Hemiparetic gait following stroke. Part II: Recovery and physical therapy

Carol L. Richards*a, Sandra J. Olneyb

aPhysiotherapy Department, Faculty of Medicine, Laval University, Quebec City, Quebec G1K 7P4, Canada
bSchool of Rehabilitation Therapy, Queen's University, Kingston, Ontario K7L 3N6, Canada

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Abstract

This part begins with a review of locomotor recovery following stroke as revealed by clinical measures. These describe a recovery curve with a rapid rise within the first 6 weeks when active rehabilitation procedures supplement natural recovery, followed by a more gradual rise thereafter to plateau at about 6 months after stroke. The sensitivity of these clinical measures is questioned for more advanced stages of recovery since gait velocity and other quantitative EMG and movement variables reveal changes indicative of recovery up to 2 years after stroke. A review is then made, from the traditional to the most recent, of physical-therapy approaches used to promote locomotor recovery. Therapy choice for different types of disturbed motor control is briefly discussed. The need for the development of new therapeutic strategies based on current science is emphasized as is the use of sensitive outcome measures and appropriate study designs to evaluate therapy efficacy.

Keywords: Stroke; Gait; Locomotor recovery; Physical therapy

1. Introduction

Part I of this review identified the pathological patterns that characterize the gait of persons who have sustained a stroke. Although many studies alluded to the fact that change occurs over time, which is referred to as the period of recovery, the reader will have noticed that reports of specific changes in gait during recovery are rare. Recovery following stroke is a complex process involving both spontaneous recovery and the effects of therapeutic interventions, changes that are difficult to separate. Also, we would expect some interaction between the stage of motor recovery and the nature of the therapeutic intervention, but this important and controversial issue remains poorly understood. Despite definite limitations in our information, we have accrued some knowledge about recovery and about therapy and, perhaps more importantly, about the issues that are central to the further development of this knowledge base. The purpose of Part II is to review, from this perspec-

tive, locomotor recovery and the physical therapy interventions that are employed after stroke.

In Canada, about 50 000 persons are newly affected by stroke each year and the resulting impairment leads to the most disabling chronic condition (for a review, see Mayo [1]). For those who survive, recovery will be variable both with regards to its time course and its extent. Primary goals of the stroke victim include to be able to walk independently and to manage to function in daily activities. For the purposes of this paper, recovery is defined loosely as changes in motor behaviour that indicate improvement in relation to normal performance levels. This review will first examine the time course of motor recovery, as determined by clinical measures to provide a general picture of the recovery process. The sensitivity of selected clinical measures of recovery will be reexamined in light of recent studies using quantitative measures such as gait velocity as indices of recovery. The importance of appropriate measures of recovery or change will be emphasized by specific examples. The questions: 'How soon after stroke do subjects walk?' and 'How fast do they walk?' will be addressed.

To understand the physical therapy approach to su-
jects with a hemiparetic gait disorder, one must identify an acute stage post-stroke that includes the period when the individual must be medically stabilized before rehabilitation procedures begin. It is not uncommon that physical therapy will begin within 1 week post-stroke when the majority of subjects have not yet recovered the capacity to walk. The intervention approach at this stage must by necessity be radically different to that provided to an independently walking subject 1 year post-stroke. An attempt will be made to relate the physical therapy approach to the stage of recovery and the type of disturbed motor control. The rationales of traditional and more recent physiotherapeutic strategies, as well as adjuncts such as biofeedback to promote locomotor recovery will be briefly reviewed and results of selected clinical studies will be reported. The importance of the choice of outcome measures, control groups and study design will be discussed in relation to efficacy studies. This review will end with a synopsis of the state of the art in terms of physical therapy interventions and directions for future studies.

2. Recovery of motor function post-stroke

Based on observations of recovery following stroke, Twitchell [2] argued that motor recovery followed a predictable and step-wise sequence, and although individuals could attain a plateau at any stage in the sequence, the order was invariant. This concept of recovery was supported by the work of Gowland [3] who studied recovery in 335 subjects. Brunnstrom [4] further separated out the process of recovery into six definable stages. In stage 1, immediately after the stroke, flaccidity is present and no movements of the limbs can be initiated. As recovery begins, stage 2, the basic limb synergies or some of their components may appear as associated reactions, or minimal voluntary movement responses may be present and spasticity begins to develop. In stage 3, voluntary control of the movement synergies appears, although full range may not be attained and spasticity increases further. Stage 4 is reached when it becomes possible to execute some voluntary movement combinations outside the synergistic patterns while spasticity begins to decline. In stage 5 more difficult movement combinations are learned as the basic synergies lose their dominance over motor acts. Finally in stage 6, spasticity has disappeared, individual joint movements become possible and coordination approaches normal. These stages form the basis of the Fugl-Meyer Sensorimotor Scale [5], one of the best known clinical instruments used to estimate recovery post stroke. Subjects are rated, by trained evaluators, on their ability to perform specific tasks or for the presence or absence of reflexes, sensation or other motor behaviours. The total scale allocates 24 points to sensation, 14 points to balance, 66 points to the upper extremity, and 34 points to lower extremity function. The scale has good reliability and validity properties [5,6].

One of the most widely used measures of functional independence is the Barthel Index [7]. The index assesses 15 items related to self-care and mobility. Activities of daily living account for 33 points, bowel and bladder control 20 points, and ambulation activities, including the capacity to walk 50 m and to negotiate stairs, 47 points. Studies have shown this index to have high reliability and validity [8,9] and moderate responsiveness [10,11].

Studies using clinical measures of recovery (such as the Fugl-Meyer Scale [5] or Barthel Index [7]) in different populations of stroke survivors have established that much of the motor recovery occurs before 3 months post-stroke and that the rising slope of the recovery profile flattens out to plateau at about 6 months post-stroke [2,12–14], although recovery has been reported up to 5 years post-stroke in special cases [15]. In a longitudinal study in a cohort of patients with a lesion due to infarction of the middle cerebral artery, Richards et al. [16] confirmed the findings of previous studies as to the time course of the recovery profile and also showed that most of the recovery occurs in the first 6 weeks post-stroke when active rehabilitation procedures supplement natural recovery.

The prediction of the natural course of recovery or the potential effect of a given therapy is a challenge to rehabilitation specialists. Gowland [3] developed regression equations for predicting the level of sensorimotor recovery of a subject during an active rehabilitation program. Of 23 clinical variables included, the stage of recovery of the leg and the number of weeks post-stroke were the most important predictors. Initial severity of the stroke [17–19], age [17] and leg strength [18,19] are also good predictors of functional outcome including independent ambulation. Prediction of recovery, an important aspect for treatment planning, has been reported to be quite accurate when made by physical therapists on the basis of a structured evaluation at entry to rehabilitation [20]. In chronic stroke subjects, static strength of lower extremity muscles is positively correlated to gait velocity [21,22] while clinical measures of motor control and balance best predict speed, independence and appearance of gait performance [23]. The Fugl-Meyer leg subscore has been shown to be correlated to gait velocity in chronic hemiparetic subjects [14] and has also been used to predict gait velocity at 6 weeks post-stroke [24].

3. Sensitivity of measures to recovery

One can question whether clinical measures are sensitive enough to document the full range of recovery post-stroke. Fig. 1 compares the measurement of recovery over time post-stroke in a group of 14 subjects. As shown in the figure, both the Barthel Index and Fugl-
RECOVERY (n=14)

Fig. 1. Mean (±1 S.D.) recovery profiles over time (w = weeks, m = months) of selected spatiotemporal variables and clinical measures in a group of 14 hemiparetic subjects with a lesion of the middle cerebral artery. Significant changes (analysis of variance for repeated measures followed by a post-hoc Scheffé test) from baseline (B = about 1 week post-stroke) or 6-week evaluations indicated by an asterisk. Changes between other evaluations indicated by horizontal line with an asterisk.

Meyer scores which represent the achievement of specific motor tasks, plateau early during recovery. Gait velocity, on the other hand, remains sensitive to change after 3 months post-stroke. The figure also illustrates that increases in gait velocity with time post-stroke are related to both longer strides and a faster cadence. These findings imply that if either of the clinical measures were chosen as outcome measures to determine change, re-
covery occurring later would not be discerned. It is also doubtful if such clinical measures would be sensitive enough to detect change in efficacy studies.

In the acute phase post-stroke, clinical measures are particularly important because they allow the evaluation of subjects even when they cannot walk or need much assistance to walk. In some cases when walking capacity is low, clinical scores may be more informative and discriminative than gait velocity. Fig. 2 (from Richards et al. [25]) compares the use of three clinical scores and gait velocity to measure recovery in nine subjects, three requiring bilateral arm support to walk (group Ia) and six who could walk with only one arm support (group Ib). Note that gait velocity is similar in groups Ia and Ib and does not discriminate between the different support needs of the two groups. The clinical measures, on the other hand, clearly show that the subjects in group Ib have higher scores for all three clinical measures. In Fig. 3 (from Richards et al. [25]), the two groups of subjects depicted in Fig. 2 are combined into one group (group I) and compared to two other groups of subjects. The groups were determined on the basis of a cluster analysis of the gait velocities (range = 12–65 cm/s) for the 18 subjects, all at 6 weeks post-stroke. Thus, subjects in group I had a mean gait velocity of 15.7 (±2.9, n = 9) cm/s, those in group II, 32.8 (±3.1, n = 6) cm/s and those in group III, 60 (±5.0, n = 3) cm/s. Note how the clinical scores associated with the gait velocity are similar for groups II and III, indicating that they are unable to discriminate the difference in gait velocity between the groups. The findings summarized in Figs. 2 and 3 thus suggest that clinical scores are more informative when the gait velocity is very low and especially if the subjects require support to walk. Gait velocity, on the other hand, should be used when the velocity of the hemiparetic subjects approaches 50% of the gait velocity of normal subjects walking slowly.

The choice of outcome measure (the measure that is expected to reveal change) is also vital to the evaluation of therapeutic efficacy. This measure must be valid, reliable and sensitive to change. Because gait velocity is more sensitive to the full range of change over time in locomotor recovery than selected clinical measures, it is not surprising that gait velocity is widely used as an outcome measure. It can be easily measured in the clinical setting by asking the patient to walk over a known distance and timing the time taken with a stopwatch, or by more complex systems [26–30]. The inter- and intrarater reliability has been established for the various ways of measuring gait velocity. In terms of validity, gait velocity actually measures one of the main goals of persons with disabilities, to walk faster (for reviews, see Malouin [31] and Wade [32]). In addition, gait velocity in subjects with hemiparesis is related to stages of motor recovery of the lower extremity [16,24,33], it is positively related to strength of lower extremity muscles [22,23] but not correlated to spasticity of the thigh muscles [23,34]. Moreover, Olney et al. [35], have shown that gait velocity in patients with hemiparesis is positively correlated to the magnitude of the push-off power burst of the hemiparetic limb.

Gait velocity, however, does not give an indication of the quality of the movements although ‘normalization’ of movement patterns is usually the therapeutic aim. Richards et al. [25] have provided data to support gait velocity as a predictor of improved locomotor movements. Fig. 4 compares the profiles of the ankle movements and activations of the triceps surae and tibialis anterior muscles during gait in a group of 18 hemiparetic subjects divided into three groups according to
their walking velocity (same subjects and subgroups as for Fig. 3). It is clear that the subjects who walked fastest (C) had movements and muscle activations closer to those of able-bodied subjects than those who walked slowest (A) or at an intermediate speed (B). Moreover, Pearson correlation coefficients calculated for a subgroup of 14 of these patients indicated that speed, cadence and stride length were all significantly and positively correlated with the magnitude of the main activation bursts in the tibialis anterior and triceps surae muscles during walking [16]. Higher gait velocity, at least in these patients, is thus related to more 'normal' muscle activations and lower extremity movements.

Because most clinical studies have described recovery as occurring mainly in the first 3 months post-stroke with a plateau reached at about 6 months post-stroke [2,12,13,16], the assumption has followed that interventions should be initiated early when the system is most receptive to change. This has been supported by studies in animals (for reviews see [36,37]). Consequently, physical therapy administered soon after stroke is becoming usual practice [19,24]. Little is known, however, about the ideal duration for intensive physical therapy. In an acute hospital, therapy is usually less than 6 weeks [24], after which the subject is transferred to a rehabilitation unit, an extended care unit or discharged. Although the duration of the stay in a rehabilitation unit is variable, the average length of stay is about 2 months in the Quebec City region. Thus in this region, the average stroke subject will receive about 3-4 months of active rehabilitation on a daily basis. If discharged to the home, few subjects will continue to receive therapy. Unfortunately, little is known about the optimal duration of active rehabilitation, the maintenance of therapeutic gains over time or of therapeutic regimes to encourage carryover of therapeutic gains.

Evidence of locomotor recovery when quantitative measures are used (gait velocity and muscle activations) after 6 months post-stroke [16] and reports of locomotor recovery in the chronic phase of subjects receiving
Bobath [38] type of physical therapy [30,39–42] cautions against assuming that recovery is complete at the end of the acute phase. Whether this later recovery occurs because patients fail to attain their full recovery potential during their early rehabilitation, because they become de-conditioned or, in fact, are continuing to recover gradually is not known at present. Since recovery follows a continuum and changes are not always perceptible, therapy may activate or accelerate the process so that change becomes measurable. We do know that at first recovery is rapid, but then slows down markedly so that it may appear that recovery is complete. Then after some time the patient may ‘step’ up to a higher level of performance as basic skills are re-acquired and integrated. We know that patients with greater impairment at onset require more time to achieve their full potential [40,43]. The fluctuations in the rate and extent of recovery make it impossible to discern the effects of a given therapeutic intervention from spontaneous recovery or other confounding variables unless the study design controls for factors such as chronicity, age, impairment, type of lesion and time after stroke. Although the need for a control group is recognized for studies in the acute phase, many studies assume that the recovery is complete (when it may not be) in the chronic phase and use test, re-test designs without the benefit of a control group [39,41,42]. Alternatively, one may argue that single-subject designs should be used to effectively analyse the disturbed motor control and degree of change with therapy in each patient [44].

4. How soon after stroke do subjects walk?

Perhaps the best answer to this question is provided by the recently published Copenhagen Stroke Study [19] that describes locomotor recovery in a total of 804 unselected stroke subjects from onset (1 week post-stroke) to the end of the rehabilitation phase. This study assessed walking function weekly by means of the Barthel Index [7] score for walking which provides three classifications: no walking function (Barthel subscore of 0 or 5 points), walks with assistance (10 points) and independent walking function (15 points). It thus provides a locomotor recovery profile in a large heterogeneous population of stroke subjects. At 1 week post-stroke, 51% had no walking function, 12% could walk with assistance and 37% could walk independently. As expected, the time course of recovery was influenced by both the degree of paresis (measured with the Scandinavian Stroke Scale [45]) and the severity of the walking disability. Jorgensen et al. [19] reported that maximal recovery (defined as a plateauing of the Barthel Index score), occurred in most by 11 weeks post-stroke but that for 80% of the subjects this plateau was reached within the first 5 weeks post-stroke. They also found that at the end of rehabilitation (median duration: 35 days), 64% of survivors had independent walking function, 14% required assistance and 22% were unable to walk. In this case, even after completion of their rehabilitation, these subjects were still in the acute phase of recovery, which may explain why previous studies that include more chronic subjects, have reported that 50–80% of stroke survivors achieve independent gait [13,29,46] and that 64% of those initially dependent in walking regain their independence [47]. Because most of the subjects in the Copenhagen study received rehabilitation based on the Bobath approach (see below), it is impossible to separate natural recovery from the effects of therapy. Although one may question the sensitivity of the Barthel Index score to the change in locomotor status once independent walking is attained, the results of this study are helpful. They provide important information in the early stages of recovery in the pre-independent gait phase, when clinical measures are more likely to be more sensitive to change, than gait velocity [25]. Thus walking status (supported or not, type of assistance) is informative of the pre-independent gait stage while gait velocity is more useful in the independent gait phase.

Friedman et al. [48] have established that subjects unable to walk at a velocity of 15 cm/s without human assistance are too dependent to live alone or in rest-homes. The type of walking aid used is related to walking velocity, with those using a quadrupod walking slowest [18]. Although some subjects remain very slow walkers, most improve so that the average gait velocity in more chronic subjects is faster. For example, Olney et al. [35] reported that the mean gait velocity of a group of subjects who had been treated in a rehabilitation unit and could tolerate 1.5 h of intermittent walking could be subdivided into three subgroups on the basis of their self-selected walking speeds: fast (mean = 63 ± 0.08 cm/s), medium (mean = 41 ± 0.08 cm/s), and slow (mean = 25 ± 0.05 cm/s).

5. Physical therapy post-stroke

Physical therapy is presently undergoing dramatic evolution from a clinical profession to a clinical science as more and more physical therapists obtain graduate degrees and engage in research activities. Current therapy is thus a combination of the old and the new. The therapeutic approaches for stroke subjects, developed 30–40 years ago [4,38,49,50] were largely based on the physiology of the pre-1950 era using the decerebrate cat model, emphasized the role of spasticity in the movement disorder and were dominated by techniques to inhibit spasticity or to use reflex activity to induce movements [51–53]. These facilitation models of therapy evolved from the muscle-oriented functional approach [54] of the post-war and polio eras. Gordon [53], outlined some common assumptions underlying these
so-called facilitation techniques: (1) the brain controls movements but not muscles; all the approaches assume that CNS lesions lead to disturbed patterns of movements rather than paralysis or weakness and that abnormal movement patterns are the direct result of the lesion rather than occur as compensations for the lesion, (2) sensory stimulation will facilitate and help re-organize correct movement patterns, (3) abnormal patterns of movement are a result of lack of inhibitory control from higher centres, and (4) the primary source of motor control deficits are due to neurophysiological rather than biomechanical or muscle factors. More recently, Carr and Shepherd [55] introduced the motor learning approach that takes into account current concepts of neural plasticity, biomechanics and motor learning. This approach has received much favour from academics because it responds to a need to develop rehabilitation strategies in light of current knowledge. It also has the appeal that it does not limit therapists to certain manually imposed movements but instead encourages innovation, the use of the environment and subject participation.

The physical therapy approach of the mid-1990s can be expected to be a product of educational background, clinical practice and location. Recent studies in Sweden [56] and Australia [57] have found a combination of therapeutic approaches to be most commonly used. When only one approach is mentioned, the Bobath [38,38] approach continues to be the most widely applied. The motor learning approach [55,59], which is still relatively new, is rapidly gaining the support of more recent graduates because it builds on current scientific knowledge. It is likely that practice in North America is similar, with regional differences related to selective influence of one approach over another. In Britain, on the other hand, the Bobath approach, with its specialized teaching centre in London, can be expected to be the approach of choice.

With the world-wide financial crisis in health care systems, and the emphasis on outcome studies to support therapeutic efficacy, one can expect dramatic changes in the future as practice will be guided by the results of these studies. Caution must be exerted, however, because outcome studies often describe but do not explain. For example, when gait velocity is the outcome measure used to determine if one therapy is better than another to promote locomotor recovery, it is important to remember that gait velocity is a descriptive variable that does not explain 'why' one group of patients walked faster than the other. To answer such a question requires a gait laboratory capable of measuring ground reaction forces and EMG activity plus analysing muscle moments and powers during walking. We know that walking speed is a function of three power bursts at the ankle, knee and hip in late stance and early swing, the A2, K3 and H3 bursts [60] and that these power bursts may be deficient in different ways in stroke patients [35,61]. Such kinetic measures should thus provide the answer as to why some subjects walk faster, and in addition, give information on the different strategies used for the generation of the necessary power to walk. This information is critical to the choice of therapeutic strategies. The danger is that with budget restrictions, granting agencies will reduce funding for relatively expensive laboratory studies and consequently limit the development of scientific theory to support new therapeutic strategies. On the other hand and more importantly, one must question the much higher human and material costs of inappropriate treatment strategies.

One can question whether a specific physical therapy approach is better than another to promote locomotor recovery post-stroke. This is not an easy question to answer for many reasons. First, it is unethical in our society to withhold therapy in order to provide a control group. Thus it is impossible to discern the effects of natural recovery from the effects of various interventions. Secondly, since the impairment and subsequent disability of each stroke victim is unique and the prognosis for recovery different, the research methodology needed to try to account for these inherent differences is very complex when comparing groups of patients receiving different therapies. Thirdly, it is difficult to control for therapeutic factors such as type, intensity and time post-stroke as well as quality of the patient-therapist interaction. Despite these difficulties, only properly designed studies that include appropriate controls have the potential of revealing the effects of therapy. Since spontaneous recovery interacts with therapeutic interventions, it may be more fruitful to examine how best to combine therapy with natural recovery processes rather than try to separate the effects of one from the other.

Despite the fact that facilitation techniques have been used for nearly 40 years, evidence of long-term effects based on controlled studies is still lacking. Immediate effects, however, of many of the facilitation techniques can be readily demonstrated in the clinical setting, and in some [62], but not in other [63] experimental studies. Several clinical studies have attempted to demonstrate the superiority of one of the facilitation models over the functional training model [64–68] or one facilitation model over the other [67,69] to promote recovery without specifically keying on locomotor recovery. In all of these studies the patients improved but there were no significant differences among the different intervention strategies. A critical analysis of the above-mentioned studies, however, reveals a number of methodological flaws which may have biased the results or prevented the finding of a true difference. One must also doubt that the outcome measures and their sensitivity to change were appropriate. It is thus not surprising, given the lack of strong evidence based on studies with appropriate designs with control groups carried out in a sufficient
number of subjects, that Reding and McDowell [70] stated that 'there is no evidence that current rehabilitation methods affect the natural recovery of motor, sensory, coordination, or visual deficit following stroke. They do allow the patient to cope with residual deficits more effectively and with greater independence.' On the basis of a meta-analysis of 36 clinical trials (between 1960 and June 1990) that investigated the effectiveness of stroke rehabilitation programs to improve functional outcomes and discharge destination, Ottenbacher and Jannell [71] supported this conclusion and further found that improvement in performance appears to be related to early initiation of treatment, age and study design but not to duration of the intervention.

Because the motor control model is relatively new, few clinical trials have attempted to demonstrate its efficacy. Malouin et al. [72], proposed a method of gait training of hemiparetic subjects that was inspired from the motor control model. The training began at the end of the first week post-stroke and was provided for 5 weeks in an acute hospital setting. Subjects were encouraged to walk early and gait pre-training activities included standing on a table and weight shifting (monitored by a limb-load feedback system), walking in parallel bars with an air splint to prevent knee buckling, reciprocal leg movements against isokinetic resistance (Kinetron) and walking on a treadmill. The specialized treadmill, capable of very slow speeds, was equipped with a harness that was connected to a hook device and capable of holding the patient upright. The harness was used only for safety and did not support the patient. In the early phase, walking was guided manually by the therapist. In addition to the specialized gait training, the subjects also received the Bobath [38,58] type of conventional therapy, especially for the paretic upper extremity. The patients responded favourably to this approach and tolerated the procedures well.

Malouin et al. [72] were thus able to demonstrate the feasibility of such an intense (1.75 h/day × 5 days/week), task-oriented training approach provided early post-stroke. The efficacy of this new therapeutic strategy was also compared to conventional therapy of the Bobath type in a pilot randomized controlled clinical trial. Richards et al. [24] reported that after 5 weeks of training, subjects in the experimental group walked on average 41% faster than the control subjects and that the difference in gait velocity (chosen as the primary outcome measure) translated into a moderate effect size of 0.58. Since the effect size [73] measures the difference between two groups in terms of a common standard deviation, an effect size of 0.58 indicates that 58% of a standard deviation separated the average subjects in the two groups. Another important finding was that when the subjects in the experimental and control groups were pooled, gait velocity was positively correlated to actual time spent practising gait or gait-related activities but not to total therapy time, indicating a task-specificity effect (Fig. 5, reproduced from Richards et al. [24]). The advantage in gait velocity gained by the patients in the experimental group by 6 weeks after stroke was, however, not maintained at 3 months after stroke. At present it is not known whether this was due to a detraining effect because patients returned to conventional therapy after the 6-week trial, to factors such as therapy cessation before the full potential was reached, to motivational or other factors not yet identified. Nevertheless, the results of the pilot study evaluating this new approach were considered promising enough to warrant a full scale clinical trial in a cohort of subjects who were more heterogenous in terms of brain lesion, age and disability to test its efficacy under conditions of usual therapy intensity and duration (1 h/day × 5 days/week × 2 months). This trial is presently in progress with gait velocity as the primary outcome variable.

Because gait velocity does not provide information as to 'why' the patient walks faster, secondary outcome measures include static strength of six muscle groups of the paretic lower extremity, and the movement, muscle activations, moment and power profiles of both the paretic and non-paretic lower extremities during gait. The push-off power burst of the plantarflexors and the pull-off burst of the hip flexors on the paretic side are of particular interest [35,61] because of their importance in the power generation needed for walking. It is expected that the hemiparetic subjects will use compensatory power generation strategies.

5.1. Type of disturbed motor control and choice of therapy

The classification of types of disturbed locomotor control in hemiparetic subjects proposed by Knutsson and Richards [74] implies that the response to certain
therapeutic procedures is predictable if the crucial aspect of the disturbed motor control is known. Another important premise is that the disturbed control is classified from an evaluation of walking performance and not from a bedside evaluation of active or passive movements which may not reveal the locomotor disturbance. For example, patients with a Type I disturbance should respond to antispastic therapy [74,75]. Also, since premature activation of the calf is usually associated with knee hyperextension during stance, it may be possible to train the subject to rotate the pelvis forward (transverse plane) when the paretic leg is in the swing phase so that the body weight is further over the support foot at weight acceptance [44]. Subjects with a Type II disturbance are more likely to respond to strengthening techniques, in particular eccentric contractions which do not stretch the spastic antagonists [76–78], a gait relearning program [44], functional electrical stimulation [79], or orthoses. The Type III disturbance, characterized by excessive coactivations of antagonist muscles, appears to be more a programming disorder with strong muscle activations. This disorder, which is less common than Types I and II, is very resistant to medical and physical therapy [74,75]. Knee hyperextension in early, mid and/or late stance may help the clinician determine the type of muscle activation disorder [44,74,80]. For example, knee hyperextension in early stance is suggestive of a Type I disturbance while a hyperextension throughout stance or in late stance may indicate a Type II disturbance.

In chronic stroke patients, there is no doubt that gait analysis may help pinpoint the critical disturbance to guide the medical and physical therapy approaches [44,74,81,82]. In clinical practice today, however, physical therapy is initiated as early as possible and it is not unusual to begin within 1 week post-stroke [19,24]. Gait analysis is impossible in this early state since the majority of patients are unable to walk. One must then question whether early, intense and appropriate physical therapy can guide the recovery process and help prevent the development of some of the disturbed motor control described in chronic patients. Although the results of Shiavi et al. [83] and preliminary results in our laboratory [25] suggest that the Type II disturbance (lack of activation or paresis) may be the most common in the acute phase of stroke, other types of activation disturbances are also present. Shiavi et al. [83] have also provided evidence that the type of disturbed control during walking may change with recovery, thus illustrating the plasticity of the gait motor control system in the early recovery phase. They do not, however, distinguish between the effects of rehabilitation procedures and spontaneous recovery but do suggest that the Type II disorder may be more responsive than the Type III disorder as previously reported [75]. Future studies should help us to better understand the interaction among type and extent of impairment, phase of recovery and physical therapy.

5.2. Weight support

The use of treadmill gait training for subjects with hemiplegia has gained increasing favor among clinicians, first as a means of cardiovascular training [84] and more recently as described above, for early gait training. Finch and Barbeau [85] proposed the use of weight support by means of a harness while treadmill walking to facilitate walking movements by removing some of the biomechanical and equilibrium constraints of full weight bearing. Manual assistance to help weight transfers and foot placements is provided as the subject walks. The amount of weight support is reduced as the walking capacity improves. Such interactive gait-training with weight support, which has also been applied with success in paraparetic subjects, is based on work in the spinal animal (for a review, see Visintin and Barbeau [86]). Further support for such an approach comes from the literature describing the ontogeny of gait in children and the role of equilibrium and anti-gravity support in the expression of the gait patterns [87–90]. To test the effectiveness of this weight-bearing strategy, Visintin et al. [91] have recently completed a randomized clinical trial with 100 subjects with hemiparesis; 50 receiving body weight support (up to 40% of body weight) during gait retraining on a treadmill while the other 50 received gait retraining under full weight bearing only. Treatment was provided for 20 min 4 x week/6 weeks. Partial results suggest the superiority of weight support but details are lacking because the final analysis remains to be reported.

5.3. Strength training in subjects with hemiparesis

Strength training of hemiparetic subjects has long been a controversial topic. Because paresis, hyperactive stretch reflexes or abnormal coactivation of antagonist muscles alone or in combination may perturb motor control, the voluntary activation of selected muscle groups may prove to be difficult or impossible. Moreover, therapists long held the belief that strenuous exercise such as strength training or even riding an ergometer or treadmill walking could increase spasticity and thus be detrimental. Although spasticity may be the salient disorder in some patients, paresis or loss of the voluntary capacity to activate muscles has become recognized as one of the key disturbances during active limb movements [92,93] or during walking [74,83]. In subjects with spastic paresis, however, the facilitation imparted by descending voluntary pathways as well as the stretching of the antagonist muscle by the movement effected by the agonist muscle during dynamic contractions may result in 'active restraint' which reduces the net force output. For this reason, in a review of the neurophysiological mechanisms, Knutsson [76] ad-
vocated the use of eccentric contractions for strengthening in patients with spastic paresis. Thus, an eccentric contraction of the quadriceps would flex the knee joint and shorten rather than lengthen the spastic hamstring muscles. Engardt et al. [78] have recently shown isokinetic eccentric strength training to be superior to concentric training for strengthening of the knee extensors of hemiparetic subjects. Moreover, they were able to show that antagonist reflex activation did not increase and that the gain in strength is beneficial to the performance of a functional task, the act of rising from sitting. These findings suggest a new and important approach to muscle strengthening for hemiparetic subjects because weakness is a primary aspect of the motor disorder and strength of lower extremity muscles is positively correlated to gait velocity [22] and other functional activities [77]. An added attraction to the training of eccentric contractions is their importance in the control of the lower extremity movements during walking [35,60,61,94], stair climbing [95–97] sitting and other functional activities [77,94]. Although eccentric muscle strengthening requires sophisticated dynamometers, they are now present in a large number of rehabilitation facilities.

5.4. Biofeedback: an adjunct to physical therapy

Locomotor therapy of subjects with hemiparesis can be considered from the standpoint of information processing, wherein feedback of performance is an essential element of the motor learning process [98]. Theoretically, the use of feedback with practice helps the acquisition of a motor skill so that the control process gradually shifts from a closed feedback loop to an open loop control system [99]. Since the first EMG biofeedback system was introduced about 25 years ago [100] various types of artificial sensory feedback have been used to train hemiparetic subjects to improve a particular aspect of their gait pattern [101]. EMG biofeedback has been used to train the voluntary activation of weak or parietic muscles or the relaxation of spastic muscles, by presenting the amplified EMG signal in auditory or visual form. Others have used joint angle position feedback to correct knee hyperextension [102–105] or to improve loading on the hemiparetic limb [106]. In general, the use of biofeedback has given positive results and even though some of the studies had methodological errors, such as not controlling for natural recovery after stroke [107], the efficacy of feedback has been confirmed in controlled studies. In a meta-analysis designed to assess the efficacy of EMG biofeedback for hemiparetic subjects (including studies on the upper limb), Schleebaker and Mainous [108], following the analysis of the eight studies published between 1966 and 1991 that had a randomized or matched control group and a functional outcome measure, yielded an effect size of 0.81, indicating that EMG biofeedback is an effective tool for neuromuscular reeducation. Using an elegant cross-over design with eight subjects (at least 7 months post-stroke) with hemiparesis and computer assisted feedback to give instantaneous feedback while the subject walked, Colborne et al. [82] compared the efficacy of: (1) physical therapy, (2) ankle joint angle feedback and (3) EMG feedback of the soleus to improve a number of gait variables. They were able to show improvements with all three types of therapy but the improvements were largest with the EMG feedback followed by the joint feedback and lastly physical therapy. Of particular interest was the increase in the affected side push-off impulse of the plantarflexors with both the ankle angle and EMG feedback because the triceps surae is resistant to recovery [16]. This study [82] thus once again confirms the potential for recovery of subjects with hemiparesis well beyond 6 months post-stroke (average time post-stroke at study initiation was 17 months) when sensitive outcome measures are used. It also provided the background for a 100-subject randomized controlled trial [109] of the effectiveness of this computerized feedback method when used during the acute phase of treatment. Feedback was targeted to increase power generation on the affected side. The planned interim analysis showed positive trends toward greater effectiveness of the experimental group, but final results await completion of the study.

We know little, however, about the retention of skills learned with the help of feedback or of weaning strategies most likely to encourage carryover. In the Colborne et al. [82] study, the subjects maintained therapeutic gains 1 month after cessation of the feedback (both ankle position and EMG feedback). Engardt [110], in a follow-up study about 3 years after the training of symmetrical weight support when rising and sitting with auditory feedback in hemiparetic subjects, found that therapy gains were lost. One has to wonder if these gains could have been maintained with a weekly or monthly practice session or if another type of withdrawal strategy was used [111]. Available evidence clearly points to the potential of feedback to focus on specific treatment goals. The challenge is to determine application strategies that will facilitate long-term retention of therapy gains. Moreover, the specificity of motor learning with the help of feedback is highlighted by the work of Weinstein et al. [112]. They showed that improvements in the symmetry of weight support of hemiparetic subjects while standing did not carryover to walking.

5.5. Anticipatory postural adjustments and mental practice

Much interest has been focused on the role of anticipatory adjustments in the performance of motor tasks and in particular the initiation [113] and termination [114] of gait and of rapid leg flexion [115] in the last
few years. The whole concept of pre-programmed responses and their potential use to promote locomotor recovery after stroke is at present a new and exciting area [113,115,116]. Once again, the experimental work on anticipatory postural responses emphasizes the importance of task-specificity and context. For therapists, the implications of pre-programmed responses are enormous and represent a dramatic change in approach wherein the emphasis shifts to eliciting a motor program rather than attempting to modify the output so that the subject will learn a more appropriate movement pattern.

The use of mental practice to promote the acquisition of skills is gradually becoming an accepted part of the routine of elite athletes (for a review, see Feltz and Landers [117]). Evidence from various fields suggests that mental practice and the physical performance of motor skills at least in part use the same neural structures for the planning and programming of motor actions. The potential of mental practice to assist motor relearning in hemiparetic patients is essentially unexplored but the finding that the performance of mental practice of an arm movement in hemiparetic subjects elicits changes in EEG behavior comparable to those obtained in healthy subjects [118] warrants further study.

6. Synopsis

Locomotor recovery after stroke is most likely a combination of natural recovery and therapeutic interventions. This review has emphasized the need to better understand the interrelationship between time post stroke, recovery potential and physical therapy. In the acute phase when the ambulation capacity is limited, it is assumed that physical therapy will benefit most patients. As this review shows, however, there is an urgent need to develop new therapeutic strategies to guide and promote locomotor recovery in this acute phase, especially in the first 6 weeks after stroke. To encourage clinicians to test the efficacy of therapeutic approaches, it is imperative to use sensitive outcome measures, such as walking velocity. Ideally, an in-depth gait analysis could be used to guide the therapy as soon as the patient attains independent gait and has the required endurance. Such analyses, however, require sophisticated and costly laboratories that are not available to most clinicians. Consequently, it is important to provide practical and less costly guidelines to help clinicians customize their therapeutic approach to individual patients. A clinical analysis of the locomotor impairments, adaptations and compensations such as outlined in part I of this review may help pinpoint primary disorders. It is also important to further our understanding of the kinetic variables that, for example, determine the speed of walking in order to provide the link between a descriptive outcome variable and the kinetic determinants that may be used to guide therapy. Finally, there is a need to incorporate learning theory principles to optimize the success of therapeutic strategies for locomotor disorders. The potential of open-loop feedforward strategies to initiate pre-programmed locomotor components or the use of mental practice to facilitate locomotor recovery remains to be confirmed.

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