Gait in Stroke: Assessment and Rehabilitation

Article *in* Clinics in Geriatric Medicine · December 1999

DOI: 10.1016/S0749-0690(18)30034-X · Source: PubMed

3 authors:

Carol L Richards
Laval University

Francine Malouin
Laval University

Catherine Mary Dean
Macquarie University

GAIT IN STROKE

Assessment and Rehabilitation

Carol L. Richards, PhD, PT, Francine Malouin, PhD, PT, and Catherine Dean, PhD, PT

This article concerns the assessment and therapy of walking problems after stroke. Information on the frequency of walking disorders the rate of recovery, and the nature of the walking disability are discussed briefly. This is followed by an analysis of selected biomechanical and clinical measures used to assess the locomotor performance. The need to assess performance of ever more challenging locomotor tasks in different environments is emphasized. This article ends with suggestions derived from clinical, biomechanical, and motor-learning research for optimizing performance after stroke.

FREQUENCY OF A WALKING DISABILITY AFTER STROKE AND RATE OF RECOVERY

The results of Jørgensen et al[38] in a large population of 804 subjects illustrate the high frequency of a walking disability and its rate of recovery

This work was supported by grants from the National Health Research and Development Program of Canada and the "Fonds de recherche en santé du Québec." Dr Catherine Dean was the recipient of a postdoctoral fellowship from the "Fonds en réadaptation de l'Université Laval."

From the Department of Rehabilitation, Faculty of Medicine, Laval University, and Rehabilitation Research Group, Rehabilitation Institute of Quebec, Canada (CLR, FN); and the School of Physiotherapy, Faculty of Health Sciences, University of Sydney, Sydney, Australia (CD)

after stroke. They assessed walking function weekly until death or discharge from rehabilitation using a modified Barthel subscore: no walking function (0–5 points), able to walk with assistance (10 points), or able to walk independently (15 points). The time of best walking function was defined as the time at which a patient's walking function no longer improved. At 1 week after stroke, 51% of the subjects were unable to walk, 12% required assistance, and 37% were independent walkers. Within 5 weeks after stroke, 80%, and by 11 weeks, 90%, respectively, of the patients scoring fewer than 15 points at baseline had reached best walking function. These results on the frequency of the presence of a walking disability and its rapid rate of recovery in the first 5 weeks after stroke in most patients are in general agreement with a large number of studies that have described recovery of impairments and disabilities after stroke on the basis of clinical measures.[28,66,77,82] As clinicians suspect, the recovery of walking capacity is not over within 3 months and has been shown in a longitudinal study with gait speed as the outcome measure to continue up to 2 years after stroke.[66,70] This finding raises the important issue of choice of outcome measure, which is considered in a later section.

THE NATURE OF THE WALKING DISABILITY AFTER STROKE

Movements of the trunk and extremities during walking can be viewed as the accomplishment of four related tasks: (1) maintaining the balance of the head, arms, and trunk, which constitute about 70% of the body mass,[94] on two hip joints; (2) transfer and support of the body mass on one limb during the stance phase; (3) floor clearance of swinging foot during the swing phase; and (4) supplying propulsive force for forward progression.[57] These tasks, accomplished with energy conservation measures in healthy subjects, challenge an impaired locomotor control system. Normal walking is characterized by a smooth succession of steps with first one leg and then the other. The time taken for each step is similar, as is the distance covered with each step during forward progression. The gait cycle consists of two steps, as body weight is accepted and transferred over first one foot and then the other during forward progression. A gait cycle thus includes a stance and a swing phase and two periods of double support at the beginning and end of the stance phase as the weight is transferred from one leg to the other.

The pathophysiologic basis of the locomotor disorder is damage to neurons and pathways in the central nervous system caused by interruption of the arterial blood supply because of thrombus or hemorrhage on one side of the brain. Two types of impaired motor control, which appear immediately after stroke, particularly affect locomotor performance. These are weakness or loss of volitional movement of the arm and leg on the side opposite the brain lesion, known as *paresis*,[40,68,71] and inappropriately timed or graded muscle activations.[3,40] Other types that appear later include hyperactive stretch reflexes[40,75] and hypoextensibility of the muscle–tendon complex.[22,37,49,80] Because damage to the motor control system varies with the nature and extent of the brain lesion, the locomotor disorder is specific to individual subjects, although general principles can be derived to guide therapy. Knutsson and Richards[40] demonstrated how each of three types of disturbed motor control—hyperactive stretch reflexes, paresis, and coactivation of agonist and antagonist muscles in synergistic patterns—could be the crucial factor in the disturbed control of individual patients with chronic stroke and related the type of disturbed control to the choice of therapy. For example, subjects with a mainly paretic type of disorder would not be expected to benefit from antispastic medication.

Because walking is bipedal, the primary motor-control disorder on one side of the body disrupts not only the movements of the affected lower extremity but also the movements of the trunk and other extremity. In terms of tasks, the acceptance and transfer of weight on the paretic lower extremity is a major challenge. Will the muscles be strong enough to maintain the positions of the lower extremity joints without collapsing? Supporting the body weight in the stance phase on one limb implies sufficient strength in the hip and knee extensor muscles and the ankle plantarflexors.[95] Will spasticity of the calf muscles prevent the forward rotation of the leg on the foot and lead to knee hyperextension?[40,65] Will paresis in the knee extensors and ankle plantarflexors lead to the choice of knee hyperextension for stability and hip hiking for foot clearance in the swing phase?[40,65] The disturbed motor control and subsequent movement disorders are associated with reduced gait speeds accompanied by changes in the timing and distance covered by each step so that subjects with hemiparesis tend to take short uneven steps and to spend a longer time in stance on the nonparetic side, especially early after stroke. For a review of studies reporting spatiotemporal characteristics or joint-movement profiles during gait in subjects with hemiparesis see Olney and Richards.[57]

The understanding of the gait-movement disorders associated with stroke has advanced markedly in the past decade with the assistance of computer-driven analyses that allow for the measurement of moments of force (a *moment* is defined as the turning effect of a force about any point) and powers (the power generated by a moment is the product of the moment acting at an axis and the angular velocity of the rigid body about that axis).[59,91] Kinetic data derived from such analyses have helped interpret the effects of the disturbed motor control on the gait-movement pattern. The reader is referred to Winter[94] for details on the equipment, data, and formulae required to calculate joint moments and powers. Moments of force are the net result of muscular, ligament, and friction forces acting to alter the angular rotation of a joint. Because in normal walking the joints do not reach their extreme positions and friction forces are minimal, the net moment can be interpreted as resulting from muscle forces,[57,94] although this may not be the case in subjects with increased passive stiffness. Also, if excessive coactivation of antagonist muscles is present, concomitant records of the muscle activations are needed to reveal the opposing muscle forces producing the net moment.

Moment of force and power profiles over the gait cycle help understand *why* a subject walks with a given movement pattern. The method of calculation of muscle powers is such that power generation (above zero on the y-axis of the curves) indicates that the power burst is achieved by a concentric contraction (at least for muscles crossing a single joint), thus further helping understanding of the force deficit. The propulsive force for forward progression is said to come mainly from the capacity to generate power at the ankle for *push-off* (about 80%) and to assist swing-phase initiation by generating a flexion force at the hip (*pull-off*) in late stance and early swing[57,94]. Figure 1 compares the movement, moment of force, and power profiles at the hip, knee, and ankle during gait of two patients with hemiparesis to values obtained in normal subjects. At the hip, the H1 power-generation burst is related to a concentric contraction of the hip extensors in early stance and the H3 burst (related to pull-off) to a concentric contraction of the hip flexors; at the ankle the A2 burst (related to push-off) is the main power-generation burst. The movement profiles indicate that the patient who walks very slowly (15 cm/s) maintains a flexed hip, knee, and ankle position throughout most of the gait cycle. This flexed

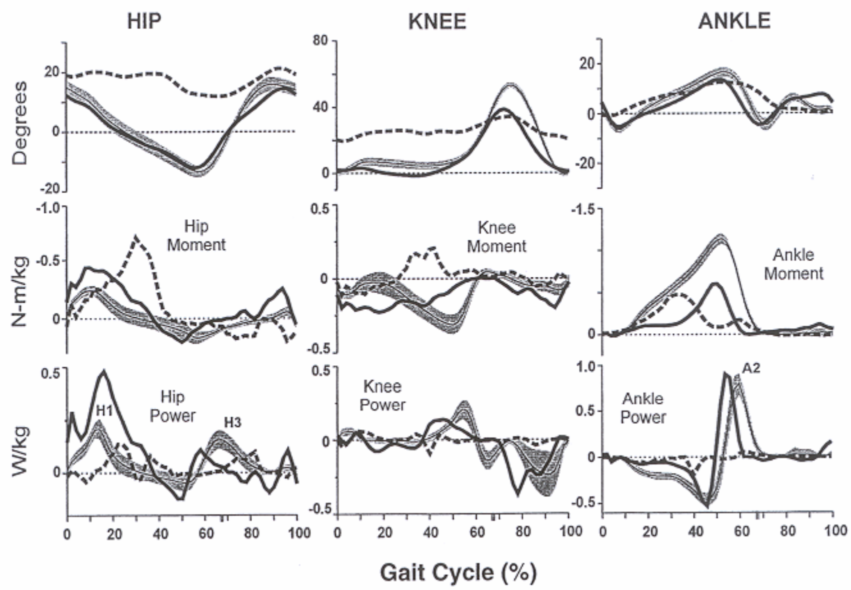

Figure 1. Comparison of the affected limb movement, moment, and power profiles over the gait cycle of two hemiparetic patients with values obtained in very slow-walking healthy subjects (*narrow solid line*) (58 ± 11 cm/s; mean ± 2 SE, n = 30). One patient, a 49-year-old man (*dashed line*) with a left-sided hemiparesis seen 67 days poststroke, had a Fugl-Meyer leg subscore of 19, a Barthel ambulation score of 25, and walked at a speed of 15 cm/s. The second patient, a 68-year-old man with left-sided hemiparesis (*heavy solid line*) seen 56 days after onset, had a Fugl-Meyer subscore of 33, a Barthel ambulation subscore of 36, and walked at a speed of 57 cm/s.

position is maintained by high extensor moments at the hip (20%–40% of the gait cycle) and knee (25% to 55% of the gait cycle) and a low or absent plantarflexor moment beyond 35% of the gait cycle. The moments and angular velocities resulted in low H1 and H3 power bursts and a very small A2 burst related to push-off at the end of stance. The subject who walks at a speed of 57 cm/s, in contrast, has movement profiles much closer to very slow normal values, and these are associated with a good A2 power burst at the ankle, a high H1 burst, and an H3 burst that approaches normal values but is not sustained.

A positive relation between the size of the A2 power burst and gait speed seen in Figure 1 is in accordance with the work of Olney et al.[59] It also has been suggested that an increased H3 pull-off power burst compensates for poor propulsive force at the ankle.[57,59] Richards et al[68] found the amplitude of the H3 (Spearman correlation coefficient = 0.62, $P < .01$, $n = 19$) and A2 ($r = 0.85$, $P < .01$, $n = 19$) power bursts in patients early after stroke (1.4 ± 0.5 months) to be significantly related to gait speed after 2 months of therapy. They also reported significant improvements in both the A2 and H3 power bursts with therapy. The capacity for improvement of both the A2 and H3 power bursts has been confirmed recently after 3 weeks of intensive task-specific training in chronic stroke patients.[20]

GAIT SPEED AS AN OUTCOME MEASURE

Although gait analyses that include kinetic measures help explain why subjects walk faster, such evaluations are not practical and are too expensive in clinical practice. For this and other reasons, gait speed has become the outcome measure of choice for clinical interventions. The measurement of gait speed has been shown to be valid, reliable, and responsive to change in locomotor performance (for reviews see Wade[86] and Richards and Olney[66]), to be positively related to strength of the muscles of the paretic leg,[9,68] to the A2 power burst at push-off,[59,68] to clinical measures of recovery of the lower extremity,[10,69] and to the quality of the gait movements,[57,67,88] and inversely related to the degree of spasticity in the plantarflexor muscles,[41] but not related to spasticity in the muscles around the knee.[53] Gait speed has also been shown to vary with the nature and tone of the instructions provided,[29] emphasizing the importance of consistent instructions.

As mentioned previously, gait speed is much reduced in subjects with hemiparesis, especially in the acute phase. Figure 2 illustrates the frequency of gait speeds in a group of 44 subjects before (50.5 ± 19 days poststroke; age range 38–89 years) and after 2 months of therapy in a rehabilitation center. Mean speed at baseline was 30 ± 18 cm/s. As can be seen in Figure 2, 39 subjects walked at speeds less than 50 cm/s. After therapy, the profile shifted to the right with 19 subjects walking faster than 50 cm/s. Gait speed did not improve systematically in all subjects, however, as indicated in Figure 3. Fifteen of the 44 subjects had less than

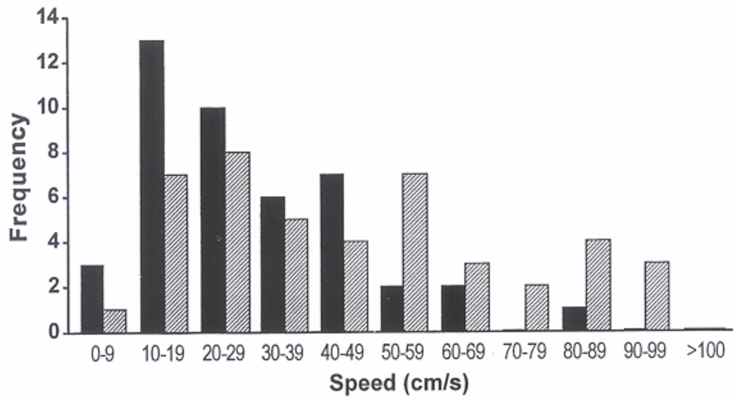

Figure 2. Frequency of gait speeds measured in the laboratory in a group ($n = 44$) of patients with hemiparesis at baseline (50.5 ± 19 days post stroke; mean ± 1 SD) and after 2 months of in-patient rehabilitation. Mean gait speed at baseline was 30.4 ± 18 cm/s and 46 ± 26 cm/s after therapy. Solid bar = baseline; hatched bar = posttherapy.

5 cm/s improvement and 27 less than 15 cm/s. Three subjects, however, improved their gait speed 55 to 64 cm/s. The mean speed after therapy was 46 ± 26 cm/s and the mean change was 16 ± 17 cm/s. A Pearson correlation coefficient of $r = 0.75$ further shows that gait speed at baseline predicts about 56% of the variance associated with the gait speed after therapy. To be unable to walk faster than 50 cm/s has a major impact on lifestyle. For comparative purposes, healthy elderly persons usually walk at speeds greater than 120 cm/s. Perry et al[62] examined the relationships among impairment, disability, and handicap in 147 subjects with stroke. They found that a gait speed of 58 ± 18 cm/s allows for the performance

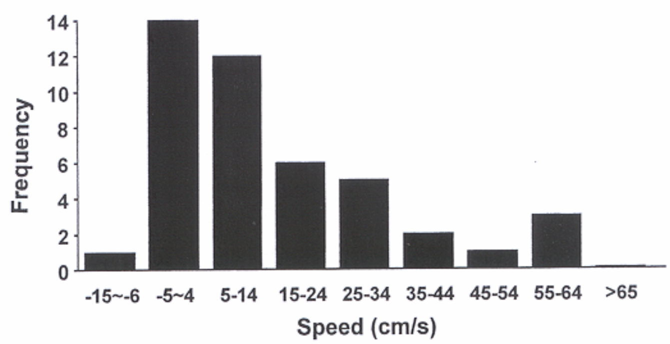

Figure 3. The number (frequency) attaining different magnitudes of change in gait speed in a group of 44 patients (n = 44) with hemiparesis after 2 months of in-patient rehabilitation (same patients as depicted in Fig. 2). Mean change after therapy was 16 ± 17 cm/s (mean ± 1 SD).

of moderate community activities without use of a wheelchair. At this speed it should be possible to manage a local store or an uncrowded shopping center (possibly with supervision), but some assistance likely is needed in a crowded shopping center. To be able to predict independence in the community, a speed of 80 ± 18 cm/s is required. To take the analysis one step further, to cross a street in a large city requires a gait speed of 120 cm/s; in smaller cities, 80 cm/s may be sufficient. Only 7 of the 44 patients (15.9%) depicted in Figures 2 and 3 attained 80 cm/s (community independence by these standards) after 2 months of therapy.

One can question whether walking speed should be the measure of choice in the full range of walking disabilities. It is evident that to measure walking speed, the subject first must have some walking capacity, referred to as the floor effect.[6] In subjects able to walk very slowly with much or little manual assistance, clinical measures (Fugl-Meyer leg subscore,[25] Barthel ambulation subscore,[47] and Berg Balance scale[4,96]) may discriminate more than gait speed between the degrees of assistance required.[67] At walking speeds higher than about 34 cm/s, however, walking speed is more discriminative because the clinical measures plateau (ceiling effect).[67] This concept of sensitivity to change over the range of recovery is very important in choosing an outcome measure to evaluate the effects of an intervention. This issue of responsiveness also is addressed in the next section.

ASSESSMENT OF LOCOMOTOR-RELATED TASKS AFTER STROKE

The assessment of locomotor-related tasks is an important part of the functional assessment of patients after stroke. Although independent walking is the ultimate goal for many patients, for others, it is only one of many milestones in a long-term process to achieve more demanding locomotor tasks (running, ascending and descending stairs, going up and down steep ramps, and walking on icy surfaces). Therefore, the assessment of the locomotor capacity should include a large spectrum of locomotor-related tasks that allow for evaluation at various stages of locomotor recovery. Locomotor-related tasks are activities that induce a displacement of the whole body in space (e.g., walking, ascending stairs, running), as well as activities that involve the lower limbs without a displacement of the whole body in space (e.g., standing up, stepping, stationary bicycling, toe-standing).

ASSESSMENT OF GAIT

Gait speed, which has been shown to be valid, reliable, and responsive,[66,86–88] has become a measure of choice to identify the level of disability and to document locomotor performance.[26,67,86] Gait velocity can be tested over different distances (5 m, 10 m, and 30 m) and thus can be used to measure walking ability of patients in different stages of recovery.[67,73,86,88] The 5-m walk test recently has been promoted as the most responsive distance to be tested.[73] Although the 5-m walk test at natural pace may have the best metrologic characteristics, one should test other aspects of locomotor performance important for successful social reintegration (maximal walking speed, walking over different surfaces, outdoor conditions, locomotor endurance, and stair ascent and descent).

NATURAL WALKING SPEED AND TESTING CONDITIONS

Gait assessment should include conditions taking into account environmental constraints. For instance, one may be tempted to discharge a patient or interrupt locomotor training if a patient has reached a moderate walking speed (>70 cm/s). Functional walking speed, however, as defined by Perry et al,[62] requires a person to walk at a speed of 80 cm/s to be independent in the community. A person who walks at a preferred speed of 70 cm/s is not capable necessarily of sprints that increase the speed by 10 to 25 cm/s to render crossing a busy street possible. Thus, locomotor training and testing should include conditions that challenge the patient to walk at maximal speed to better estimate the functional walking ability. Ascending a slope and moving the head (horizontal and vertical turns) during walking are other conditions that challenge gait performance. The capacity to adjust to these disturbances should be taken into account in a global locomotor assessment. Recently, Shumway-Cook and Woollacott[76] described a gait dynamic index that is designed to assess the influence of internal and external stimuli on gait performance. Such an index must be validated in patients with neurologic impairments.

LOCOMOTOR ENDURANCE

If walking faster always has been a common goal, walking over a longer distance recently has gained the attention of therapists and researchers in the field of neurologic rehabilitation. This new focus on locomotor endurance has led to the use of tests that measure the distance walked during a fixed period of time, such as the 6-minute walk, which tests how far a subject can walk in a time frame, with rest stops if needed. This test, developed to assess cardiopulmonary function,[29] now is being used as an outcome measure for assessing locomotor endurance of patients after a stroke.[21,23] Because this locomotor test requires the patients to be able to stand for 6 minutes and walk over a certain distance, its use may be delayed in more impaired patients. Results from a recent pilot study indicate that 30 days poststroke, moderately impaired patients (gait speed of about 40 cm/s) were able to complete the 6-minute walk.[23] Recent observations, however, underline the need to train locomotor endurance even in patients who have attained a high level of locomotor recovery. For instance, patients already walking at a preferred velocity up to 142 cm/s had a 25% impairment of their walking endurance as measured by the 6-minute walk.[20] Following an intense locomotor training program (nine sessions of 1 hour each, over a 3-week period) these patients improved their walking endurance by more than 21% (from 21.2%–28.5%). Three patients with a preferred gait speed (10-m walk test) ranging from 122 cm/s to 142 cm/s increased their walking distance by a mean of 110 m (±5.3 m), corresponding to increases ranging from 27.6% to 28.5% (Fig. 4). At the end of the training all three covered a distance of over 495 m during the 6-minute walk (mean gait speed of 140 cm/s), which is a distance close to normal values.[24] These pilot results emphasize the need to train locomotor endurance even in patients with a very good walking performance and confirm the sensitivity to change of the 6-minute walk for patients with this high level of locomotor performance. Finally, preferred gait speed during the 10-m walk test was improved in all patients (Fig. 4), further suggesting that preferred gait speed also can detect changes in locomotor performance even in patients with a normal gait speed.

OTHER LOCOMOTOR-RELATED TASKS

The timed up-and-go (TUG) measures the time a subject 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 was developed as a measure of basic mobility.[63] It is a simple test which is quick and easy to administer, making it attractive to clinicians. An interesting feature is the inclusion of a series of tasks that challenge dynamic balance in different ways. Rising up from a chair and sitting down induces changes of body position that require adjustments that differ from those during walking and turning around. Like gait, the TUG challenges balance and mobility, but under

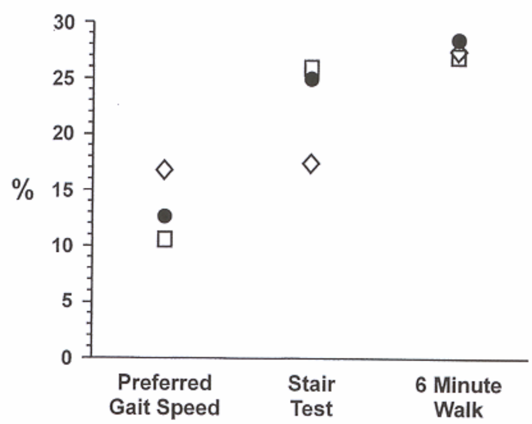

Figure 4. Percent improvement measured with three different outcome measures after a 3-week locomotor training program in three chronic stroke patients with a normal walking speed at onset of training. Circle = patient 1; square = patient 2; diamond = patient 3.

more variable conditions and changes of pace imposed by the requirements of successive tasks. For the TUG test, the patients are allowed to use a walking aid (cane, orthosis), but they cannot receive external assistance. Even so, the use of a walking aid makes the test accessible to patients in the earlier stage of locomotor recovery with insufficient strength or balance for independent walking. Results from an ongoing study indicate that 2 months after stroke onset, 18 patients (34%) out of a group of 53 were unable to complete the TUG, 24 (45%) were able to perform the test without a walking aid, and 11 (21%) needed an aid. The patients with the slowest gait speed (range: 9 to 24 cm/s; mean 15 cm/s) were not able to complete the TUG.

THE TUG PERFORMANCE OVER TIME

Figure 5, illustrates the performance over time of a group of 35 patients who were assessed at 2 and 4 months after stroke. In Figure 5*A*, the patients are grouped according to gait velocity (slow, moderate and fast), and in Figure 5*B*, as to whether they completed the TUG with or without a walking aid. At 4 months a third group of patients walking faster than 71 cm/s is added. The time to complete the TUG could be as long as one minute in slow walkers (Fig. 5*A*) or in patients who required a walking aid (Fig. 5*B*). The patients who still walked at less than 30 cm/s (slow speed) 4 months poststroke needed nearly 50 seconds to complete the TUG. Patients who still completed the TUG with a walking aid at 4 months, however, required half the time (mean 32 seconds) taken at 2 months (Fig. 5*B*). In the group of faster walkers, however, the TUG score was close to normal values; patients walking at 120 cm/s and faster had a TUG score within 1 or 2 seconds of the mean normal value. These results indicate that although the TUG is applicable to a large proportion of patients during locomotor recovery, one should be aware that for patients who walk at or close to a normal speed, the TUG is not useful as an outcome measure because both measures (gait speed and TUG) are closely related. The latter relationship is illustrated in Figure 6, which gives the TUG performance relative to gait speed. A Pearson correlation coefficient of $r = -0.80$ was calculated between the TUG score and the gait velocity for the whole group of 35 patients (Fig. 6*A*). The relationship was even stronger for the group without an aid ($r = -0.85$), and, conversely, the group needing an aid had a lower relationship ($r = -0.57$). In comparison to other clinical measures (Fig. 6*B*) the relationship with gait speed was stronger for the TUG as expected because the TUG includes walking per se.

RESPONSIVENESS OF THE TUG

In the same group of 35 patients, the mean change score (percentage) calculated between scores obtained 2 and 4 months after stroke for each

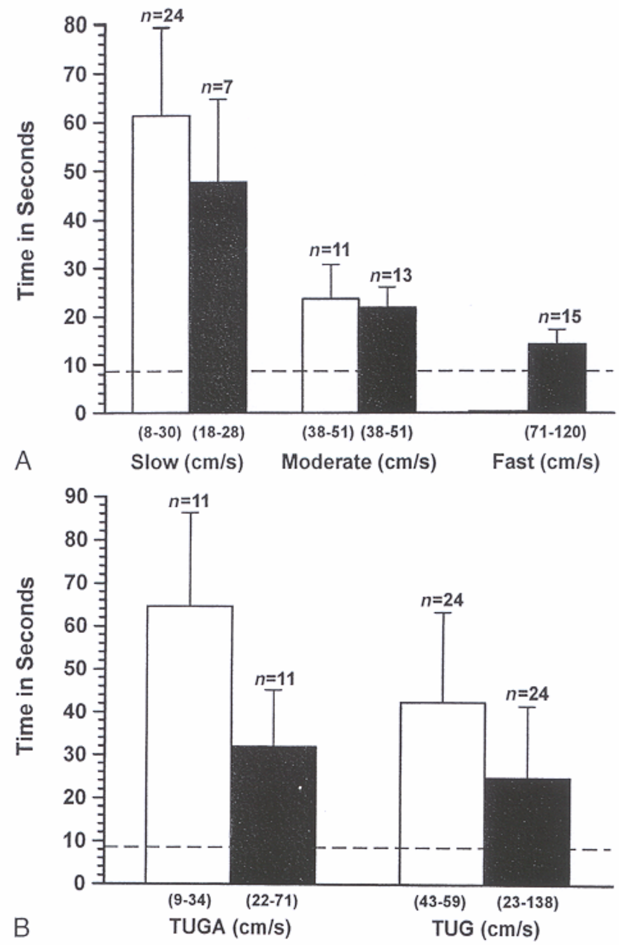

Figure 5. *A,* Mean time in seconds (±1 SD) to complete the Timed Up and Go (TUG) in three groups of patients with different walking speeds. *B,* Time to perform the TUG in patients who used a walking aid (TUGA) or did not (TUG); range of walking speeds given in parentheses. Open bar = 2 months; solid bar = 4 months. Dashed horizontal line gives the mean time for control subjects. (*Data for dashed line from* Podsiadlo D, Richardson S: The Timed "up and go": A test of basic functional mobility for frail elderly persons. J Am Geriatr Soc 39:142, 1991.)

of the clinical tests and respective responsiveness measures are reported in Table 1. Except for the Fugl-Meyer leg[25] and the Berg[4] tests, the mean changes were generally larger than 30%, with the TUG showing improvements above 200%. Table 1*B* gives two measurements of responsiveness: the effect size and the standardized response measure (SRM). The effect

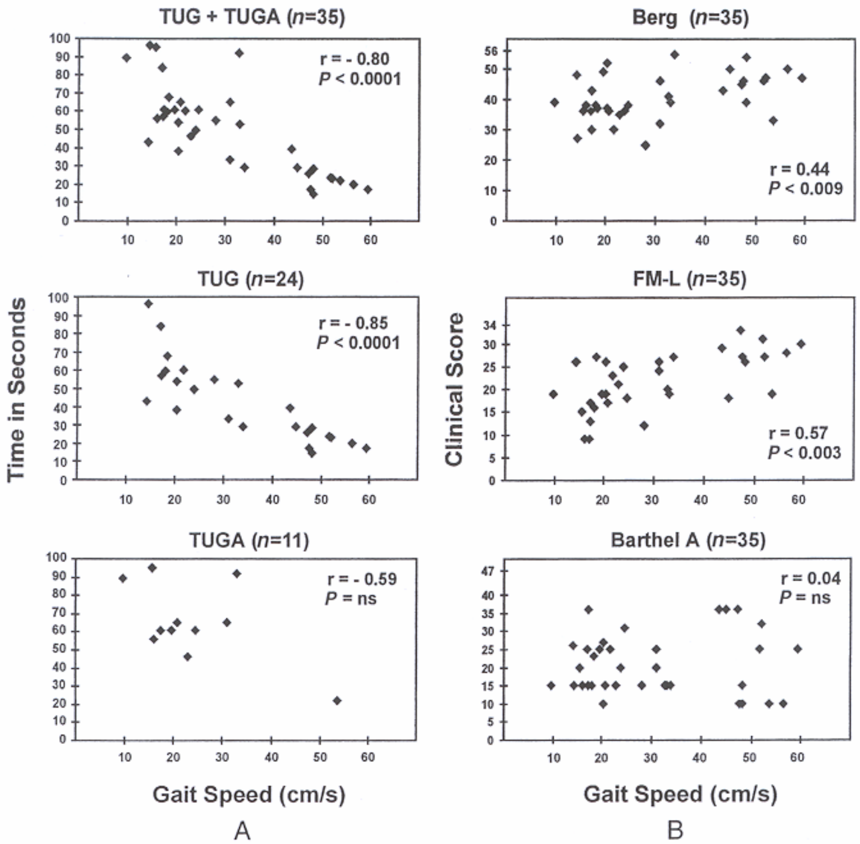

Figure 6. *A,* Relationship of the Timed Up and Go (TUG) performance times and gait speed in the total group of patients (TUGA [with walking aid] + TUG [without aid]) and in each of the two groups separately. *B,* The relationship between gait speed and Berg score (*top*), Fugl-Meyer leg subscore (*middle*) and Barthel ambulation subscore (*bottom*).

Table 1. CHANGE SCORES (IN PERCENT) IN CLINICAL MEASURES (A) AND RESPONSIVENESS (B) OVER A 2-MONTH PERIOD

	Barthel	Berg	F-ML	Gait Speed	TUG
A					
$n = 35$	41.5	14.0	6.0	31	264
$n = 24$	42.0	13.3	6.3	36	208
$n = 11$	40.7	15.6	6.4	20	386
B					
ES	2.36	1.03	0.35	2.42	0.95
SRM	2.37	1.29	0.66	1.14	0.99

Barthel = Barthel ambulation subscore; F-ML = Fugl-Meyer leg motor subscore; TUG = timed up & go; ES = effect size; SRM = standardized response measure.

size represents the average change score (posttest–pretest values) divided by the SD of the pretest values, while the SRM represents the average change score over a set period of time divided by the SD of that change. The TUG demonstrated a high (>0.80) level of measurement responsiveness; the least responsive measure was the Fugl-Meyer leg test, which is a measure of impairment (Table 1*B*). Thus, the TUG can be expected to be highly responsive as long as the gait speed is not too close to normal values. In the latter case, more demanding locomotor-related tasks, such as the stair test (see subsequent section) should be used to monitor change in locomotor performance.

PREDICTIVE VALUE OF THE TUG

The Pearson correlation coefficient calculated between the baseline TUG score and gait speed 2 months later, in the group of 24 patients who completed the TUG without a walking aid, was $r = -0.58$, indicating that the TUG is a good predictor of locomotor outcome. In patients who needed a walking aid to complete the TUG, baseline score had no predictive value ($r = 0.16$) on the locomotor outcome. These results are consistent with the stronger relationships computed between the TUG score and both balance and gait speed in patients able to complete the TUG without a walking aid.

THE TUG AND THE BERG TEST

As expected scores of the patients in the no-aid group were strongly related ($r = -0.77$) to the Berg scores. This was in contrast with the poor relationship in the aided group ($r = 0.24$). These results indicate that the TUG performance in patients who can complete the test without a walking aid is a good indicator of both walking and balance performance, whereas it is merely an indicator of the walking performance for patients who need a walking aid for the TUG.

THE STEP TEST

The step test is a relatively new measure of dynamic standing balance.[34] It involves stepping one foot on, then off, a block 7.5 cm high, as quickly as possible. The number of repetitions in 15 seconds is recorded, and the stepping is performed by each leg separately. Test and retest in healthy subjects ($n = 41$) and patients with a stroke ($n = 41$) yielded intraclass correlation coefficients of 0.94 and 0.93 respectively,[34] and the stepping performance was correlated with gait speed ($r = 0.83$). In this group of 41 relatively acute patients (mean 2 months after stroke), the mean number of repetitions was 7.7 ± 3.9 with the unaffected limb and 7.0 ± 4.0 with the affected limb, corresponding, respectively, to 42% and

39% of control (17.7 $\pm$ 3.2) values.[34] In another study of 29 patients, the change in the number of repetitions after a 1-month in-patient rehabilitation revealed mean increases of 74% (from 4.2 to 7.3 repetitions) and 71% (from 3.8 to 6.5 repetitions), respectively, in the affected and unaffected limbs.[6] The responsiveness reported for the step test in this group of patients was even higher (SRM = 0.95) than that of gait speed (SRM = 0.80). Two months poststroke, 41% of the patients were unable to perform the step test with either limb, but this percentage dropped to 14% at retesting 4 weeks later.[6] Thus, the proportion of patients unable to perform the step test is similar to that for the TUG in patients tested 2 months poststroke. A special feature of the step test is that performing the test with the unaffected leg and then the other provides two types of information about the paretic limb: its movement capacity (when stepping with the affected leg) and postural function (when stepping with the unaffected leg). This discriminative feature of the step test may provide insight about specific locomotor impairments. For instance, a lower number of steps with the affected leg in comparison with the unaffected leg may be associated with a predominant lack of flexion during the swing phase of gait. If such a discriminative feature of the step test is confirmed, it would make the step test most valuable, not only for assessing treatment outcomes but also for setting therapeutic goals.

THE STAIR TEST

This test could be defined as an advanced version of the TUG. The stair test includes the series of tasks from the TUG, with the addition of two tasks: stair ascent and stair descent. The patients are instructed to rise from a chair, walk 3 m, ascend a flight of 14 stairs, turn around, descend the stairs, walk back to the chair, and sit down. They can use a walking aid and the rail if necessary. This test has been used as a disability measure in patients with a total hip arthroplasty,[61] and more recently to monitor the effects of a locomotor-training program in a group of patients with a chronic stroke.[20] The performance of the group of stroke patients recorded before (pretest) and after a 3-week (posttest) training program is reported in Figure 7. It was possible with the Stair test, like for the 6-minute walk test, to detect improvement even in patients with a normal walking speed (>115 cm/s) at the study onset. Examination of individual segments of the stair test (Fig. 7) indicated that the patients performed the last two tasks of the stair test (stair descent and walking back, and sitting down) the least successfully. After training, however, the patients improved their performance by more than 26%, and their performance was close to control values for all tasks except for the last segment of the test (walking back and sitting down). The stair test also can be performed by patients with a moderate gait speed (70 cm/s), but these patients take a total time for the stair test that is more than three times that taken by control subjects.[20] Again these results underline the value of more demanding tasks to measure change in locomotor performance in patients who have at-

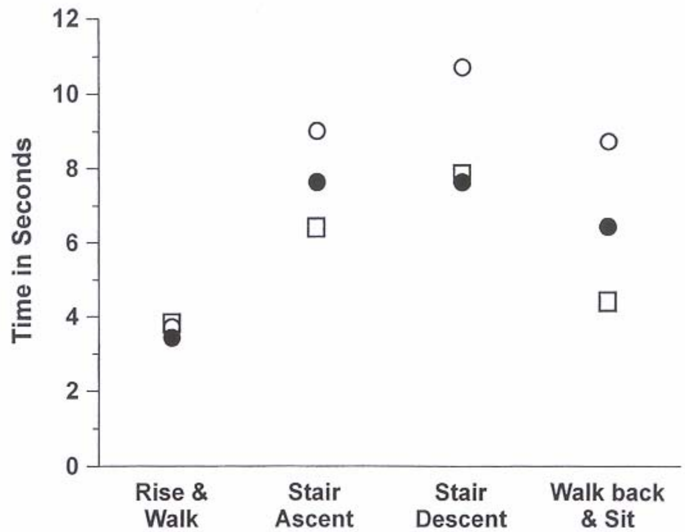

Figure 7. Mean duration of individual tasks in the stair test before locomotor training, (*open circle*) and after locomotor training (*solid circle*) in three patients with chronic stroke in comparison with a control group (*square*).

tained a high level of performance. Moreover, timing each segment of the stair test helped identify the more difficult tasks.

SUMMARY

The assessment of locomotor performance should extend beyond the traditional 10-m walk and include other locomotor-related tasks to provide a more complete picture of locomotor capacity after a stroke. The stage of locomotor recovery dictates the choice of outcome measures, and gait speed remains a good indicator for such selection. Patients with a moderate gait velocity generally are able to perform most of the locomotor-related tasks (TUG, step test, 6-minute walk, stair test). With improvement of the locomotor performance, however, assessment should rely on the more demanding tests to provide guidance for therapeutic goals.

TRAINING GAIT

Rehabilitation aims to reduce disability and handicap after stroke. It is becoming clearer that the muscle weakness and loss of coordinated movement are the major causes of disability after stroke.[12,17,42,71,78] Traditionally, however, approaches to stroke rehabilitation have focused primarily on spasticity and abnormal reflexes. For example, Bobath therapy assumes that abnormal postural reflex activity is the major cause of dysfunction, and as such a significant proportion of therapy time involves inhibiting spasticity and other abnormal responses.[78] There is a growing body of evidence, however, that directly challenges this clinical assumption. For instance, the clinical phenomenon of increased resistance to passive movement, commonly called *spasticity*, often reflects changes in the passive mechanical properties of muscle rather than hyperactive reflexes,[22,37,49,55,56] but also that if spasticity is reduced through biofeedback training[52] or dorsal rhizotomy,[3] this does not necessarily lead to improvement in function.

In contrast to the traditional approaches to rehabilitation, Carr and Shepherd,[13,15,16] for some time have advocated that movement rehabilitation should be task-related and derived from the scientific information available in the movement sciences, particularly information pertaining to the biomechanical characteristics of effective performance, how people learn and acquire skill in movement, and also the effects of pathology on function. To improve walking, task-related training involves strategies to increase strength, coordination, and weight-bearing capacity of the affected lower limb, to maintain a flexible musculoskeletal system and provide the opportunity for intensive practice to develop skill in walking.[15] Task-related training is gaining theoretic,[13,15,16,27,36,48,89,90] and experimental support as a means of improving walking after stroke.[23,31–33,45,50,70,84] For example, Richards et al[70] found that intensive, gait-focused therapy involving use of a tilt table and a limb-load monitor, resisted exercises in the upright position with an isokinetic device, and walking on a treadmill improved walking ability in acute stroke patients.

Given the contribution of muscle weakness to walking disability, specific strengthening exercises are a critical component of any task-related training[50,70] program after stroke. Ideally the strengthening exercises should resemble as closely as possible the tasks or components of the task being trained. Nugent et al[54] reported that the number of repetitions of an exercise specifically designed to strengthen the lower-limb extensor muscles was correlated with an improvement in walking outcome. The exercise was derived from an understanding of the biomechanical requirements of the stance phase of walking and involved extension of the affected hip, knee, and ankle joints in a weight-bearing position. This exercise made the subjects practice generating an overall extensor or support moment through their affected lower limb as is required in walking.[95] Carr and Shepherd[15] have described in detail a series of exercises designed to strengthen the affected lower limb in a functionally relevant way. Many of the exercises described involve stepping up and down off blocks forwards and sideways and are designed to promote the use of the affected lower limb to support, balance, and propel the body mass. Strengthening exercises must be tailored to the patient's ability. Changing the height of the blocks and setting the number of repetitions are ways to individually tailor the exercise so that individuals activate muscles in the affected limb rather than use adaptive movements to compensate with the stronger unaffected lower limb.

To improve coordination, practice of the whole task is also a crucial component of task-related training programs. Treadmill training with a harness if necessary to provide partial body-weight support has been found to be useful in improving gait after stroke.[31,33,83–85] Hesse et al,[32] for example, found that individuals who had received regular physiotherapy based on Bobath for at least 3 weeks without marked improvement in gait showed significant improvements in velocity, cadence, and stride length after 25 treadmill-training sessions.

Treadmill training after stroke appears beneficial for a number of reasons. First, one of the barriers to completion of walking practice by severely disabled patients is that the marked muscle weakness and poor coordination results in the inability of stroke patients to practice the whole task alone. Such patients often require the assistance of one or more people to take a few steps. Treadmill training with partial weight support may be particularly beneficial for such patients because it provides the opportunity to complete large amounts of practice of the whole task, that is, many repetitions of complete gait cycles. It has been suggested that practice of critical biomechanical components of a task may be necessary if the individual's motor impairments preclude effective practice of the whole task.[14] The partial body-weight support provided by an overhead harness is an example of modifying the task so that the patient can practice in the presence of poor muscle function without using adaptive movements. The overhead harness prevents collapse, reduces the muscle-force requirements, and decreases postural demands and anxiety, allowing the individual to practice walking in the presence of muscle weakness and loss of coordinated movement. Visintin et al[84] found that treadmill training with body-weight support that was reduced as training progressed was more effective than without support. To promote weight-bearing and task-related muscle activations, however, other research has suggested that the body weight supported by the harness should not exceed 30%.[33]

Treadmill training also may be beneficial because the motion of the treadmill enforces the appropriate timing relations between the lower limbs and also ensures that the hips are extended during stance phase, both of which are critical biomechanical components of walking.[59,74,92,93] Olney et al[58] have identified that lack of hip extension in stance phase of walking is a major problem following stroke. One may question, however, whether the entrainment by the treadmill provides ideal practice for the ankle plantarflexors at push-off.

Practice of walking over variant surfaces and obstacles and up and down slopes, escalators and stairs is critical in improving gait after stroke. Such practice not only improves strength and endurance but also assists the patient in learning to adapt walking to environmental demands.[5,15,60] In addition, walking over obstacles may be useful to promote practice of specific components of gait. For example, placement of obstacles can be used to encourage hip extension of the affected leg by forcing a longer step by the unaffected leg. Similarly, stepping over obstacles may be useful in assisting an individual who has difficulty flexing the hip and knee sufficiently in swing phase.[15]

Walking practice and strengthening exercises in the weight-bearing position also should assist in preventing secondary deleterious muscle adaptations such as muscle shortening and increased muscle stiffness.[15] Additional intervention may be necessary, however, to maintain a flexible musculoskeletal system. The calf muscles and hip flexor muscles are prone to shortening if the person spends large periods of the day sitting and inactive. Prolonged low-load stretching of these muscle groups such as standing with the hip extended and ankle dorsiflexed may be necessary, because shortening of these muscles interferes with the movement of the body mass over the foot in the stance phase of gait.[2]

Endurance training is also an important component of task-related training. Although many individuals attain some form of independent locomotion,[18,86] only a small proportion can walk with sufficient speed and endurance to enable them to function effectively within their community.[35,62] Macko et al[46] found that exercise capacity measured during treadmill walking was limited by generalized fatigue and not paretic leg fatigue, suggesting that endurance training should be incorporated into rehabilitation aimed at improving locomotor skills after stroke. Furthermore, studies that have investigated endurance training or included endurance components using treadmill walking or cycling have demonstrated beneficial results.[11,23,45,64] For example, Macko et al[45] reported that 6 months of low-intensity treadmill endurance training produced substantial and progressive reductions in energy expenditure and cardiovascular demands of walking.

Because practice is critical to skill acquisition, stroke rehabilitation must be organized to maximize the opportunity for practice with task-related training sessions under the guidance of a therapist as well as self-monitored practice sessions. Observational analysis of rehabilitation units over the past 15 years, however, has found consistently that stroke patients in rehabilitation spend large proportions of the day alone and inactive.[39,44,81] These findings suggest that the rehabilitation setting that is typically part of a hospital setting is not providing an appropriate environment to promote the activity necessary to improve skill. More recently, changes in healthcare structures have led to the early discharge of individuals. This move may be beneficial, as hospital environments are not very challenging nor representative of the demands posed in the community. Moreover, Tangemann et al[79] have suggested that individuals are more motivated to improve after returning home and seeing how their disability impacts their everyday life. There is some evidence of beneficial results of home-based rehabilitation.[23] Regardless of the rehabilitation setting, as stroke patients typically have difficulty performing a range of everyday tasks, and generalization of training effects appears limited to biomechanically similar actions,[21] in order to improve function they need to complete training and practice sessions for a range of tasks.

Table 2. TASK-RELATED TRAINING WORKSTATIONS INCORPORATED INTO A CIRCUIT CLASS

Workstation	Activity
1	Reaching for objects beyond arm's length in sitting
2	Sit-to-stand from various chair heights
3	Stepping up and down, forwards, and sideways onto blocks of various heights
4	Heel lifts in standing
5	Reaching for objects in standing including down to the floor with the base of support constrain
6	Reciprocal leg flexion and extension using the Kinetron in standing
7	Standing up from a chair, walking a short distance and returning to the chair
8	Walking on a treadmill
9	Walking over various surfaces and obstacles
10	Walking over slopes and stairs

Provision of practice books and exercise classes may be ways to increase the amount of practice completed during rehabilitation.[1] In addition, to reduce walking disability and handicap after stroke, provision of and access to exercise classes after discharge is necessary because individuals frequently are discharged with locomotor skills inferior to that required for community ambulation.[35,43,62] The authors investigated the effects of a circuit class aimed at improving the performance of locomotor-related tasks in chronic stroke.[21] The classes involved subjects completing practice at a series of workstations, as well as participating in walking races and relays. The workstations incorporated into the circuit are outlined in Table 2. The results of this preliminary study suggest that this training is effective at improving the walking speed and endurance in chronic stroke.

The assessment, analysis, and locomotor training of patients after stroke should be based on knowledge of the biomechanical characteristics of walking in healthy subjects and an understanding of the effects of the neurologic impairment in the patients. The authors recommend that this knowledge as well as information about skill acquisition be used to design task-related training strategies that allow for the completion of large amounts of practice. Current knowledge supports training approaches that emphasize task-oriented strengthening and coordination exercises to promote endurance in various contextual and motivational environments. In addition, the selection of a few appropriate outcome measures reduces redundant information and allows greater opportunity for more supervised practice.

References

1. Ada L, Canning CG, Westwood P: The patient as an active learner. *In* Ada L, Canning C (eds): Key Issues in Neurological Physiotherapy. Oxford, Butterworth-Heinemann, 1990, p 99

2. Ada L, Canning C: Anticipating and avoiding muscle shortening. *In* Ada L, Canning C (eds): Key Issues in Neurological Physiotherapy. Oxford, Butterworth-Heinemann, 1990, p. 219
3. Adams J, Cahan LD, Perry J, et al: Foot contact pattern following selective dorsal rhizotomy. Pediatr Neurosurg 23:76, 1995
4. Berg K, Wood-Dauphinee S, Williams JI, et al: Measuring balance in the elderly: Preliminary development of an instrument. Physiotherapy Canada 41:304, 1989
5. Bassile C, Bock C: Gait training. *In* Craik RL, Oatis CA (eds): Gait Analysis: Theory and Applications. St Louis, Mosby, 1995, p 420
6. Bernhardt J, Ellis P, Denisenko S, et al: Changes in balance and locomotion measures during rehabilitation following stroke. Physiotherapy Research International 3:109, 1998
7. Bobath B: Adult Hemiplegia: Evaluation and Treatment. London, Heinemann Medical Books, 1970
8. Bobath B: Adult Hemiplegia: Evaluation and Treatment, ed 3. London, Heinemann Medical Books, 1990
9. Bohannon RW: Strength of lower limb related to gait velocity and cadence in stroke patients. Physiotherapy Canada 38:204, 1986
10. Brandstater ME, de Bruin H, Gowland C, et al: Hemiplegic gait: Analysis of temporal variables. Arch Phys Med Rehabil 64:583, 1983
11. Brinkman JR, Hoskins TA: Physical conditioning and altered self-concept in rehabilitated patients. Phys Ther 59:859, 1979
12. Burke D: Spasticity as an adaptation to pyramidal tract injury. Adv Neurol 47:401, 1988
13. Carr JH, Shepherd RB: A motor learning model for rehabilitation. *In* Carr JH, Shepherd RB (eds): Movement Science: Foundations for Physical Therapy in Rehabilitation. Rockville, MD, Aspen Press, 1987, pp 31–92
14. Carr JH, Shepherd RB: A motor learning model for stroke rehabilitation. Physiotherapy 89:372, 1989
15. Carr JH, Shepherd RB: Neurological Rehabilitation: Optimizing Motor Performance. Oxford: Butterworth Heinemann, 1998
16. Carr JH, Shepherd RB: A Motor Relearning Programme for Stroke, ed 1. Oxford, Butterworth-Heinemann, 1982
17. Carr JH, Shepherd RB, Ada L: Spasticity: Research findings and implications for intervention. Physiotherapy 81:421, 1995
18. Dean CM, Mackey FM: Motor assessment scale scores as a measure of rehabilitation outcome following stroke. Aust J Physiother 38:31, 1992
19. Dean CM, Shepherd RB: Task-related training improves the performance of seated reaching tasks after stroke: A randomized controlled trial. Stroke 28:722, 1997
20. Dean CM, Malouin F, Richards CL: Locomotor task-related training for chronic stroke: multiple single case design. Clin Rehabil, August, 1999
21. Dean CM, Richards CL, Malouin F: Task-related training improves the performance of locomotor tasks in chronic stroke. A randomized, controlled pilot study. Arch Phys Med Rehabil, in preparation
22. Dietz V, Quintern J, Berger W: Electrophysiological studies of gait in spasticity and rigidity: Evidence that mechanical properties of muscles contribute to hypertonia. Brain 104:431, 1981
23. 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 29:2055, 1998
24. Enright PL, Sherrill DL: Reference equations for the six-minute walk in healthy adults. Am J Respir Crit Care Med 158:1384, 1998
25. Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S: The post-stroke hemiplegic patient: 1. A method for evaluation of physical performance. Scand J Rehabil Med 7:13, 1975
26. Goldie PA, Matyas TA, Evans OM: Deficit and change in gait velocity during rehabilitation after stroke. Arch Phys Med Rehabil 77:1074, 1996
27. Gordon J. Assumptions underlying physical therapy intervention: Theoretical and historical perspectives. *In* Carr JH, Shepherd RB (eds): Movement Science: Foundations for Physical Therapy in Rehabilitation. Rockville, MD: Aspen Press, 1987, p 1

28. Gresham GE, Duncan PW, Stason WB, et al: Post-Stroke Rehabilitation (Clinical Practice Guideline, No. 16). Rockville, MD: US. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, 1995
29. Guyatt GH, Pugsley SO, Sullivan MJ, et al: Effect of encouragement on walking test performance. Thorax 39:818, 1984
30. Guyatt GH, Sullivan MJ, Thompson PJ, et al: The 6-minute walk: A new measure of exercise capacity in patients with chronic heart failure. Can Med Assoc J 132:919, 1985
31. Hesse S, Bertelt C, Jahnke MT, et al: Treadmill training with partial body weight support compared with physiotherapy in nonambulatory hemiparetic patients. Stroke 26:976, 1995
32. Hesse S, Bertelt C, Schraffin A, et al: Restoration of gait in nonambulatory hemiparetic patients by treadmill training with partial body weight support. Arch Phys Med Rehabil 75:1087, 1994
33. Hesse S, Helm B, Krajnik J, et al: Treadmill training with partial body weight support: Influence of body weight release on the gait of hemiparetic patients. J Neurol Rehabil 11:15, 1997
34. Hill K, Bernhardt J, McGann A, et al: A new test of dynamic standing balance for stroke patients: Reliability and comparison with healthy elderly. Physiotherapy Canada 48:257, 1996
35. Hill K, Ellis P, Bernhardt J, et al: Balance and mobility outcomes for stroke patients: A comprehensive audit. Aust J Physiother 43:173, 1997
36. Horak, FB: Assumptions underlying motor control for neurological rehabilitation. *In* Proceedings of the 11 STEP Conference: Contemporary Management of Motor Control Problems. Alexandria, Foundation for Physical Therapy, 1991, p. 11
37. Hufschmidt A, Mauritz K-H: Chronic transformation of muscles in spasticity: A peripheral contribution to increased tone. J Neurol Neurosurg Psychiatry 48:676, 1985
38. Jørgensen HS, Nakayama H, Raaschou HO, et al: Recovery of walking function in stroke patients: The Copenhagen Stroke Study. Arch Phys Med Rehabil 76:27, 1995
39. Keith RA: Activity patterns of a stroke rehabilitation unit. Soc Sci Med 14A:575, 1980
40. Knutsson E, Richards CL: Different types of disturbed motor control in gait of hemiparetic patients. Brain 102:405, 1979
41. Lamontagne A, Malouin F, Richards CL: Locomotor-specific measure of spasticity of plantarflexors muscles after stroke. Gait & Posture, August, 1999
42. Landau, WM: Spasticity: What it is? What is it not? *In* Feldman RG, Young RR, Koella WP (eds): Spasticity: Disordered Motor Control. Miami, Symposia Specialists, 1980, p 17
43. Lerner-Frankiel MB, Vargas S, Brown MB, et al: Functional community ambulation: What are your criteria? Clinical Management 6:12, 1986
44. Mackey F, Ada L, Heard R, et al: Stroke rehabilitation: Are highly structured units more conducive to physical activity than less structured units? Arch Phys Med Rehabil 77: 1066, 1996
45. Macko RF, DeSouza CA, Tretter LD, et al: Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients: A preliminary report. Stroke 28:326, 1997
46. Macko RF, Katzel LI, Yataco A, et al: Low-velocity graded treadmill stress testing in hemiparetic stroke patients. Stroke 28:988, 1997
47. Mahoney FI, Barthel DW: Functional evaluation: the Barthel Index. Mar Med J 14:61, 1965
48. Majsak MJ: Application of motor learning principles to the stroke population. Topics in Stroke Rehabilitation, 3:27, 1996
49. Malouin F, Bonneau C, Pichard L, et al: Non-reflex mediated changes in plantarflexor muscles early after stroke. Scand J Rehabil Med 29:147, 1997
50. Malouin F, Potvin M, Prevost J, et al: Use of an intensive task-oriented gait training program in a series of patients with acute cerebrovascular accidents. Phys Ther 72:781, 1992
51. Moffet H, Comeau F, Malouin F, et al: A new gait analysis system using binary image processor: Intra and inter-rater reliability of kinetic and kinematic measurements. *In* Proceedings VIth Conference of the Canadian Society for Biomechanics, Quebec City, August, 1990, p 179

52. Neilson PD, McCaughey J: Self-regulation of spasm and spasticity in cerebral palsy. J Neurol Neurosurg Psychiatry, 45:320, 1982
53. Norton BJ, Bomze HA, Sahrmann SA, et al: Correlation between gait speed and spasticity at the knee. Phys Ther 55:355, 1975
54. Nugent JA, Schurr KA, Adams RA: A dose-response relationship between the amount of weight-bearing exercise and walking outcome following cerebrovascular accident. Arch Phys Med Rehabil 75:399, 1994
55. O'Dwyer NJ, Ada L: Reflex hyperexcitability and muscle contracture in relation to spastic hypertonia. Curr Opin Neurol 9:451, 1996
56. O'Dwyer NJ, Ada L, Neilson PD: Spasticity and muscle contracture following stroke. Brain 119:1737, 1996
57. Olney S, Richards CL: Hemiparetic gait following stroke: Part I. Characteristics. Gait & Posture 4:136–148, 1996
58. Olney SJ, Griffin MP, McBride ID: Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: A regression approach. Phys Ther 74, 872, 1994
59. Olney SJ, Griffin MP, Monga TN, McBride ID: Work and power in gait of stroke patients. Arch Phys Med Rehabil 72:309, 1991
60. Patla AE: A framework for understanding mobility problems in the elderly. *In* Craik RL, Oatis CA (ed): Gait Analysis: Theory and Applications. St Louis, Mosby, 1995, p 436
61. Perron M, Malouin F, Moffet H: Relationship between impairments and disabilities after total hip arthroplasty. Can J Rehabil 11:255, 1998
62. Perry J, Garrett M, Gronley JK, et al: Classification of walking handicap in the stroke population. Stroke 26:982, 1995
63. Podsiadlo D, Richardson S: The timed "up and go." A test of basic functional mobility for frail elderly persons. J Am Geriatr Soc 39:142, 1991
64. Potempa K, Lopez M, Braun LT, et al: Physiological outcomes of aerobic exercise training in hemiparetic stroke patients. Stroke 26:101, 1995
65. Richards CL, Knutsson E: Evaluation of abnormal gait patterns by intermittent-light photography and electromyography. Scand J Rehabil Med Suppl 3:61, 1974
66. Richards CL, Olney S: Hemiparetic gait following troke: Part II. Recovery and physical therapy. Gait & Posture 4:149, 1996
67. Richards CL, Malouin F, Dumas F, et al: Gait velocity as an outcome measure of locomotor recovery after stroke. *In* Craik RL, Oatis CA (ed): Gait Analysis: Theory and Applications. St Louis, Mosby, 1995, p 355
68. Richards CL, Malouin F, Dumas F, et al: Recovery of ankle and hip power during walking after stroke. Can J Rehabil 11:271, 1998
69. Richards CL, Malouin F, Dumas F, et al: The relationship of gait speed to clinical measures of function and muscle activations during recovery post-stroke. American and Canadian Societies of Biomechanics, Proceedings of the Second North American Congress on Biomechanics, Chicago, August, 1992, p 299
70. Richards CL, Malouin F, Wood-Dauphinee S, et al: Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Arch Phys Med Rehabil 74:612, 1993
71. Sahrmann SA, Norton BS: The relationship of voluntary movement to spasticity in the upper motor neurone syndrome. Ann Neurol 2:460, 1977
72. Salbach NM: Gait Speed as a Measure of Stroke Outcome. Master's thesis, McGill University, 1997
73. Salbach NM, Mayo N: Predictors of gait speed recovery in acute stroke. Can J Rehabil 4:228, 1998
74. Saunders JB, Inman VT, Eberhart HD: The major determinants in normal and pathological gait. J Bone Joint Surg 35A:543, 1953
75. Shiavi R, Bugle HJ, Limbird T: Electromyographic gait assessment, part 2: Preliminary assessment of hemiparetic synergy patterns. J Rehabil Res Dev 24:24, 1987
76. Shumway-Cook A, Woollacott M: Motor Control Theory and Practical Applications. Baltimore, William & Wilkins, 1995
77. Skilbeck CE, Wade DT, Hewer RL, et al: Recovery after stroke. J Neurol Neurosurg Psychiatry 46:5, 1983

78. Tang A, Rymer WZ: Abnormal force–EMG relations in paretic limbs of hemiparetic human subjects. J Neurol Neurosurg Psychiatry 44:690, 1981
79. Tangemann P, Banaitis D, Williams A: Rehabilitation for chronic stroke patients: Changes in functional performance. Arch Phys Med Rehabil 71:876, 1990
80. Tardieu C, Tardieu G, Colbeau-Justin P, et al: Muscle hypoextensibility in children with cerebral palsy: II. Therapeutic implications. Arch Phys Med Rehabil 63:103, 1982
81. Tinson DJ: How stroke patients spend their days: An observational study of the treatment regime offered to patients in hospital with movement disorders following stroke. International Disability Studies 11:45, 1989
82. Twitchell TE: Restoration of motor function following hemiplegia in man. Brain 74:443, 1951
83. Visintin M, Barbeau H: The effects of body weight support on the locomotor pattern of spastic paretic patients. Can J Neurol Sci 16:315, 1989
84. Visintin M, Barbeau H, Korner-Bitensky N, et al: A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. Stroke 29:1122, 1998
85. Waagfjord J, Levangie PK, Certo CME: Effects of treadmill training in a hemiparetic patient. Phys Ther 70:549, 1990
86. Wade DT: Measurement in Neurological Rehabilitation. Oxford, Oxford University Press, 1992
87. Wade DT, Hewer RL: Functional abilities after stroke: Measurement, natural history and prognosis. J Neurol Neurosurg Psychiatry 50:177, 1987
88. Wade DT, Wood VA, Heller A, et al: Walking after stroke: Measurement and recovery over the first 3 months. Scand J Rehabil Med 19:25, 1987
89. Winstein CJ: Designing practice for motor learning: Clinical implications. *In* Proceedings of the 11 STEP Conference: Contemporary Management of Motor Control Problems. Alexandria, VA, Foundation for Physical Therapy, 1991, p 65
90. Winstein CJ, Knecht HG: Movement science and its relevance to physical therapy. Phys Ther 70:759, 1990
91. Winter DA: A. B. C. (Anatomy, Biomechanics and Control) of Balance During Standing and Walking. Waterloo, Ontario, Waterloo Biomechanics, 1995
92. Winter DA: Biomechanics of Human Movement. New York, Wiley, 1979
93. Winter DA: The Biomechanics and Motor Control of Human Gait. Waterloo, Ontario, University of Waterloo Press, 1987
94. Winter DA: The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological, ed 2. Waterloo, ON, University of Waterloo Press, 1991
95. Winter DA: Overall principle of lower limb support during stance phase of gait. J Biomech 13:923, 1980
96. Wood-Dauphinee S, Berg K, Bravo G, et al: The Balance Scale: Responsiveness to clinically meaningful changes. Can J Rehabil 10:35, 1997

Address reprint requests to

Carol L. Richards, PhD, PT
Rehabilitation Institute of Quebec
Rehabilitation Research Group
Local B–77
525 Boul. Wilfred-Hamel Est
Québec (P. Qué.) G1M 2S8

e-mail: carol.richards@pht.ulaval.ca