological devices: a treadmill to practice walking safely and intensively, a Kinetron for muscle strengthening while performing walking movements, and use of a limb-load monitor to specifically train loading of the affected limb in standing and stepping positions. The control (CTL) group, on the other hand, received locomotor training by walking over ground under varying conditions without the help of special devices. As the study progressed, it became evident that conventional therapy was highly influenced by the task-oriented approach,[15,41] which advocates the practice of locomotor-related tasks in changing environments and conditions (e.g., inclines, stairs, outdoors). Because the therapist diaries of the therapy provided to both groups documented a similar number of hours devoted to the practice of locomotor-related tasks in both groups, this study evolved into a comparison of 2 modes of delivering task-oriented locomotor training. The main results of this trial were presented at the World Conference for Physical Therapy.[42]

METHODS

Participants

A total of 63 persons with a stroke participated in the study. All persons admitted to 2 rehabilitation units in Québec City—the Institut de réadaptation en déficience physique de Québec, site François Charon (CFC) and the Centre Hospitalier Saint Augustin (CHSA)—for rehabilitation following a stroke were screened by a physiotherapist coordinator for entry into the study. The inclusion criteria included men and women between 30 and 89 years old with a 1st or 2nd episode (if good recovery from the 1st stroke was documented) of hemiplegia of thromboembolic origin with a residual deficit, right- or left-sided lesions that affected the ability to walk but not the ability to understand and follow verbal instructions. To ensure that the patients would be able to participate and benefit from task-oriented locomotor training, only patients with a Barthel ambulation subscore (BAMB) of at least 10 and a gait speed between 10 and 60 cm/s at entry were included. The exclusion criteria were stroke due to cerebral and subarachnoid hemorrhage; major medical problems such as heart conditions, diabetes, and cancer apt to interfere with the rehabilitation process; receptive and/or expressive aphasia; and musculoskeletal disorders of the lower extremities affecting walking capacity. The ethics committees of both institutions approved the study protocol, and all patients who agreed to participate signed an informed consent.

Experimental Design

The design was a single-blind, randomized, stratified controlled trial. Patients were stratified for 3 factors: 1) time since stroke; those entering the program < 45 days poststroke and those entering 46 to 90 days poststroke; 2) level of disability, Fugl-Meyer leg subscore (FM-L) < 25 at baseline and those ≥ 25; and 3) location: rehabilitation center or extended care hospital. Given the changes in health care in general and particularly with changing practices for referral of patients after a stroke for rehabilitation in-service therapy, patient accrual proved to be difficult throughout the study, especially in the later phase. The tendency to reduce the length of the institutional stay made it difficult to keep the patients for 8 weeks, the usual length of stay at study initiation. The prolonged duration of the study also led to changing PT practices in the type of therapy provided to the CTL group, and the project was terminated to avoid restricting the use of a treadmill and other devices given the evolution of clinical practice.

Allocation to Treatment Regimens

Patients meeting the inclusion criteria were seen by a study physician who evaluated the patient's neurological status and determined if the patient was medically capable of participating in the project. Patients then underwent further evaluations by independent evaluators who were physiotherapists from rehabilitation establishments other than the one in which the patient was treated. The patients were then stratified and randomly assigned to one of two treatment groups using a randomization scheme of randomly assigned block sizes (4,6,8) within each stratum. The treating therapists were then informed of the group assignment.

TREATMENT REGIMENS

All patients received speech and occupational therapy as indicated. Only PT treatments were controlled. The intensity (1 h per day, 5 days per week) and duration (2 months when possible) were similar in both groups, but the content and approach differed. In both rehabilitation units, a team of 3 to 5 experienced physical therapists provided the treatments. Therapists were randomly assigned to treat patients in one of the groups exclusively to limit contamination. To further reduce contamination, participants in the EXP group were treated in a separate area out of view of the participants in the CTL group. Treatment of the upper extremity was similar in both groups.

The participants in the CTL group received conventional PT. At study initiation, the conventional approach used by the therapists who participated in the study was an amalgamation of several methods. Thus, elements of the traditional neurodevelopmental approach[9] were incorporated with a motor learning, task-oriented approach.[15] Influences from undergraduate students, postgraduate courses, and visiting lecturers led to increased use of the task-oriented approach over the course of the study. Locomotion in the CTL group was initiated as soon as possible with external support. Other locomotor activities such as stair-climbing, walking on inclined planes, and various transfers were gradually added to permit training in a variety of gait-related tasks. Participants in the CTL group did not, however, practice walking on a treadmill, or use an isokinetic device, or a limb-load monitor.

The patients in the EXP group received therapy similar to that described for the CTL group. In addition, patients received specialized locomotor training. This approach, including progressive steps, has been described in detail.[18] Briefly, it consists of intensive training using a tilt table, if needed to hold the patient upright; the use of a limb-load monitor to induce weight bearing on the affected side; reciprocal stepping on a Kinetron isokinetic device; and treadmill walking with full weight bearing. The goal was to promote gait re-learning through locomotor activities that were adapted to the individual level of motor recovery.

Therapy Logbooks

The physical therapists treating the patients in both groups kept detailed charts on the content of the therapy provided and the number of 5-min intervals devoted to each aspect to permit the calculation of total time devoted to the different components of the PT program.

Measurement of Patient Outcomes

The primary outcome measure, gait speed, was measured by recording the time needed to walk 5 m, 10 m, or 30 m according to the method suggested by Wade.[43] Time to walk a given distance, be it 5 m, 10 m, or 30 m at preferred speed with an aid or personal support if needed, has been shown to be a valid, reliable, and responsive measure that has been used in many studies involving stroke survivors.[43-45] Walking speed has been shown to be a robust indicator of walking capacity. It is positively correlated to motor recovery,[7,46] static muscle strength of the lower extremity,[47] and the size of the "push-off" plantarflexor moment[34] but negatively correlated to spasticity of the plantarflexors.[26] Faster walking speeds are also indicative of quality of the lower extremity movements.[3,48] Walking speed was measured clinically and in the gait laboratory (see below).

The walking test (5 m, 10 m, or 30 m) at baseline and thereafter was tailored to the patient's ability. At baseline, 4 patients required a cane, 36 a quadripod cane, and 5 a walker. The need for walking aids decreased at posttest, with 20 requiring only a cane, 17 a quadripod cane, and 3 a walker. At follow-up, of the remaining 51 patients, 15 used a cane and 11 a quadripod.

Secondary Outcome Measures

Functional performance was evaluated using the Fugl-Meyer (FM) assessment method[49] and the Timed Up and Go (TUG).[50] The FM scale has good measurement properties[49,51] and subscores such as the Fugl-Meyer leg (FM-L) and arm (FM-A) subscores have been used in many trials. The TUG measures the time a patient takes to complete the following tasks: rise from a chair, walk a distance of 3 m, turn around, return to the chair, and sit down. It is a simple test, quick and easy to administer, that challenges balance and mobility. Functional levels of independence were assessed with the Barthel Index,[52] which can be subdivided to measure discrete functions of self-care and mobility (BAMB).[53,54] This index has been widely used and has been shown to have high reliability and validity and moderate responsiveness[55] to changes in functional ability over time. The Balance Scale[56] was used to assess balance in the clinical setting. It has good measurement properties[57,58] and has been shown to be responsive to recovery in patients with hemiparesis.[6,58,59]

Clinical Evaluations

When possible, the same independent evaluator, who was blind to group assignment, assessed the patient at baseline, after 2 months of therapy (or discharge), and at 3 months after discharge from rehabilitation (to assess carryover or retention effects). The evaluator followed a standard protocol to obtain scores for the FM-L, FM-A, BAMB, Balance Scale, and TUG.

Laboratory Evaluations

A complete gait analysis was made at baseline and posttherapy in the Motor Evaluation Laboratory by means of a custom-built 2-dimensional video system[60] combined with footswitches, an embedded force plate, and specialized software.[61,62] Reflective markers were placed on body landmarks on the affected side of the body, and footswitches were taped to the soles of both shoes to demarcate periods of support and nonsupport (stance and swing phases of the gait cycle). Gait movements were recorded using a 2-dimensional video camera system (250×255 pixels, Panasonic, model WV-BD400, Secaucus, NJ, USA) that was connected to rails parallel to the walkway and a motorized device that allowed it to follow the participant to capture the gait movements, which were recorded simultaneously with the footfall patterns. Ground reaction forces were recorded with a force plate (AMTI model OR6-5-1000, Advanced Mechanical Technology, Inc., Watertown, MA, USA) embedded in the walkway. Participants walked at free speed without orthosis or walking aids, but manual support was provided by holding one or both arms if needed. Because participants were not instructed how to place their affected foot on the force plate, it was not always possible to capture the ground reaction forces, especially in the more affected participants. Records of gait movements were made over a 6 m central portion of the walkway. Movement, force, and footswitch signals were sent to an IBM-compatible computer for analysis by means of specialized software. This study reports only gait speed and the mechanical power produced by the ankle plantarflexor muscles at the end of the stance phase. Average gait speed was derived from the footswitch signals and the distance covered by the hip marker over 3 to 5 gait cycles. Joint moments were calculated using an inverse dynamic model combined with a link segment model, and ankle muscle power over the gait cycle was derived from the moment and joint angular values and output as ankle power curves over the gait cycle. Peak values for the A2 power burst were obtained from the computer output of the power curve. Because of the difficulty in recording force plate data, reliable baseline and posttest data were available in only half of the participants, and in many cases the power measures were often derived from a single gait cycle. Gait analyses were not made at follow-up.

Sample Size

The number of participants to be enrolled in the trial was determined using nQuery, version 3.0, and results from our pilot study.[7] The power analysis relied on the following specifications regarding the primary outcome measure, gait speed: type 1 error = 5%, standard error = 15 cm/s, speed at pretest = 25 cm/s; speed at posttest = 40 cm/s for the CTL group and 50 cm/s for the EXP group, pre-post correlation in speed measurements = 0.75, and power to detect a significant interaction effect = 80%. According to this power analysis, 24 participants per group must be available for analysis. Anticipating an attrition rate of 20%, the total number of participants to be enrolled was set at 60.

STATISTICAL ANALYSIS

Groups were compared at baseline using the *t* test for independent samples or its nonparametric equivalent the Mann-Whitney *U* test for the continuous variables. The chi-square and the Fisher exact test were used for categorical data, depending on the distribution of the data. An intention-to-treat analysis was carried out. Different methods were used to impute values for missing data. For most variables, the imputed value was derived from a linear regression equation defining the change between 2 evaluations of a given variable in participants with complete data. Because values between the posttest and follow-up for gait speed were poorly correlated, imputed values for gait speed were derived from regression equations describing the relation between gait speed and the FM-L subscore. The missing value for the BAMB subscore was derived by calculating the mean change and adding this value to baseline values to obtain the follow-up value. For 1 participant lacking both posttest and follow-up TUG scores, the mean group value was attributed. As all outcome variables were normally distributed, to test the main study hypothesis we chose ANOVAs with repeated measures with a between-subject factor at 2 levels (the 2 groups) and a within-subject factor at 3 levels (the time: baseline, posttraining, and follow-up), followed by post hoc Scheffé tests. The interaction of group and time served to determine the efficacy of the EXP program on the outcome measures. Analyses were done with and without imputed values, and results were similar.

Student *t* tests were used to compare the values for ankle power generation obtained at pre- and posttests and with values obtained by the same methods in healthy elderly participants walking at different walking speeds.[33,63] Lastly, a Pearson correlation coefficient was used to examine the relationship between the change in ankle power generation (impairment) and change in gait speed assessed in the laboratory. The α level was preset at 0.05. All analyses were performed with GB-Stat version 5.0. All reported *P* values are 2-sided.

RESULTS

The Study Sample

Of a total of 63 participants, 32 in the EXP group entered the study. Table 1 summarizes the characteristics of these participants. The groups were comparable on all characteristics at baseline ($P > 0.10$). Three participants, 2 in the EXP group (hip fracture and cardiac problems) and 1 in the CTL group, withdrew and did not complete the training period. Because baseline values were obtained for 1 of the 2 participants in the EXP group, and for the participant in the CTL group, these 2 participants were kept in the analysis and missing values were imputed. Twelve participants, 5 in the EXP and 7 in the CTL group, were unavailable for follow-up evaluations. The number of participants was main-

Table 1. Subject Characteristics

Group	Age (years)	Gender	Height (m)	Weight (kg)	Time Since CVA (days)	Affected Side
EXP (*n* = 32)	62.9 (12)	M = 22 F = 10	1.7 (0.1)	67.1 (11)	52.0 (22)	R = 17, L = 15
CTL (*n* = 31)	60.7 (12)	M = 21 F = 10	1.6 (0.1)	69.2 (12)	52.6 (18)	R = 11, L = 20

Values give mean (standard deviation); M = male, F = female, R = right, L = left.

tained at 31 per group at follow-up by imputing values (see Methods).

Adherence to Therapy Regimen and Details of the Components of the PT Regimens

Table 2 details adherence to the therapy protocol planned for 8 weeks and 40 therapy sessions. Actual therapy duration was close to 8 weeks on average in both groups. During 33.6 and 31.8 sessions, 36.3 and 39.1 h of PT was provided for patients in the EXP and CTL groups, respectively. The bar graphs in Figure 1 summarize the therapy-related information provided by the therapists. Thus, the 2 groups received a similar amount of passive movements, adjunct therapies and balance and mobility activities, whereas the CTL group practiced more upper limb activities ($P = 0.02$) and the EXP group more walking activities ($P = 0.001$). When the balance and mobility activities and the walking activities are taken together as locomotor-related activities, both groups received a similar ($P = 0.69$) amount of locomotor tsaining time (EXP: 27.9 ± 9.3 h; CTL: 26.9 ± 9.4 h). In the EXP group, patients used the treadmill a mean of 6.4 ± 1.9 h and the Kinetron 2.2 ± 1.4 h over the treatment period. Thus, patients used the treadmill an average of 11.5 ± 2.8 min and the Kinetron 3.9 ± 2.5 min per session, the sum of the 2 corresponding to 25.1% ± 7.8% (mean, *SD*) of the therapy time provided in all the sessions.

Effects of Therapy

Primary outcome measure. Figure 2 gives the means and standard deviations for walking speed obtained by the clinical and laboratory methods. Laboratory measures were not taken at follow-up, thus comparisons for the 3 time points relate to the clinical measures. The pattern of change with therapy is the same for both gait speed evaluation methods. The results of the clinical and laboratory evaluations provide complementary information and are thus both presented. Results from the ANOVA for gait speed revealed a main effect for time ($P < 0.01$) but not for group, and no time-by-group interaction, indicating that both groups significantly increased their gait speed over time and that the increase was similar in both groups. Post hoc analyses showed that the increase in speed was significant between baseline and posttherapy values ($P < 0.01$) but not between posttherapy and follow-up values. Further comparisons between clinical and laboratory measures of walking speed indicated that both methods measured similar values at baseline but not posttherapy, where the increase in speed was greater ($P < 0.01$) with the clinical method. For the clinical measures, posttest gait speeds were more than double baseline values ($P < 0.01$) and the mean change was 28 and 33 cm/s in the EXP and CTL groups, respectively.

Secondary outcome measure. Figure 3 presents the mean values for the secondary clinical outcomes at the different evaluations for the EXP (Figure 3, A-C) and CTL (Figure 3, D-F) groups. Values for all outcomes improved significantly between baseline and at posttests in both groups ($P < 0.01$). As for gait speed, results from the ANOVAs for secondary measures revealed a main effect for time ($P < 0.01$) but not for group, and there was no time-by-group interaction. Likewise, significant changes were found between baseline and posttherapy ($P < 0.01$) only, with slight increments ($P > 0.05$) between posttest and follow-up values, indicating retention of gains. As expected, measures of impairment, the FM-A ($P < 0.05$) and FM-L ($P < 0.01$), revealed small but significant increments of change, whereas measures of disability (BAMB, Balance Scale, and TUG) showed larger changes ($P < 0.01$).

Figure 4 compares the peak A2 push-off power generation burst at the end of the stance phase on the paretic limb of the persons after stroke in the present study, with values obtained with the same methodology in elderly (57 ± 8 years) healthy participants walking at normal (N: 126 ± 15 cm/s), slow (S: 91 ± 13 cm/s), and very slow (VS: 58 ± 11

Table 2. Compliance to the Planned Protocol of Physical Therapy (PT)

Group	Duration of Therapy (weeks)	Number of PT Sessions	Total Treatment Time (h)	Mean Duration of Each Session (h)
EXP (*n* = 30)	7.83 (1.1)	33.63 (5.7)	36.31 (10.8)	1.06 (0.2)
CTL (*n* = 30)	7.76 (1.07)	31.8 (5.07)	39.11 (12.3)	1.22 (0.3)

Three participants did not complete the therapy; values give mean (standard deviation).

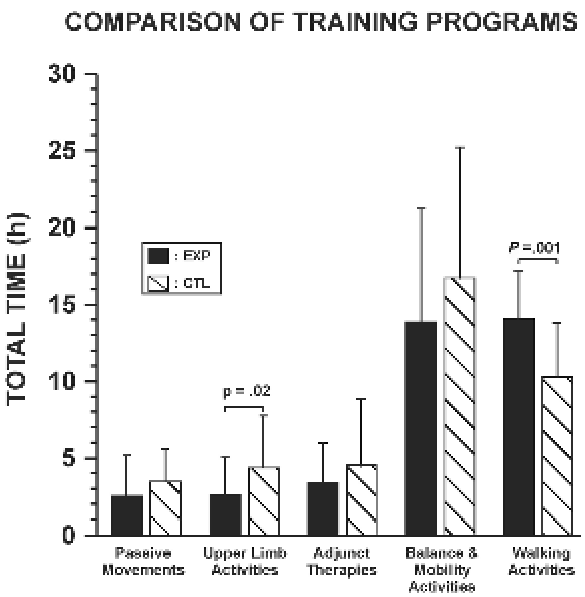

Figure 1. Time in hours (h) devoted to different components of the physical therapy (PT) training program during the total treatment period. Bar graphs give mean (+ 1 *SD*) values for the experimental (EXP) and control (CTL) groups derived from detailed records kept by the treating therapist. Adjunct therapies refer to the use of ice, heat, or electrical stimulation. Walking activities include stair climbing, walking up and down inclines, walking on different surfaces or outdoors, and avoiding obstacles.

cm/s) walking speeds.[35,62] At baseline, the participants in both the CTL and EXP groups generated about 50% of the power generated by the healthy group walking very slowly. After therapy, the peak power generation for both groups was in the same order of magnitude as that measured for the healthy participants at VS walking speed, indicating recovery in the capacity to produce muscle power for gait propulsion. This increase was significant within groups posttherapy ($P < 0.01$–0.05), but again, there was no difference in the increase between the groups ($P > 0.05$).

Figure 5 illustrates the relationship ($r = 0.52$; $r^2 = 0.27$; $P = 0.0034$, $n = 30$) between the change in the A2 power generation burst and the change in gait speed from the beginning to the end of therapy for the pooled participants. The r^2 value indicates that

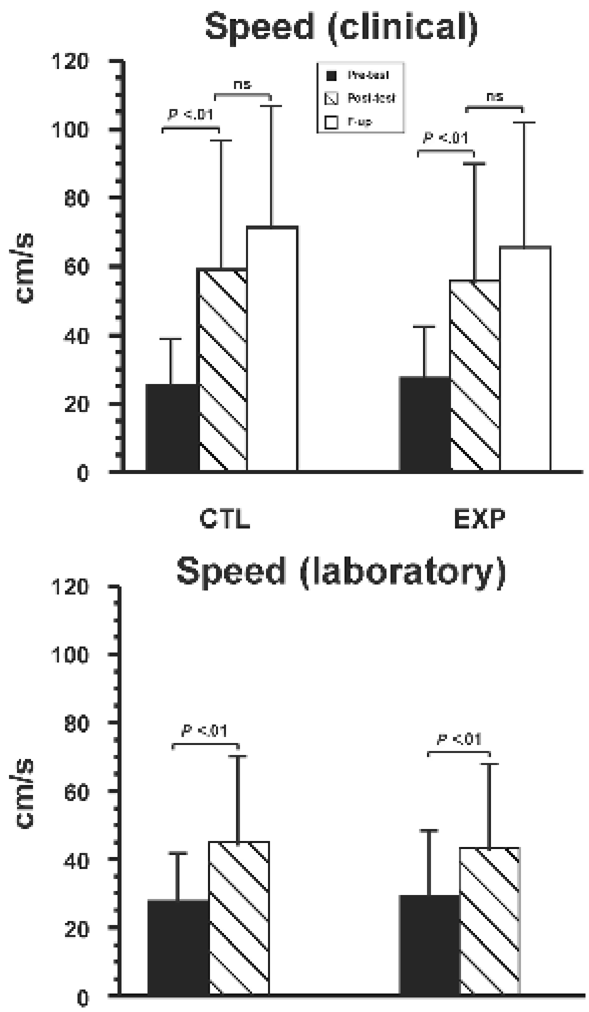

Figure 2. Comparison of gait speed in the control (CTL) and experimental (EXP) groups measured by the clinical (upper graph) and laboratory (lower graph) methods at the different evaluations: baseline (pretest), posttest, and follow-up (F-up). Values give mean + 1 *SD*. Follow-up evaluations were not made in the laboratory.

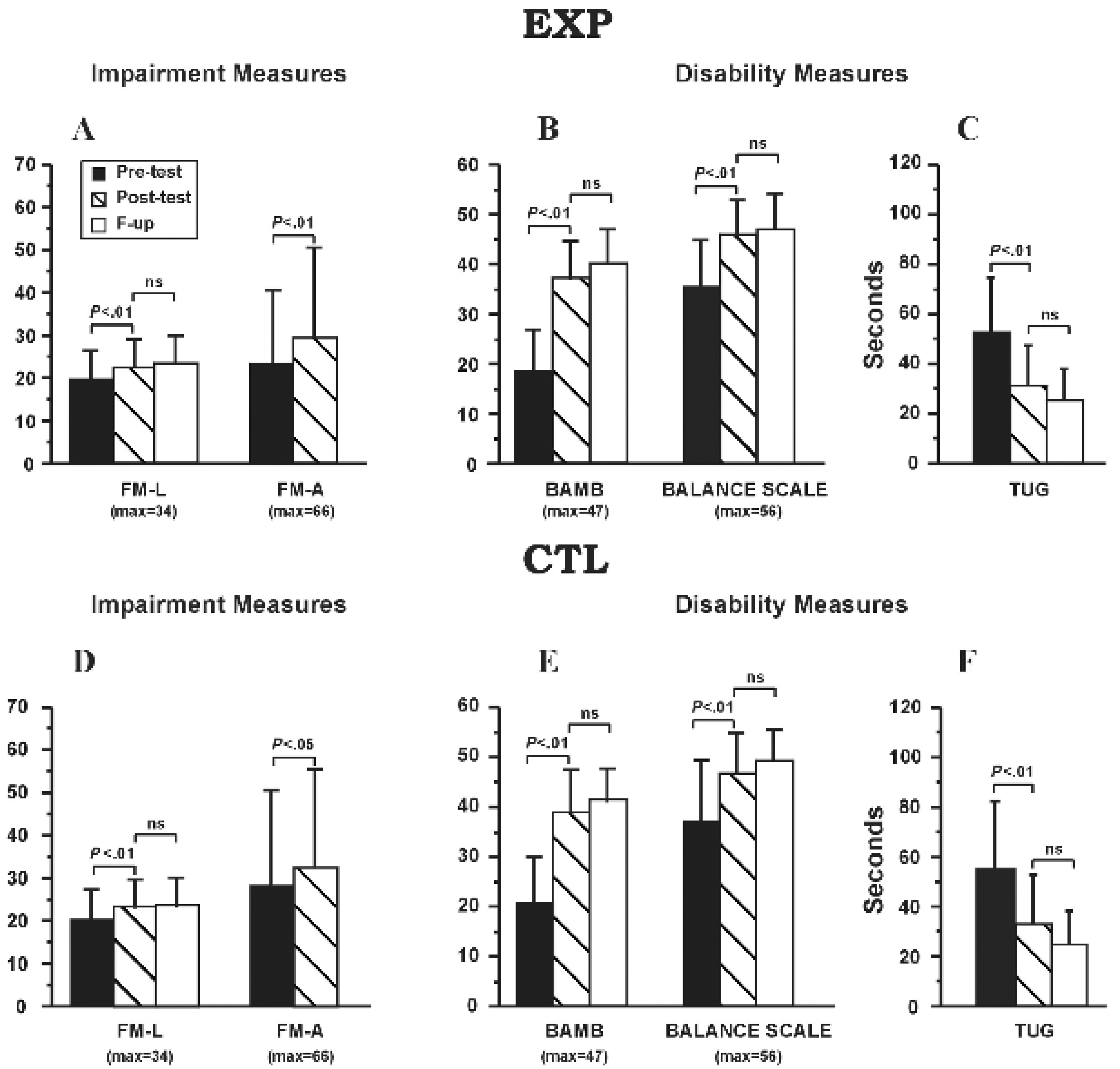

Figure 3. Comparison of values for the impairment and disability outcome measures at the 3 evaluations (pretest, posttest, and follow-up [F-up]) for the experimental (EXP:A-C) and the control (CTL:D-F) groups. The measures and maximum value for each measure are given from left to right along the *x*-axis: Fugl-Meyer Leg subscale (FM-L), Fugl-Meyer Arm subscale (FM-A), Barthel index ambulation subscore (BAMB), Balance Scale, and Timed Up and Go (TUG). Note that decreasing values in seconds to perform the task denotes improvement in TUG values. Vertical bars give mean + 1 *SD*. F-up values not recorded for FM-A.

27% of the improvement in gait speed is associated with recovery in the capacity to generate propulsive force at the ankle.

DISCUSSION

The main finding is that irrespective of the training mode, the persons in the subacute phase poststroke who received task-oriented physical therapy markedly increased their gait speed and maintained the gain over at least 3 months. The failure to detect a difference between the groups posttherapy in gait speed as well as in all the secondary measures demonstrates that the provision of task-oriented training is not limited by the availability of rehabilitation technology such as treadmills, Kinetron isokinetic dynamometers, or limb-

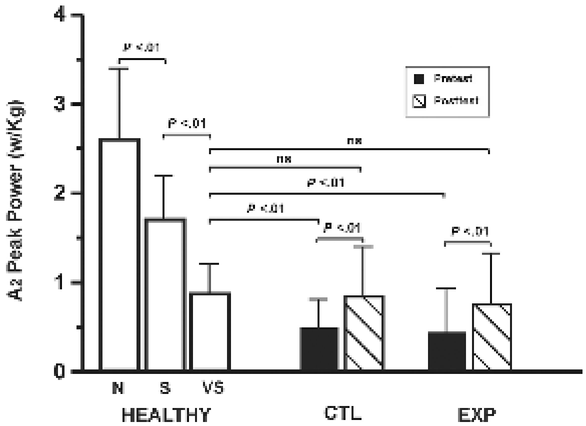

Figure 4. Comparison of values for peak ankle power generation obtained from the A2 power burst of the persons with stroke in the control (CTL) and experimental (EXP) groups in pre- and posttherapy tests with values obtained in healthy participants with a mean age of 57 ± 8 years walking at normal (N: 126 ± 15 cm/s), slow (S: 91 ± 13 cm/s), and very slow (VS: 58 ± 11 cm/s) speeds. Values give mean + 1 *SD*.

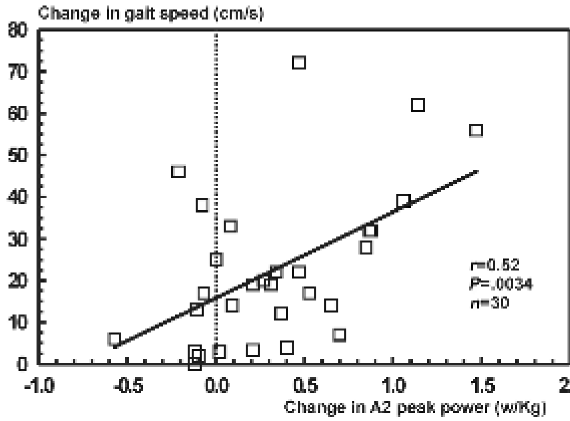

Figure 5. Relationship defined by a Pearson correlation coefficient between the change in gait speed (*y*-axis) and the change in the peak A2 power generation burst (*x*-axis) between baseline to posttest values for the experimental and control groups combined.

load monitors, but depends rather on successfully transferring the concepts of the task-oriented approach to the individual patient.

These results are in line with findings from at least 2 randomized, controlled clinical trials where training with or without rehabilitation technology was compared in persons with acute or subacute stroke. Nilsson et al.[20] in a trial comparing walking training on a treadmill with weight support and walking training on the ground concluded that the 2 approaches were comparable. In a large trial comparing computer-assisted biofeedback gait training with gait training provided by physical therapists without additional technology to retrain gait in early stroke, Olney et al.[38,64] also found the positive results comparable. Taken together, these 3 trials relay important messages. First, that it is the therapist who is the key to providing task-oriented training, and second, that sophisticated and often costly rehabilitation equipment is not essential to the provision of this therapy. This is not to say that such equipment is not to be recommended but rather that it is not essential to provide the therapy and that its use does not promise better results. This is an important finding for physical therapy departments of limited financial means, home-based therapy, and physical therapy in developing countries.

The magnitude of the gain in gait speed posttherapy in the present study in comparison to previous reports supports the efficacy of the task-oriented approach. The clinical measures revealed that the gait speed more than doubled after therapy and that this increase was maintained at follow-up. The magnitude of the gait speed increase posttherapy (28 and 33 cm/s in the EXP and CTL groups, respectively) is larger than that reported in other studies involving slightly more impaired,[20] similar, or less impaired participants,[19,65] as judged by reported Fugl-Meyer leg subscores at baseline. This large improvement in gait velocity is likely linked to the time spent during nearly 2 months practicing locomotor-related activities in both groups.[7] Detailed comparisons, however, of locomotor-related practice provided in other studies cannot be made because they do not give precise information on the time dedicated to individual therapy components. In the present study, we know that both groups practiced such activities about 27 of the total hours (EXP: 36.3 ± 10.8 h; CTL: 39.1 ± 12.3 h) of physical therapy, during almost 2 months within a therapy context wherein personal training time or activities of daily living were not monitored. Whether this amount of locomotor-training within a therapy context is enough to promote optimal recovery is unknown. One can also question whether the increment of speed gain at follow-up could have reached statistical significance if the in-patient locomotor-training had been longer or if they had been required to practice on their own.

In the present study, the lack of superiority of the technology-driven task-related approach in promoting recovery of gait speed was less surprising once the time dedicated to the different therapy components was broken down. Interestingly, the participants in the CTL group received more balance and mobility training but less actual walking practice. After discussions with the therapists, it was decided that the balance and mobility exercises could be considered locomotor preparatory tasks, and this incited us to combine the balance and mobility activities with walking activities. This revealed that both groups had received about 27 h of gait-related practice.

The task-oriented approach proposed by Carr and Shepherd[15,41] to promote locomotor recovery has gradually gained favor and has been the choice of approach in recent studies designed to evaluate other factors affecting recovery.[7,12,19,20,40,66,67] The results of the present study further support the use of this approach.

The results of the present study also provide behavioral evidence of recovery by demonstrating that persons in the subacute phase after stroke can improve the production of propulsive power for walking by employing a strategy similar to healthy participants walking at VS speed. Without a complete power analysis, we cannot affirm that many did not compensate by pulling-up with their hip flexors at the beginning of the swing phase.[34,35] Nevertheless, about 27% of the increase in walking speed is associated with augmented power of their plantarflexors at push-off on the affected side. This supports recovery of function and argues for the need to specifically train these muscles. Given the propulsive role of the hip extensors in early stance and the hip flexors in early swing, these muscles should also be targeted within the context of the task-oriented approach.[34-38] An unexpected result was the finding that at posttherapy the persons with stroke produced an A2 power burst comparable to that of healthy participants voluntarily walking very slowly. Unfortunately, we did not test the capacity of the persons with stroke to increase their walking speed beyond free speed, so we cannot establish a speed-related profile as for the healthy participants. The work of Olney et al.,[34] however, suggests that persons with stroke who walk faster do so with a larger A2 power burst than those who walk slowly, even when taking into consideration the adaptive role of shifting more power to the hip flexors in early swing.[37]

Only 2 other studies have, to our knowledge, examined the effects of PT on the A2 power burst. Teixeira-Salmela et al.[36] reported a near-doubling of the peak A2 burst on the affected side from 0.97 ± 0.63 to 1.71 ± 1.06 W/kg following 10 weeks of muscle strengthening and physical conditioning in a group of 13 chronic stroke survivors. This power increase was related to a mean increase in gait speed of 16 cm/s (60 to 76 cm/s). Interestingly, the size of the A2 power burst and mean walking speed for these chronic stroke survivors are in the same order of magnitude of these respective values for the subacute stroke survivors in the present study. On the other hand, Kim et al.[68] reported that 6 weeks of either voluntary or passive isokinetic knee exercises in chronic stroke participants resulted in little change in the capacity to produce ankle power. These results in chronic stroke survivors are encouraging and support the potential for recovery of the A2 burst with appropriate practice.[36,40] Although there is evidence that static muscle strength of the quadriceps and other lower extremity muscles is positively related to gait velocity[47,69] and that gait velocity is slightly increased after isokinetic strengthening of knee muscles in chronic stroke survivors,[70,71] there is no evidence that such training affects the A2 power burst. In fact, one can question the specific carry-over value of non-task-oriented muscle training, be it static or isokinetic training.

The explosion of interest brought on by the documented reorganization of the brain following the "recovery" of disturbed hand and arm movements has renewed interest in neurological rehabilitation.[17,72] More important, such studies have demonstrated the importance not only of task-specific practice but also of factors such as increasing task complexity and motivating factors to promote adequate brain reorganization and motor recovery. Changing cortical maps after forced use of the upper extremity of stroke survivors revealed by transcranial magnetic stimulation mapping studies[22] support the link between brain organization and use of the involved extremity. The recent fMRI-documented progressive changes in locomotor-related brain areas of chronic stroke survivors associated with a series of locomotor training periods on a treadmill[23] confirm the potential of appropriate practice to promote locomotor recovery.

Both laboratory and clinical measures of gait speed are reported, in part to illustrate the differences between the methods and also to provide comparative data with most studies that report only clinical measures of walking speed. Ideally, the independent evaluators would have evaluated gait speed over the same distance for all participants at all the time points. Because some participants had difficulty at baseline covering 10 m, they were evaluated over 5 m, and this led to the practice of evaluating the participants at posttests over a longer distance if they could manage. Because the participants in both groups were measured with the same criteria, this practice affected both groups in the same way. Because we had both clinical and laboratory data, we were able to quantify the notion that persons with stroke walk faster when unencumbered by laboratory apparatuses and constraints. At baseline, the walking speeds were similar, but at posttests both groups of participants walked about 12 cm/s faster when speed was measured clinically, representing a difference of about 27%. Assuming equal accuracy of the measures with both methods, behavioral factors have to be considered. One can argue that at baseline, the poorer walking capacity was captured by both methods. After therapy, walking capacity was improved and participants likely had better control over their performance. Some of the good walkers were also evaluated over a longer distance (30 m). Measuring gait speed over 5 or 10 m does not affect the recording of comfortable walking speed in the same participants with subacute stroke,[45] and it is unlikely that increasing the distance to 30 m affects the recorded speed. The likely explanation is that the clinical method of recording gait speed allows the participant to use, if needed, assistive devices and personal walking aids and provides more freedom to complete the task unencumbered and away from the need to perform for the camera and laboratory personnel.

This study also provided important indicators of change in the secondary clinical measures. The FM-L and FM-A subscores describe a typical population of subacute persons with stroke referred for in-service rehabilitation. The small yet significant changes in the FM-L and FM-A subscores give indicators of the size of the change scores after 8 weeks of therapy. The relatively small change in the FM-L subscore was related to a more than doubling of the clinical measure of gait speed, indicating the nonlinear relation between the impairment and disability measure, wherein gait speed changes little until the FM-L subscore reaches about 25 and then increases rapidly with increasing FM-L scores from 26 to 34.[7] This nonlinear relationship can be expected when clinical measures that have ceiling effects are compared to quantitative measures such as gait speed[6] and bring into question the sensitivity of clinical measures to change when performance approaches healthy values. The baseline and change scores with therapy for the BAMB, Balance Scale, and TUG also provide data on the performance of a population of subacute stroke survivors. The similarity in scores at baseline and posttherapy for the 3 measures between the groups was remarkable. This similarity was not a function of the type of scale, in that the ordinal and timed measure gave similar results. Analysis of the relative merits of each measure was not, however, the subject of the present article. Rather, it was to demonstrate the importance of the therapist in designing and executing physical therapy programs to promote locomotor recovery for persons after stroke.

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REFERENCES

1. Mayo NE, Neville D, Kirkland S, et al. Hospitalization and case-fatality rates for stroke in Canada from 1982 through 1991. The Canadian Collaborative Study Group of Stroke Hospitalizations. *Stroke* 1996;27:1215-20.
2. Skilbeck CE, Wade DT, Hewer RL, Wood VA. Recovery after stroke. *J Neurol Neurosurg Psychiatry* 1983;46:5-8.
3. Wade DT, Hewer RL. Functional abilities after stroke: measurement, natural history and prognosis. *J Neurol Neurosurg Psychiatry* 1987;50:177-82.
4. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients. The Copenhagen Stroke Study. *Arch Phys Med Rehabil* 1995;76:27-32.
5. Friedman PJ. Gait recovery after hemiplegic stroke. *Int Disabil Stud* 1990;12:119-22.
6. Richards CL, Malouin F, Dumas F, Wood-Dauphinee S. The relationship of gait speed to clinical measures of function and muscle activations during recovery post-stroke. In: *Proceedings of the Second North American Congress of Biomechanics*; 1992:299-302.
7. Richards CL, Malouin F, Wood-Dauphinee S, Williams JI, Bouchard JP, Brunet D. Task-specific physical therapy for optimization of gait recovery in acute stroke patients. *Arch Phys Med Rehabil* 1993;74:612-20.
8. Duncan PW, Goldstein LB, Horner RD, Landsman PB, Samsa GP, Matchar DB. Similar motor recovery of upper and lower extremities after stroke. *Stroke* 1994;25:1181-8.
9. Bobath B. *Adult hemiplegia: evaluation and treatment*. London: Heinemann Medical Books; 1970.
10. Brunnstrom S. *Movement therapy in hemiplegia*. New York: Harper & Row; 1970.
11. Knott M, Voss D. *Proprioceptive neuromuscular facilitation*. New York: Harper & Row; 1968.
12. Kwakkel G, Wagenaar RC, Twisk JW, Lankhorst GJ, Koetsier JC. Intensity of leg and arm training after primary middle-cerebral-artery stroke: a randomised trial. *Lancet* 1999;354:191-6.
13. Teasell RW, Foley NC, Bhogal SK, Speechley MR. An evidence-based review of stroke rehabilitation. *Topics Stroke Rehabil* 2003;10:29-58.

14. Gordon J. Assumptions underlying physical therapy intervention: theoretical and historical perspectives. In: Carr JH et al., eds. *Movement science: foundations for physical therapy in rehabilitation*. Rockville, MD: Aspen; 1987:1-30.
15. Carr JH, Shepherd R. *Movement science: foundations for physical therapy in rehabilitation*. Rockville, MD: Aspen; 1987.
16. Horak F. Assumptions underlying motor control for neurologic rehabilitation. In: Lister MJ, ed. *Contemporary management of motor problems: proceedings of the II step conference*. Alexandria, VA: Foundation for Physical Therapy; 1991:11-7.
17. Nudo RJ, Plautz EJ, Frost SB. Role of adaptive plasticity in recovery of function after damage to motor cortex. *Muscle Nerve* 2001;24:1000-19.
18. Malouin F, Potvin M, Prevost J, Richards CL, Wood-Dauphinee S. Use of an intensive task-oriented gait training program in a series of patients with acute cerebrovascular accidents. *Phys Ther* 1992;72:781-9; discussion 789-93.
19. Duncan P, Richards L, Wallace D, et al. A randomized, controlled pilot study of a home-based exercise program for individuals with mild and moderate stroke. *Stroke* 1998;29:2055-60.
20. Nilsson L, Carlsson J, Danielsson A, et al. Walking training of patients with hemiparesis at an early stage after stroke: a comparison of walking training on a treadmill with body weight support and walking training on the ground. *Clin Rehabil* 2001;15:15-27.
21. Nudo RJ, Plautz EJ, Milliken GW. Adaptive plasticity in primate motor cortex as a consequence of behavioral experience and neuronal injury. *Semin Neurosci* 1997;9:13-23.
22. Liepert J, Graef S, Uhde I, Leidner O, Weiller C. Training-induced changes of motor cortex representations in stroke patients. *Acta Neurol Scand* 2000;101:321-6.
23. Sullivan KJ, Dobkin BH, Tavakol M, Davis B, Knowlton BJ, Harkema S. Post-stroke cortical plasticity induced by step training. *Proc Soc Neurosci* 2001;27.
24. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979;102:405-30.
25. Shiavi R, Bugle HJ, Limbird T. Electromyographic gait assessment, Part 2: Preliminary assessment of hemiparetic synergy patterns. *J Rehabil Res Dev* 1987;24:24-30.
26. Lamontagne A, Malouin F, Richards CL. Locomotor-specific measure of spasticity of plantarflexor muscles after stroke. *Arch Phys Med Rehabil* 2001;82:1696-704.
27. Lamontagne A, Richards CL, Malouin F. Mechanisms of disturbed motor control in ankle weakness during gait early after stroke. *Gait Posture* 2002;15:244-55.
28. Dietz V, Quintern J, Berger W. Electrophysiological studies of gait in spasticity and rigidity. *Brain* 1981;104:431-49.
29. Malouin F, Doyon J, Dumas F, Jackson PL, Evans AC, Richards CL. Mental representation of locomotion: a PET study. *Neuroimage* 1997;5:132.
30. Lamontagne A, Malouin F, Richards CL. Contribution of passive stiffness to ankle plantarflexor moment during gait after stroke. *Arch Phys Med Rehabil* 2000;81:351-8.
31. Bogårdh E, Richards CL. Gait analysis and relearning of gait control in hemiplegic patients. *Physiother Can* 1981;33:223-30.
32. Knutsson E. Analysis of gait and isokinetic movements for evaluation of antispastic drugs or physical therapies. *Adv Neurol* 1983;39:1013-34.
33. Richards CL, Malouin F, Dean C. Gait in stroke: assessment and rehabilitation. In: Duncan PW, ed. *Clinics in geriatric medicine*. vol. 15. New York: W. B. Saunders; 1999:833-55.
34. Olney SJ, Griffin MP, Monga TN, McBride ID. Work and power in gait of stroke patients. *Arch Phys Med Rehabil* 1991;72:309-14.
35. Richards CL, Malouin F, Dumas F, Lamontagne A. Recovery of ankle and hip power during walking after stroke. *Can J Rehabil* 1998;11:271-3.
36. Teixeira-Salmela LF, Nadeau S, McBride I, Olney SJ. Effects of muscle strengthening and physical conditioning training on temporal, kinematic and kinetic variables during gait in chronic stroke survivors. *J Rehabil Med* 2001;33:53-60.
37. Olney SJ, Richards CL. Hemiplegic gait following stroke: Part I. Characteristics. *Gait Posture* 1996;4:136-48.
38. Olney SJ, Nymark J, Zee B, Martin C, Brown S. Effects of BioTrac computer-assisted feedback for gait rehabilitation in subjects with stroke: interim analysis of RTC. *Proc World Confed Phys Ther* 1995;0771.
39. Olney SJ, Griffin MP, McBride ID. Multivariate examination of data from gait analysis of persons with stroke. *Phys Ther* 1998;8:814-28.
40. Dean CM, Richards CL, Malouin F. Task-related training improves performance of locomotor tasks in chronic stroke. A randomized, controlled pilot study. *Arch Phys Med Rehabil* 2000;81:409-17.
41. Carr J, Shepherd R. *Neurological rehabilitation. Optimizing motor performance*. Boston: Butterworth Heinemann; 1998.
42. Richards CL, Malouin F, Bravo G, Dumas F, Wood-Dauphinee S. The role of technology in task-oriented locomotor training in acute stroke: a randomized controlled trial. *Proc World Confed Phys Ther* 2003;1592.
43. Wade DT. *Measurement in neurological rehabilitation*. Oxford, UK: Oxford University Press; 1992.
44. Richards CL, Olney SJ. Hemiparetic gait following stroke. Part II: recovery and physical therapy. *Gait Posture* 1996;4:149-62.
45. Salbach NM, Mayo NE, Higgins J, Ahmed S, Finch LE, Richards CL. Responsiveness and predictability of gait speed and other disability measures in acute stroke. *Arch Phys Med Rehabil* 2001;88:1204-12.
46. Brandstater ME, de Bruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. *Arch Phys Med Rehabil* 1983;64:583-7.
47. Bohannon RW. Decreased isometric knee flexion torque with hip extension in hemiparetic patients. *Phys Ther* 1986;66:521-3.
48. Richards CL, Malouin F, Dumas F, Tardif D. Gait velocity as an outcome measure of locomotor recovery after stroke. In: Craik RL, Oatis CA, eds. *Gait analysis: theory and application*. St Louis, MO: Mosby; 1995;25:355-64.
49. Fugl-Meyer AR, Jaaskp L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegia patient: I. A method for evaluation of physical performance. *Scand J Rehabil Med* 1975;7:13-31.
50. Podsiadlo D, Richardson S. The timed "up & go": a test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc* 1991;39:142-9.
51. Kusoffsky A, Wadell I, Nilsson BY. The relationship between sensory impairment and motor recovery in hemiplegia. *Scand J Rehabil Med* 1982;14:27-32.
52. Mahoney FD, Barthel DW. Rehabilitation of the hemiplegic patient: a clinical evaluation. *Arch Phys Med Rehabil* 1954;35:359-62.
53. Granger CV, Greer DS, Liset E, Coulombe J, O'Brien E. Measurement of outcomes of care for stroke patients. *Stroke* 1975;6:34-41.
54. Granger CV, Dewis LS, Peters NC, Sherwood CC, Barrett JE. Stroke rehabilitation: analysis of repeated Barthel Index measures. *Arch Phys Med Rehabil* 1979;60:14-7.
55. Gresham GE, Phillips TF, Labi MLC. ADL status in stroke: relative merits of three standard indexes. *Arch Phys Med Rehabil* 1980;61:355-8.

56. Berg K, Wood-Dauphinee S, Williams JI, et al. Measuring balance in the elderly: preliminary development of an instrument. *Physiother Can* 1989;41:304.
57. Berg K, Wood-Dauphinee S, Williams JI, et al. Measuring balance in the elderly: validation of an instrument. *Can J Pub Health* 1992;83(Suppl 2):57-61.
58. Berg K, Wood-Dauphinee S, Williams JI. The Balance Scale: reliability assessment with elderly residents and patients with acute stroke. *Scand J Rehabil Med* 1995;27:27-36.
59. Wood-Dauphinee S, Berg K, Bravo G, Williams JI. The Balance Scale: responding to clinically meaningful changes. *Can J Rehabil* 1996;10:35-50.
60. Boivin D, Laurendeau D. A computer vision based apparatus for the measurement of planar movement: an application in physiotherapy. *Proc Vision Interface Conf* 1989:1-15.
61. Moffet H, Comeau F, Richards CL, Tardif D. A new gait analysis system using a binary image processor: intra and interrater reliability of kinetic and kinematic measurements. *Proc 6th Biennial Conf Can Soc Biomechan*; 1990;179-80.
62. Lamontagne A, Richards CL, Malouin F. Coactivation during gait as an adaptive behaviour after stroke. *J Electromyogr Kinesiol* 2000;10:407-15.
63. Richards CL, Dumas F, Malouin F. Hip and ankle strategies during walking after stroke. *Proc Soc Neurosci* 2001;831.6.
64. Olney SJ, Nymark JR, Zee B. Computer-assisted feedback and gait re-education in stroke patients. *Can J Rehabil* 1993;7:31-3.
65. Visintin M, Barbeau H, Korner-Bitensky N, Mayo NE. A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. *Stroke* 1998;29:1122-8.
66. Kwakkel G, Wagenaar RC, Twisk JW, Lankhorst GJ, Koetsier JC. Intensity of leg and arm training after primary middle-cerebral-artery stroke: a randomised trial. *Lancet* 1999;354:191-6.
67. Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Muscle strengthening and physical conditioning to reduce impairment and disability in chronic stroke survivors. *Arch Phys Med Rehabil* 1999;80:1211-18.
68. Kim CM, Eng JJ, MacIntyre DL, Dawson AS. Effects of isokinetic strength training on walking in persons with stroke: a double-blind controlled pilot study. *J Stroke Cerebrovasc Dis* 2001;10:265-73.
69. Andrews AW, Bohannon RW. Distribution of muscle strength impairments following stroke. *Clin Rehabil* 2000;14:79-87.
70. Engardt M, Knutsson E, Jonsson M, Sternhag M. Dynamic muscle strength training in stroke patients: effects on knee extension torque, EMG activity and motor function. *Arch Phys Med Rehabil* 1995;76:419-25.
71. Sharp SA, Brouwer BJ. Isokinetic strength training of the hemiparetic knee: effects on function and spasticity. *Arch Phys Med Rehabil* 1997;78:1231-6.
72. Byl N, Roderick J, Mohamed O, et al. Effectiveness of sensory and motor rehabilitation of the upper limb following the principles of neuroplasticity: patients stable poststroke. *Neurorehabil Neural Repair* 2003;17:176-91.