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Weakness and strength training in persons with poststroke hemiplegia: Rationale, method, and efficacy

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Abstract—Several converging lines of contemporary evidence suggest that weakness presents a more serious compromise to movement function in poststroke hemiplegia than spasticity. This review examines the clinical and functional phenomena of weakness in poststroke hemiplegia, currently available evidence identifying physiologic substrates contri-buting to weakness, and reports of early investigations involving high-resistance training targeted at improving strength and the transfer of strength to improvements in functional capacity. Based on this information, we describe some unsolved problems and indicate some likely lines of development to increase our knowledge regarding how resistance training can be included in effective stroke rehabilitation.

Key words: adaptation, physiological; cerebrovascular accident; evidence-based medicine; hemiplegia; muscles, skeletal; muscle weakness; recovery of function; rehabilitation; treatment outcome.

INTRODUCTION

Stroke is a leading cause of long-term disability in the Western world, with a prevalence of approximately 900 per 100,000 persons. Over 600,000 new cases of stroke, or cerebrovascular accident (CVA), occur in the United States each year and over 25,000 in Sweden, accounting for more than half of all acute inpatient neurological hospital admissions and over US$30 billion in costs for healthcare and lost productivity. Because of remarkable improvements in the acute management of stroke, the majority of persons now survive and recover, experiencing only a modest decrease in life expectancy [1].

Abbreviations: ADL = activity of daily life, CSA = cross-sectional area, CT = computed tomography, CVA = cerebrovascular accident, LBM = lean body mass, MRI = magnetic resonance imaging, 1-RM = one repetition maximum, PRT = progressive resistance training, SSWS = self-selected walking speed.

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Over the last decade, the number of stroke survivors has increased 30 percent, such that worldwide, we now experience the largest cohort of persons surviving stroke in history. Currently, the United States has over 3.5 million stroke survivors, and this number can be expected to continue to increase. These persons return home, and often to work, with expectations to resume their daily activities and assume their normal social roles. Because the incidence of stroke doubles with each decade beyond 60 years of age, we have grown to consider stroke a problem of an aging population. It is thus significant that the incidence of stroke has increased dramatically in younger individuals, such that at least 20 percent of stroke survivors are less than 65 years of age [2]. This changing landscape for persons surviving stroke underscores the critical importance of providing effective rehabilitation with the potential to optimize recovery of function, minimize long-term disability, and enable reintegration and participation in meaningful activities of daily life (ADL).

The sequelae of stroke are multifactorial and depend heavily on the mechanism, extent, and location of the vascular lesion. The primary concern addressed in physical rehabilitation is restoration of the requisite motor function to perform the myriad of tasks encountered in daily life. These tasks range from grasping, reaching, and manipulation to more physical demanding transitional movements and complex coordinated movements, such as locomotion. Common to these motor tasks is control of muscular force, which becomes compromised with central nervous system damage and manifests as impaired intersegmental coordination, hyperreflexia or spasticity, and unilateral weakness [3].

Many of the traditional perspectives on neurorehabilitation held that, of these motor sequelae, spasticity presented the most significant limitation to recovery of normal motor function. Moreover, because physical exertion was clinically observed to exacerbate spasticity, therapeutic activities using forceful contractions became strictly proscribed for persons with nervous system injury. One prominent approach to treatment of adult hemiplegia thus centered on the concept of managing muscle hypertonia [4], while the general goal of neurorehabilitation treatment approaches evolved to focus on improving control, and especially the quality, of movement. Interestingly, however, companion meta-analyses that examined the effects of commonly used interventions for rehabilitation of both the upper and lower limb in poststroke hemiplegia reported a lack of compelling evidence that any of the existing approaches to neurorehabilitation have demonstrated superior efficacy for promoting recovery of motor function [5,6].

In stark contrast, currently emerging evidence suggests that weakness may be directly responsible for compromised motor function [7–9]. This premise has motivated research demonstrating that neither effortful activities nor strength training, per se, exacerbate spasticity (**Table 1**) [10–16]. Positive effects of resistance exercise have been demonstrated in persons with poststroke hemiplegia, and in some cases, concomitant influences on performance of functional tasks have been observed. Taken together, these indices suggest that high-intensity activities, including resistance training, could form an important component of rehabilitation programs for persons with poststroke hemiplegia.

In this review, we—

- Present evidence that pervasive weakness in poststroke hemiplegia contributes to significant functional consequences.
- Review the evidence pertaining to high-intensity and resistance-training activities and their potential for rehabilitation of poststroke hemiplegia.
- Report clinically and functionally important improvements associated with resistance training.
- Describe some unsolved problems.
- Indicate some likely lines of development to increase our knowledge regarding how to effectively include resistance training in stroke rehabilitation.

CLINICAL PERSPECTIVES ON MUSCLE STRENGTH AND WEAKNESS IN POSTSTROKE HEMIPLEGIA

The capacity to produce muscle force, or strength, involves—

1. Structural factors, i.e., muscle size: Muscle mass or cross-sectional area (CSA), which depends on the number, size, and relative proportions of muscle fiber types.
2. Mechanical factors, including the length-tension and force-velocity relationships of muscle.
3. Neural factors, i.e., the capacity of the nervous system to activate muscle through motor unit recruitment and rate coding.

Compromise to any of these factors affects the capacity to exert force and comprises the operational definition of weakness. Weakness is a prominent finding in a variety of

Table 1.
Effects of exertion on spasticity/hypertonia.

Citation	Population	Prescription	Strength	Mode of Measurement	Effect on Spasticity
Fowler, 2001 [10]	24 children with spastic diplegia, 12 controls	Isometric, isokinetic, and isotonic exer to quadriceps; 5 reps of each type of exer were performed, isokinetic (at 60°/s) and isometric exer on Kin-com, isotonic using cuff weights	No strength measured	Pendulum, measured by swing excursion, number of lower leg oscillations, duration of oscillations	No differences in swing excursion, lower leg oscillations, or duration of oscillations before/after for either group (differences did exist between control group and children with diplegia both pre- and postexer)
Miller & Light, 1997 [11]	2 mo to 6 yr since stroke, N = 9	Isometric 10 reps at 25%, 50%, and 75% of MVC	No strength measured	Modified Ashworth, Percentage of EMG cocontraction during quick isometric contraction of biceps	No change in Modified Ashworth pre- and postexer. Time on for biceps w/o triceps incr in posttest for P-exer, P-no exer and NP-no exer (a greater incr was seen for P-exer than P-no exer). Time on for biceps w/o triceps decr for NP-exer.
Brown & Kautz, 1998 [13]	15 persons w/ chronic stroke, and 12 healthy aged-matched controls	Pedaling at 12 randomly ordered workload (45, 90, 135, and 180 J) and cadence (25, 40, and 55 rpm) combinations	No strength measured	Measurement of mm activity (EMG) during quadrants of pedaling cycle	W/incr speed and workload, P-leg total integrated EMG incr w/o a greater incr in EMG activity during inappropriate quadrants of the pedaling cycle (P-leg mm did show overall greater percentages of activity during inappropriate quadrants when compared to controls)
Sahrmann & Norton, 1977 [14]	Persons w/ upper motor neuron lesions of 2 mo to 15 yr duration, N = 16	Surface and indwelling EMG (selected Ss only) recorded from biceps, brachioradialis, and triceps during (1) passive elbow ROM, (2) maximal isometric flex & ext, and (3) voluntary repetitive elbow flex/ext at both slow and rapid paces	No strength measured	EMG during passive movement and maximum isometric elbow flex/ext	Qualitative analysis of EMG activity during isotonic movement reveals that prolonged recruitment and delayed cessation of agonist (rather than exaggerated antagonist stretch reflex) is the primary limitation to movement
Bohannon et al., 1991 [15]	Acute, N = 23	Elbow flex strength was measured using a hand-held dynamometer	Average P-elbow flex force was 13.6 ± 10.5 kg	Modified Ashworth Scale	Correlations were significant between hand-to-mouth scores, impaired mm strength, and movement deficits ($p < 0.01$). No correlation existed between hand-to-mouth scores and elbow-extensor mm tone.
Sinkjaer & Magnussen 1994 [16]	9 persons with spastic hemiparesis, 8 controls	None	Average MVC in P-leg was 23%, in NP-leg 65% relative to MVC in healthy Ss	EMG from soleus mm with ankle stabilized in neutral. Torque was measured during a stretch with and without presence of a stretch reflex in extensor mm.	Passive stiffness of the P-leg was incr by 278% and NP-leg was incr by 95% compared with healthy controls. No difference in intrinsic stiffness. Reflex stiffness at upper edge of normal for P-leg.

Note: See main paper reference section for detailed references.
decr = decrease, EMG = electromyography, exer = exercise, ext = extension, flex = flexion, incr = increase, mm = muscle, MVC = maximum voluntary contraction, NP = nonparetic, P = paretic, ROM = range of motion, rpm = revolutions per minute, Ss = subjects

central and peripheral neurological disorders, as well as aging, all of which are conditions involving immobilization or markedly decreased physical activity and all of which typically involve other systemic clinical conditions.

Weakness following stroke is referred to as either hemiparesis—mild to moderate degree of weakness—or hemiplegia—severe or complete loss of motor function on one side of the body. However, evidence is now emerging that weakness also occurs on the "uninvolved," or ipsilesional side (traditionally termed the "nonparetic"), within a short time frame postacute stroke [17]. In the literature, poststroke weakness has been described not only as impaired force magnitude [18] but also as a more broadly defined phenomenon, including slowness to produce force [19,20], a rapid onset of fatigue [21], an excessive sense of effort [22], and difficulty with producing force effectively within the context of a task [23]. Throughout this review, we use the term "poststroke weakness" to include all aspects of weakness following stroke.

Cocontraction of antagonist muscles has also been posited to impair force magnitude, rate of force production, and intersegmental coordination by acting as an "antagonist restraint" [24]. However, contemporary investigation has failed to produce evidence of significant antagonist cocontraction during movements. Rather, significant impairment of agonist activation has been demonstrated in the paretic limb [8,14,24,25]. Such observations lead predictably to questions of whether and how agonist activation can be improved and whether such improvement in physiologic function leads to clinically and functionally important differences in motor performance.

Classical Perspective—Spasticity/Hyperreflexia in Poststroke Hemiplegia

The upper-motor neuron syndrome as described by John Hughlings Jackson involves a combination of negative signs—weakness and impaired dexterity or coordination—and positive signs—spasticity or hyperreflexia [26]. Early approaches to neurorehabilitation emphasized treatment from the perspective of diminishing positive Jacksonian signs and focused on techniques to normalize tone, facilitate normal patterns of movement, and decrease cocontraction of paired antagonist muscles [4]. A fundamental tenet of this perspective was the broadly held belief that intense, effortful, or high-exertion activities exacerbate hypertonia and reinforce aberrant motor pathways. Effortful activities were thus proscribed in the therapeutic regime for neurologic patients.

Contemporary Perspectives

A substantial body of evidence now exists to demonstrate that exaggerated resistance to passive movement, traditionally termed "spasticity" or "hypertonia," involves changes in the passive mechanical properties of the muscle-tendon complex [12,16]. These muscle and tissue changes may be more profound than either changes in the reflex threshold or alterations in intrinsic motor neuron excitability, which traditionally were believed to cause hyperactive stretch reflexes [27]. Indeed, such changes in passive tissue properties may have a compensatory role and may possibly simplify movement control or optimize compromised motor function [28]. Moreover, multiple investigators have now soundly refuted the fundamental tenet that effortful exercise exacerbates spasticity (see **Table 1**). In addition, a recent investigation of associated reactions in the ipsilesional limb demonstrated no consistent relationship between the presence of associated reactions and either the degree of hypertonia or the weakness [29]. Taken together, such observations shift the focus away from spasticity toward weakness as a prominent problem corresponding with motor compromise in poststroke hemiplegia. Such a marked change in the scientific perspective regarding motor impairment in poststroke hemiplegia motivates a redirection in the emphasis of activities used in neurorehabilitation.

DISTRIBUTION OF POSTSTROKE WEAKNESS

The distribution of poststroke weakness has been described following various investigations [18,30]. Adams and coworkers assessed 20 patients with moderate to severe hemiplegia and found that the mean degree of strength in the involved limb varied from 23 to 94 percent of that on the ipsilesional side [18]. The average degree of weakness showed that the residual strength was 37 percent for ankle plantar flexion, 45 percent for ankle dorsiflexion, 51 percent for knee extension, 53 percent for knee flexion, 64 percent for hip extension, and 68 percent for hip flexion, indicating that following stroke, weakness is more pronounced distally than proximally. Bohannon and Andrews studied bilateral isometric strength in 48 hemiplegic persons across eight muscle actions [30]. Most of the strength measures correlated significantly with one another, indicating that poststroke weakness demonstrated in one muscle action will reflect weakness in other muscle actions. These findings were

extended by Sunnerhagen et al. who investigated 16 subjects with minor motor impairment following a stroke [31]. They found that isovelocity torque in the paretic leg was reduced 9 to 29 percent relative to the ipsilesional leg. While attention in poststroke hemiplegia generally focuses on paretic limb weakness, recently reported findings describe significant weakness in the ipsilesional side as early as 1 week following stroke [17]. Poststroke weakness thus appears to correspond to the severity of the stroke and is a relatively consistent phenomenon within and between paretic limbs. Importantly, however, the recent evidence that the ipsilesional side is also significantly affected by stroke suggests that weakness in the paretic side reported relative to the ipsilesional side may be considerably more profound than has been previously appreciated.

FUNCTIONAL CONSEQUENCES OF POSTSTROKE WEAKNESS

Poststroke hemiplegia is associated with significant impairments of motor function that are believed to compromise ADL performance and lead to loss of independence. However, a direct causal relationship between strength or weakness and motor function has not been established. Traditionally, a strong bias has existed against quantifying strength in hemiplegic persons. As a result, the majority of clinical research in this population has focused on outcome measures at the activity and participation levels [32]. Despite this, several available reports correlate strength with various functional activities, such that taken together, evidence strongly suggests impaired strength may play a prominent role in compromised functional performance.

Bohannon and Andrews [33] observed that gait performance in 17 hemiparetic persons was significantly correlated with knee extensor torque ($r = 0.57$, $p < 0.05$) but not with spasticity and further that knee extension muscle performance measured either isometrically or isokinetically correlated significantly with gait velocity [20]. Nakamura and coworkers also observed that spasticity was unrelated to locomotor impairments [34,35], rather that isokinetic knee extension strength in the paretic limb was strongly associated with self-selected walking speed (SSWS). Lindmark and Hamrin observed a moderate relationship between SSWS and either motor scores or knee extension torque, which improved in predictive power when examined in a multivariate statistical model [36]. Pohl and coworkers observed that the combination of peak isometric knee extension force and rate of force acquisition explained a significant 12 percent of variance related to gait speed in hemiparetic adults [37]. They further observed that elimination of peak torque did not significantly affect the model predictions, while eliminating the rate of force acquisition did reduce its predictive power. Similarly, Davies and coworkers found that SSWS correlated significantly with maximal paretic-leg knee extension velocity and was not associated with antagonist muscle cocontraction [25]. In a sample of highly functioning hemiparetic persons, Nadeau and coworkers found a significant relationship between hip flexor strength and SSWS [9]. When plantar flexion strength was added to the model, its explanatory power increased such that it became possible to predict maximal gait speed [38].

Important strength-function relationships have been evidenced in other motor activities as well. Suzuki et al. found torque of the affected leg related to stability and postural sway [39], while Bohannon and coworkers found that isometric strength of the elbow flexors corresponded with performance of three separate hand-to-mouth maneuvers [15]. Similarly, Boissy and coworkers observed that maximal grip strength was highly correlated with multiple indicators of upper-limb impairment and function while Engardt and coworkers observed that deficits in knee extension torque were related to the asymmetry of bodyweight distribution between paretic and ipsilesional limbs during sit to stand [40,41].

Each of the investigations just described focused on isolated muscle groups or actions. However, functional movement involves simultaneous activation and coordination of multiple muscles. This disparity may contribute in part to failure to demonstrate a direct relationship between strength and function [42]. Nonetheless, as was discussed earlier, weakness is similar among muscles on the paretic side [30]. Thus, measurement of a single key action such as grip strength for the upper limb [40] or knee extension for the lower limb [29] may be considered a representative surrogate for quantifying motor impairment and disability. Also noteworthy is that task-dependent strength deficits have been observed, such that coordination of multijoint activity or production of force in more than one direction leads to widely varying deficits of strength in a particular muscle [23]. Little published work is available in this regard, but this topic holds potential for greater insight

regarding the nature of the relationship between motor impairment and poststroke weakness.

When strength-function relationships are considered, it is important to recognize that the predominant clinical perspective defines strength as force magnitude, which is usually assessed under isometric conditions with the use of manual muscle tests, hand-held myometry and, in rare cases, force transducers. Nominally, observations of dynamic torques would provide a more detailed description of neuromuscular performance and improved opportunity to relate this aspect of strength to functional performance. To observe torque production under dynamic conditions requires a dynamometer and considerably more time and effort than is typically available in the clinical setting. The value of such measurements is, however, significant as they afford considerably more information regarding specific aspects of motor performance that may become impaired in hemiplegia and, further, may be more strongly associated with functional task performance and/or as indicators of progression through different stages of motor recovery. For example, Dvir and David report an indicator of suboptimal muscle performance that is highlighted only by evaluating differences in the ratio of eccentric to concentric torque between high and low movement speeds [43]. As was presented earlier, increasingly dynamic aspects of force production, including rate of force production [19,37], movement speed [36], and power [44], are included in investigations seeking to understand relationships between strength and functional performance. The reliability of dynamic measures of strength has been examined and, in general, established in hemiparetic adults [45,46]. A number of methodological issues remain pertaining to measurement of dynamic motor performance in hemiparetic adults; thus investigative work in this area should continue to address issues of reliability and seek to establish the minimal effect sizes necessary to conclude that relevant and important clinical changes have occurred [47].

MECHANISMS OF POSTSTROKE WEAKNESS

Direct correlates of weakness and the physiologic mechanisms that underlie this weakness remain poorly understood in persons who have suffered neurologic insult. Because functional muscular force is the product of both muscular and neural factors, compromise to either of these factors impairs the capacity to produce and regulate force. Because of the significant supraspinal damage pathogneumonic of stroke, weakness might be directly attributed to compromised neural activation. However, inactivity and impaired muscular activation could lead to atrophy and changes in the muscle fiber population that might also readily explain weakness in persons with chronic hemiplegia.

Muscular Factors

In a study of hemiparetic persons with minor motor impairment, Sunnerhagen et al. used computed tomography (CT) and found no differences in muscle CSA between the affected and ipsilesional limbs [31]. Similarly, a recent investigation by Jorgensen and Jacobsen using dual-energy X-ray absorptiometry (DEXA) demonstrated that patients who were nonambulatory at 2 months poststroke lost only 6 percent of lean body mass (LBM) in the paretic leg, while a concurrent 5 percent loss on the ipsilesional leg was regained completely at 12 months poststroke [48]. Patients who were ambulatory at 2 months poststroke had increased LBM in the ipsilesional leg by 5 percent after 1 year, while no significant changes were found in the paretic leg at either 2 or 12 months poststroke. An increased amount of noncontractile tissue, e.g., fatty infiltration, can contribute to maintenance of gross muscle CSA and might explain the lack of difference in muscle CSA observed using low- resolution imaging methods such as CT.

Only a few studies have examined the fiber-type composition in hemiplegic muscles. Despite this, the findings are reasonably consistent and can explain a component of weakness in poststroke hemiplegia. There is predominant atrophy of Type II fibers that can be accompanied by compensatory hypertrophy and an increased proportion/predominance of Type I fibers [49–54]. Histochemical studies reveal accumulation of lipofuscin and lipid droplets and qualitative pathological changes, including nuclear internalization [54], fatty infiltration, as well as denervation and fiber type grouping—all of which indicate collateral reinnervation in response to motor neuron loss. These types of changes are not specific to stroke because they are also common findings in muscles of healthy older adults and in cases of severe inactivity [55,56]. Type II muscle fiber atrophy is observed in persons with poststroke hemiplegia [51]; however, the degree of atrophy appears to be more closely related to spontaneous daily physical activity than stroke severity, time since onset, or ADL score. Similarly, increased signs of denervation and reinnervation as

just described appear not to be associated with time since onset of illness, age, or clinical status but rather with functional mobility status.

Remarkably little information is available regarding muscle structure in persons with poststroke hemiplegia. While structural changes in muscle may occur in poststroke weakness, considerable variability can be found between individual subjects. Moreover, the available data suggest effects of immobility and inactivity more than of intrinsic neuropathic change. Because the limited available data have been obtained without benefit of the most current high-resolution imaging techniques, we lack the information to differentiate contractile and noncontractile components of muscle. Magnetic resonance imaging (MRI) is currently the preferred method for noninvasive imaging of biological tissues because it allows for clear distinction between and quantification of muscle tissue elements [57,58]. Future research using currently available imaging sequences holds promise for substantial advancements in our understanding of this area. In addition, use of more sensitive contemporary techniques to determine the significance of changes in the fiber-type composition, for example, analyses of myosin heavy chain content and contractile properties of single muscle fibres, might also contribute to an increased understanding of the muscular factors underlying weakness in poststroke hemiplegia.

Neural Factors

Motor Unit Properties

Without evidence that structural differences in muscle contribute significantly to hemiparetic weakness, attention turns to the neural aspects of strength and control of force at the motor unit level. The neural mechanisms controlling muscular force involve task-dependent motor unit activity: recruitment, rate coding of already active motor units, and the interaction of which affords the infinite gradation of muscle forces involved in motor execution [59]. Damage of brain tissue following stroke affects corticospinal and other supraspinal motor pathways and, it is thought, leads to transsynaptic degeneration at the segmental level [60]. The consequent reduction in neural traffic at the spinal segmental level results in motor neuron loss and disruption of these primary force control mechanisms. Following CVA, impairment in agonist muscle force production can thus result from several sources of compromise: frank loss of motor units [61]; increased motor unit innervation ratios resulting from collateral sprouting [62–64]; altered biophysical properties of the motor unit affecting recruitment, recruitment order, discharge rate, or discharge pattern [65]; disruption in the joint behavior of motor units affecting the quality and magnitude of force production [66,67]; and altered excitability of the motor neuron pool affecting the probability of motor unit activation [68].

Previously conducted studies of motor unit activity in hemiplegic subjects have been fraught with inconsistencies. These studies have involved limited numbers of subjects [69], populations of individuals with widely varied duration of lesion (i.e., 7 days to 16 years [70] or 1 to 20 years [53] within the same study), or multiple causes of hemiplegia (e.g., CVA/multiple sclerosis/“spinal involvement” [70], CVA and traumatic brain injury [69]). Consequently, it is difficult to draw straightforward conclusions regarding whether impaired muscular control in hemiplegic individuals should be attributed primarily to disruption at the supraspinal level or rather to intrinsic changes in the motor neuron or potentially the contractile properties of muscle. However, taken together, this literature suggests that compromise to motor units in hemiplegia is nonuniform between and within persons, considerable motor unit remodeling occurs between 2 and 6 months postonset, motor unit firing rates tend overall to be decreased relative to ipsilesional limbs, and reduced firing rates may affect the capacity to produce fused contraction. Also noteworthy, the bulk of previous investigations of motor unit activity in persons with poststroke hemiplegia has been performed in small muscles of the hand or muscles of the upper limb [24,69,71–73]. Only a few studies have been performed in the lower limb, and without exception, these have examined activity in the tibialis anterior muscle [53,60,70]. Moreover, all of these studies have been conducted using submaximal contraction forces, so the potential to understand altered motor unit recruitment and rate coding has not been fully tested.

Activation Impairment

Several notable investigators have speculated that weakness in poststroke hemiplegia results from impaired agonist motor unit activation [21,22,72,74]. A recent study demonstrated that electrically evoked contractile properties are similar between muscles of the paretic and ipsilesional limbs and concluded from these observations that impaired voluntary force production results from

impairment of central motor unit drive [75]. It is, however, difficult to assess the extent of motor unit recruitment through either surface EMG or motor unit firing patterns. Thus, more direct evidence of activation impairment in poststroke hemiplegia has been presented only recently through the use of superimposed electrical stimulation techniques. Harris et al. demonstrated impaired activation in the ipsilesional limb as early as the first week following hemiplegic stroke [17]. Newham and Hsiao studied activation of the leg extensors in 12 persons with hemiplegia at 1, 2, 3, and 6 months following stroke [76]. Throughout the entire 6-month study period, significant activation impairment was observed in not only the paretic but also in the ipsilesional legs, indicating an important disruption in the volitional capacity to produce maximal muscle force. These observations confirm that strength (or force) measurements from the ipsilesional side should be used with caution as reference values for comparison with the paretic side.

Although very few data report clear evidence of activation impairment in poststroke hemiplegia, quadriceps motor unit discharge patterns observed in our laboratory demonstrate that both motor unit recruitment thresholds and firing-rate modulation are significantly compromised in persons with poststroke hemiplegia [77]. We have also studied activation impairment in the upper limb of hemiparetic persons using MRI of muscle function [78]. Following maximal-effort contractions, T2-weighted MRIs of muscle function demonstrate a significant reduction in the CSA of activated muscle in the paretic arm. Moreover, the intensity of metabolic activity observed in the paretic arm is significantly lower. Collectively, these data strongly suggest activation impairment is an important mechanism contributing to poststroke weakness. It thus remains a significant question of both clinical and scientific merit whether activation impairment is reversible in poststroke weakness or whether loss of descending input to the spinal motor pool causes irreversible changes at the segmental level.

IS IT POSSIBLE TO COUNTERACT POSTSTROKE WEAKNESS?

Because poststroke weakness involves both neural and muscular changes, it seems appealing to suggest an analogy with other physiological conditions, such as aging, for which very clear benefits of strength training have been demonstrated [79]. Currently, available evidence regarding strengthening in hemiplegia indicates that significant strength gains are attainable in persons with poststroke hemiparesis at acute, subacute, and chronic stages of recovery [80–82]. However, the physiological mechanisms responsible for these therapeutically induced improvements have not been demonstrated. Consequently, it remains unclear whether these mechanisms have been optimally exploited. Because poststroke weakness results from an upper-motor neuron lesion, one needs to ask the question, Is there evidence that strengthening exercise actually influences neural drive at either the supraspinal or spinal level? Further, is there evidence that strength training influences muscle structure in poststroke weakness?

Functional and Task-Specific Training

Recent efforts for stroke rehabilitation have been directed toward functional and task-specific therapies that focus primarily on ADL and on grossly related precursor activities [83,84]. A common element to these more recent approaches is substantially increased therapeutic intensity relative to traditional approaches. However, a significant divergence in thought exists regarding whether this increased intensity is defined by a substantially increased volume of therapeutic participation (i.e., repetition, massed practice) [85], an increased amount of direct participation in therapeutic activities (e.g., time in therapy), or performance of activities at a higher level of the subject's functional capacity [86]. Current controversy thus centers around whether the critical variable for therapeutic efficacy is the task specificity or the intensity of effort involved in therapeutic activities. Modality-specific neural adaptations observed in animal models (i.e., angiogenesis in response to aerobic activity, synaptogenesis in response to motor skill training) [87,88] are typically used as evidence in favor of a task-specific approach. In contrast, proponents of increased intensity report generalizable improvements in both upper- and lower-limb function that transfer to ADL [89]. Also noteworthy, nontrivial gains in strength have been observed in response to motor learning [90,91]. Improvements in skill or functional task performance following strength training are, however, less straightforward. A recent study compared training-related neural adaptation in animals performing "power reaching" (analogous to strength or resistance training) or "skilled reaching" (analogous to task-specific training or motor learning) [92]. Significant cortical

reorganization was observed in both the power- and skilled-reaching groups. In addition, the power-reaching, or strength, group demonstrated significant synaptogenesis onto spinal motor neurons. Thus, while skill-based, task-specific interventions clearly promote important use-dependent cortical reorganization, resistance training apparently can promote additional, beneficial plasticity elsewhere in the neuraxis. In all likelihood, the most effective therapeutic intervention involves a combination of elements. However, the question regarding the critical variable(s) for optimizing recovery of function in poststroke hemiplegia remains at the forefront of our efforts to develop the most effective and efficient rehabilitation strategies.

Strength Training Defined

Following inactivity and immobilization, almost any vigorous activity will improve strength. However, not all exercise can or should be considered strengthening exercise. Strength training, or progressive resistance training (PRT), generally refers to training with progressively increasing resistive loads beginning at a minimum of 60 percent of that load that can be lifted once (one repetition maximum [1-RM]) [93]. The 1-RM is regularly tested at least every 2 weeks, and the resistive load is progressively increased to maintain a sufficiently intense training stimulus. There is a positive relationship between the resistive load and the degree of improvement. This model has been successfully used in older adults and has produced remarkable improvements in strength (as defined by 1-RM), functional mobility, and hypertrophy [94].

Effects of Strength Training in Poststroke Weakness

The literature on the effects of exercise, physical activity, and training in stroke patients falls into two categories: (1) interventions involving general exercise in conjunction with some component of resistance exercise and a significant increase in intensity over traditional therapy [83,86,89,95–98] and (2) studies involving resistance training. In this section, we will focus on resistance training (**Table 2** [41,80–82,98–104]).

Table 2 reports the nine studies that so far have evaluated PRT in persons with poststroke weakness. The majority of these studies examined chronic (>6 months postonset), while two studied acute (up to 45 days postonset) hemiplegic subjects. Although all of these studies involved some form of PRT, the specific parameters of resistance training have varied considerably. All of these studies reported positive adaptations to strength training. Consistent with a number of strength-training studies conducted in healthy populations, the duration of training ranged between 6 and 12 weeks. With one exception, all studies strongly suggest positive effects of strength training on various indices of functional outcome such as gait speed [99], stair-climbing ability, chair rise, and ADL. Interestingly, those protocols that involved a component of eccentric exercise appear to have demonstrated more significant gains in strength, which generalize to different muscle actions and to functional activities. Only three of these studies evaluated retention beyond the training period and found that improvements in either strength or functional performance were retained to some degree [80,81,100]. Thus, while insufficient data exist to draw firm conclusions at this time, functional effects of strengthening appear persistent. Four of the available studies evaluated effects of strength training on spasticity and found no deleterious effects. Finally, the effects of long-term (e.g., >12 weeks) strength training in poststroke hemiplegia remain to be determined.

Because of the limited number of investigations available to date, it is premature to establish definitive recommendations for resistance training in hemiparetic persons. There are, however, common themes among the available studies and these generally agree with commonly accepted guidelines for working with elders and older adults transitioning to frailty [105]. Accordingly, to induce improvements in strength in hemiparetic persons, studies recommend working at a minimum intensity of 60 percent 1-RM and a maximum of 12 repetitions per set. Outside of these parameters (i.e., loads of less than 60% 1-RM or greater than 12 repetitions per set), exercise will improve muscular endurance. They further recommend that three sets each of 8 to 10 exercises be performed three times a week [94,106], training span a minimum period of 6 to 12 weeks, and performance be monitored to adjust the resistive load to maintain the minimum desired training target (e.g., 60%–80% 1-RM). While longer term effects of resistance training in hemiparetic persons have not yet been demonstrated, PRT over a period of 24 months has not led to plateaus in increased strength and function in older adults [94]. Ongoing resistance training should thus be a fruitful avenue for promoting improved strength and function in hemiparetic persons.

While a compelling argument appears to be in favor of strength training for poststroke hemiplegia, it is important

to note that Level I evidence obtained from randomized controlled trials has been reported from only two studies to date [80,82]. All the remaining studies have involved either pre- or quasi-experimental designs. In addition, with notable exceptions [81,99], the sample sizes have been quite small, ranging between 7 and 20 subjects, which limits the

Table 2.
Effects of resistance training in poststroke hemiplegia.

Citation	Design	Population	Training Mode	Prescription	Adjunct Treatment	Strength Gains	Functional Outcomes	Retention	Effect on Spasticity
Badics et al., 2002 [99]	Preexperimental, nonrandomized	3 wk to 10 yr post-CVA, N = 56	Resistive exercise of UE and LE	4 wk 3–5 sets of 20 reps at 30%–50% of maximal mm strength including leg press w/hip and knee ext and arm press with elbow ext and shoulder retroversion	None	Mean strength gain for the LEs was 31% and for the UEs was 36.8%	Not tested	Not tested	No change as measured using the Ashworth scale
Winstein et al., 2004 [80]	RCT	Acute, between 2–35 d post-CVA, N = 60	3 groups: 1 group received standard inpatient rehabilitation only, other 2 groups received TRT or PRE in addition to SC	SC inpatient rehabilitation: SC group served as control and received no other therapy	- TRT group focused on systematic and repetitive practice of tasks - PRE group trained shoulder, elbow, wrist, and hand using free weights, TB, or grip devices	Significant incr in composite isometric torque when comparing TRT and PRE vs. SC posttreatment	When divided by severity, differences were seen in Fugl-Meyer and Functional Test of the Hemiparetic UE in less severe groups (TRT and PRE > SC)	Functional and strength training groups were comparable at 9 mo follow-up. When less severe subgroups were examined, TRT surpassed PRE for composite isometric torque ($p < 0.05$)	Not tested
Kim et al., 2001 [82]	RCT	Chronic, N = 20, randomly distributed into equally sized exp and control grps	Conc isokinetic strength training using Kin-Com device	- Exp grp received 3 ×/wk for 6 wk, 3 sets × 10 reps of max effort conc hip, knee, and ankle flex/ext - Control received PROM on Kin-Com device	None	Trend toward incr strength in exp grp ($p = 0.06$), mean composite strength score incr by 507% ± 559 for exp grp vs. 142% ± 193 for controls	Stair climbing and gait vel improved in both groups but no statistically significant difference between groups	Not tested	Not tested
Weiss et al., 2000 [101]	Preexperimental, nonrandomized	Chronic, N = 7	Conc/eccen strength training using weight machines	2 ×/wk, 12 wk, 3 sets of 8–10 reps at 70% 1-RM	None	Strength gains of 68% in P LE and 48% in NP side, improvements in hip flex, ext and abduction, knee ext (all $p < 0.01$), no incr in leg press	Chair stand time decr by 21% ($p < 0.02$) and stair climb time improved by 11% ($p < 0.07$). Gait vel and leg stance time no change. 9% incr in Motor Assessment Scale ($p < 0.04$), 12% incr in Berg Balance Scale ($p < 0.004$).	Not tested	Not tested

Table 2. (Continued)
Effects of resistance training in poststroke hemiplegia.

Citation	Design	Population	Training Mode	Prescription	Adjunct Treatment	Strength Gains	Functional Outcomes	Retention	Effect on Spasticity
Teixera-Salmela et al., 1999, 2001 [102,103]	RCT	Chronic, N = 13 (7 subjects first served as controls w/testing occuring initially, at 10 wk w/o intervention, and then again after 10 wk of training)	Isometric, eccen, and conc exer using sand-bags and TB	30 min 3 ×/wk for 10 wk, 3 sets of 10 reps to the hip, knee, and ankle at 50% 1-RM (incr to 80% by 2 wk) and reassessed every 2 wk thereafter	- 5–10 min warm-up - 10–20 min aerobic conditioning consisting of 10–20 min graded TM walking, stepping, or cycling at 70% HR max, - Cooldown 5–10 min	42.3% incr in strength of P limb as assessed during conc isokinetic torque (30° and 60°/s)	28% incr in gait vel (p = 0.000), 37.4% incr in stair climbing (p = 0.000). Improved ability to do household chores as reported by 39.2% incr in AAS taken from the HAP. 77.8% improvement in NHP (p = 0.001). Trend toward improvement in max PF angle at push-off and knee flex angle during swing in both limbs. Incr in moment, power and work in hip and ankle.	Not tested	No change in Pendulum Test
Thielman et al., 2000, 2002 [98,100]	Preexperi-mental	5–18 mo post-CVA, N = 12	TRT or PRE group (further divided within groups into high- and low-level group)	- PRE group: PRE in proximal and distal UE muscles using TB - TRT: Task-related reaching and grasping diverse objects while minimizing compensatory movements especially at the trunk	None	Not tested	Kinematic analysis of arm and trunk revealed high PRE subjects incr independent arm motion. Low-level subjects incr trunk. Smoother velocity profiles during reaching were observed for both groups posttraining.	1 yr follow-up movement time was longer and velocity profiles smoother for all groups. High PRE maintained independent arm motion. Low PRE has incr trunk use to contralateral targets.	Not tested
Sharp & Brouwer, 1997 [104]	Quasi-experimental	Chronic, N = 15	Isokinetic, conc training of quads and HS	3 ×/wk for 6 wk, 3 sets of 6–8 reps at max effort at 30°, 60°, 120°/s	5 min warm-up on bike, 4 × 15 s stretches for quads/HS	Significant gains (p < 0.05) seen for P quads and HS at all 3 speeds, quads strength improved from 15%–19% and HS strength from 37%–154%	Gait vel incr 5.3% and 6.8% at follow-up (p < 0.05). No change in TUG or stair climbing; 25% incr in AAS, and 36% incr in AAS at follow-up (p < 0.01).	After 4 wk of detraining, improvement was still evident but no longer significant compared to baseline except for quads at 30° (p < 0.05).	Pendulum w/ surface EMG, no change in hypertonicity (p > 0.87). Correlation between training-related torque gains and posttraining relaxation index values were poor for quads (r = 0.26) and HS (r = 0.35).

Table 2. (Continued)
Effects of resistance training in poststroke hemiplegia.

Citation	Design	Population	Training Mode	Prescription	Adjunct Treatment	Strength Gains	Functional Outcomes	Retention	Effect on Spasticity
Engardt et al., 1995 [41]	Preexperimental, nonrandomized	Chronic, N = 20	Isokinetic training of knee extensors conc group and eccen group	2 ×/wk for 6 wk 60°/120°/180°/120°/60°/120°/180°; 10 reps each incr to 15	None	- Conc group: 25%–57% gains in conc strength, 13%–17% incr in eccen strength ($p < 0.05$); - Eccen group: 25%–30% incr in both conc and eccen strength ($p < 0.05$)	Gait vel incr in both groups ($p < 0.05$), nearly equal weight bearing in P and NP legs in StS for eccen group, but no change in body wt distribution in StS in either group.	Not tested	Incr cocontraction observed using surface EMG in conc group only
Inaba et al., 1973 [81]	RCT	Acute N = 77	Functional retraining and stretching (F-S) vs. functional retraining, stretching, and active exer (FAE) vs. functional retraining, stretching and PRE	1–2 mo of daily functional retraining and selective stretching in all groups during inpatient stay. - F-S group served as a control and received no additional treatment	- FAE group received group active exer involving bilateral hip/knee flex/ext, hip abd, LE coordination exer, trunk exer - PRE group received PRE = 5 reps at 50% 10-RM followed by 10 reps at 10-RM hip/knee ext	PRE group incr in strength 13.5 lb, FAE group 7 lb, and F-S group 4 lb	In 1 mo, PRE group incr 64% in ADL vs. 38% FAE and 30% F-S	No differences between PRE and functional groups at 2 mo follow-up	Not tested

Note: See main paper reference section for detailed references.
AAS = adjusted activity scores, ADL = activities of daily living, AMAT = Arm Movement Activity Test, conc = concentric, control = control group, decr = decrease, eccen = eccentric, EMG = electromyography, exp grp = experimental group, ext = extension, flex = flexion, gait vel = gait velocity, GRF = ground reaction force, GT = gait training, HAP = Human Activity Profile, HR = heart rate, HRR = heart rate reserve, HS = hamstrings, incr = increase, LE = lower extremity, mm = muscle, NHP = Nottingham Health Profile, NP = nonparetic, 1-RM = 1 repetition maximum, P = paretic, PF = plantarflexion, PRE = progressive resistance exercise, PROM = passive range of motion, quads = quadriceps, RCT = randomized controlled trial, SC = standard care, StS = sit to stand, TB = thera-band, 10-RM = 10 repetitions maximum, TM = treadmill, TRT = task-related training, T/t = Torque/time, TUG = timed up and go, UE = upper extremity, WT = weight

generalizability of the results and increases the risk of Type II errors. Finally, we know of no study that has evaluated the mechanisms underlying improvements in strength and function following rehabilitation for poststroke hemiplegia.

WITHER?

Given the current evidence regarding the effects of strength training, can we recommend its incorporation into neurorehabilitation? From our point of view, as clinicians working in neurorehabilitation, strength training clearly has a role in reversing poststroke weakness. Strength training should not, however, be seen as a replacement for effective functional training. Rather, resistance training can be a significant adjunct or augmentation to traditional rehabilitation. However, more studies are needed to fully understand the specific parameters that produce optimal treatment effects and promote efficient attainment of functional outcome. Accordingly, we propose the following areas for future research.

How Significantly Does Poststroke Weakness Influence Attainment of Functional Outcome?

The ultimate goal of rehabilitation following stroke is to promote improvements in function, activities, and

participation. Collective efforts are thus required to design effective and efficient rehabilitation interventions. We appreciate that weakness is not the only impairment in poststroke hemiplegia. However, evidence clearly indicates that weakness plays a significant contributory role to motor disability. One area for future research is to determine the significance of weakness relative to other motor impairments and to understand weakness in the perspective of the individual's capacity to pursue meaningful ADL following stroke. Moreover, examining the various facets of weakness (i.e., force magnitude, slow force production, fatigability, excessive sense of effort, ineffective task-dependent force production) will provide a more detailed understanding of the specific nature of motor impairment and will identify potential strategies to mitigate its effects and promote improved functional performance and participation in activities.

What Mechanisms Are Involved in Poststroke Weakness?

An equally important area for future research is developing a greater understanding of the mechanisms underlying poststroke weakness. Without this information, we are restricted in our efforts to design appropriate rehabilitation interventions to counteract compromised function associated with poststroke weakness. Studies combining contemporary high-resolution techniques such as MRI, muscle biopsy, and electrophysiology will help us understand to what degree neural versus muscular factors are responsible for poststroke weakness. Moreover, these studies should be conducted in the context of the clinical phenomenon of poststroke hemiplegia to identify critical clinical features, such as severity, chronicity, lesion location, and comorbidities, and how these issues affect successful attainment of functional outcome.

Is Strength Training Simply a Case of "More Therapy Is Better"?

Clearly, more therapy is better [80,81,95]. Recent research evidence indicates that "task-specific" therapy (**Table 3** [83,84,89,95–97,107–110]) produces superior outcomes as compared to traditional therapeutic approaches [89,107]. However, there is also evidence that increased intensity of therapy leads to more significant functional outcome. Is it simply the case that strength training affords a means for providing a higher volume and/or intensity of therapy? There is a need to establish the effectiveness of strength training in relation to task-specific therapies because it may be the case that strength training is an efficient means for delivering high-intensity therapy. Given the significant constraints currently imposed by the healthcare delivery system, a need exists to determine the optimal cost-benefit given the available resources (both patient and facility/system) for therapy. To define and implement suitable protocols of strength training into stroke rehabilitation programs, future research should explore the specific factors such as the types of exercise (i.e., eccentric vs. concentric vs. isokinetic vs. closed and open chain), the frequency, intensity and time spent in strength training, and the number of specific exercises. Moreover, the long-term effects, both long-term training and retention of training, need to be understood. Finally, once gains in strength have been achieved we need to understand how they translate to functional gains and how they are best maintained.

How Do We Assess "Real-World" Changes Following Strength Training?

While the overriding goal of rehabilitation is to improve function and promote the individual's participation in meaningful activities, rehabilitation treatment has traditionally focused on reducing impairments. However, to be fully successful, it is important for an intervention therapy not only to alleviate impairments but also to reduce disability. Improving strength without a concomitant impact at the activity level would thus not be considered a fully successful intervention. Presently, we are limited in our ability to demonstrate transfer of strength gains into meaningful changes in activity, participation, and quality of life. Accordingly, there is a strong need to agree on a profile of sensitive, reliable, and appropriate outcome measures for the effects of intervention for poststroke weakness to be assessed. It will remain important to elucidate the relationship between improvements in strength (i.e., alleviating impairment) and improvements in function (i.e., reduce activity limitation and participation restrictions) through future research.

Is Resistance Training Beneficial for All Persons with Poststroke Hemiparesis?

Previously, we discussed (see **Figure**) [111] that the relationship between strength and function may not be linear. Therefore, most likely, the effective transfer between strength training and function will differ depending on the degree of poststroke weakness. The characteristics of hemiplegic subjects have been poorly

Table 3.
Effects of usual, activity-based, and constraint-induced therapies.

Citation	Population	Therapeutic Approach	Prescription	Adjunct Treatment	Strength Gains	Functional Outcomes	Effect on Spasticity
Dean et al., 2000 [107]	Chronic, N = 12	TRT	3 ×/wk for 4 wk, 1 h of TRT for both grps. - Exp grp received circuit training in class format directed toward walking tasks. - Control received TRT for UE also in class circuit-training format.	None	Strength not measured directly; improved GRF for exp grp during StS suggests incr force production in LE	Significant improvement in all 6 functional measures (i.e., StS, 6 min walk, walking speed w/ and w/o assistive device, and step test) compared to UE group. All were greater immediately & 2 mo after.	Not tested
Silver et al., 2000 [108]	Chronic, N = 5	TM training	3 ×/wk for 3 mo beginning @ 40% of HRR, progressing to 40 min @ 60%–70% of HRR	None	Strength not measured	Timed "get-up and return to sit" decr from 8.2 ± 1.4 s to 6.5 ± 0.8 s ($p < 0.05$). Timed "straight-away walk" segment decr from 3.7 ± 1 s to 2.8 ± 0.7 s ($p < 0.05$). Mean gait vel improved from 0.9 to 1.2 m/s, a 33% incr ($p < 0.01$).	Not tested
Miltner et al., 1999 [109]	Chronic, N = 15	Constraint-induced therapy	Placed NP arm in sling for 90% of waking hours and training of P arm for 7 h on 8 weekdays during 12 d period	None	Strength not measured	Significant improvement in Motor Activity Log from first contact to 6 mo follow-up ($p < 0.0001$, effect size mean = 2.15). Wolf Motor Function Test FA ($p < 0.0001$) and performance time ($p = 0.095$) from baseline to follow-up (effect size mean = 1.02).	Not tested
Kwakkel et al., 1999 [89]	Acute, N = 101	Task-specific training	30 min 5 ×/wk for 20 wk - Exp grp 1 received LE training - Exp grp 2 received UE training - Control received UE and LE air splints	All groups received 15 min LE, 15 min UE and 1.5 h ADL retraining 5 d/wk	Strength not measured	At 6 wk, LE had higher scores than control and UE grp for ADL, walking, and dexterity. At 20 wk, LE group had higher scores than control for ADL, walking ability, and dexterity. UE group differed significantly from control in dexterity only at 20 wk. No significant difference between UE and LE at 20 wk.	Not tested
Smith et al., 1999 [96]	Chronic, N = 14	TM training	3 ×/wk for 3 mo beginning @ 40% of HRR, progressing to 40 min @ 60%–70% of HRR	None	Conc T/t production incr by 50% in P ($p < 0.05$) and 31% in NP ($p < 0.01$) HS. Eccen T/t production incr by 21% in P ($p < 0.01$) and 22% in NP ($p < 0.01$) HS.	Not measured	Isokinetic dynamometry; reflexive T/t production in P HS decr by 11% ($p < 0.027$) and did not change in NP HS ($p = 0.45$)

Table 3. (Continued)
Effects of usual, activity-based, and constraint-induced therapies.

Citation	Population	Therapeutic Approach	Prescription	Adjunct Treatment	Strength Gains	Functional Outcomes	Effect on Spasticity
Duncan et al., 1998 [95]	Subacute, N = 20	Home-based exercise program consisting of either PNF or TB	- Exp group received 3 ×/wk for 8 wk (plus 4 wk independently w/o PT) 15 min of TB or PNF - Control group was visited by research assistant every 2 wk to assess activity level	Exp grp also received balance exer, functional UE tasks, and walking program or exer bike	Strength not measured	Improvement in LE Fugl Meyer scores and improvement in gait vel in group that received LE TB resistance training.	Not tested
Dean & Shepherd, 1997 [83]	Chronic, N = 20	TRT	- Exp group received 10 sessions spread over a 2 wk period involving reaching tasks beyond arm's length - Control groups received sham training involving cognitive-manipulative tasks while seated at a table	None	Strength not measured	Exp grp significantly incr maximum-reaching distance compared to baseline and compared to control ($p < 0.001$). Exp grp performed reaching tasks in less time than controls (ipsilateral $p = 0.08$, contralateral $p = 0.001$). Exp grp had incr in GRF of affected foot compared to control ($p < 0.001$). No change observed in control grp.	Not tested
Taub et al., 1993 [110]	Chronic, N = 9	Constraint-induced therapy	- Exp grp NP arm constrained in a sling 90% of waking hours for 14 d. Subject spent 6 h performing tasks with P arm on weekdays during this time. - Comparison group told to focus attention on P arm	None	Strength not measured	Exp grp Emory Motor Function test and AMAT task completion times decr by 30%, whereas comparison group incr by 2.2%. Quality of movement and functional ability were improved significantly on Emory Test and AMAT for exp grp ($p < 0.003$).	Not tested
Richards et al., 1993 [97]	Acute, N = 27 w/MCA CVA	TRT	- Exp grp received intensive treatment (1.75 h/d), focused on GT using tilt table, Kinetron and TM - Control groups received conventional therapy w/o focus on locomotion (1st group receiving at same intensity and 2d group at a slower pace as it had been delivered previously at hospital, 0.75 h/d)	None	Strength not measured directly	41% incr in gait vel in exp grp compared to controls. Time dedicated to GT but not total therapy time was correlated to gait vel ($r_s = 0.63$). Type of therapy was more important than time in therapy. Differences in gait vel disappeared by 3 mo.	Not tested
Malouin et al., 1992 [84]	Acute, starting at day 8 after CVA, N = 10	TRT	60 min 2 ×/day 5 d/wk for 5 wk treatment included special GT and traditional therapy; gait training introduced ASAP and preparatory pregait activities included Kinetron II	None	Strength not measured	Intense gait relearning was tolerated well immediately after stroke.	Not tested

Note: See main paper reference section for detailed references.
AAS = adjusted activity scores, ADL = activity of daily living, ASAP = as soon as possible, conc = concentric, control = control group, CVA = cerebrovascular accident, decr = decrease, eccen = eccentric, Exp Grp = experimental group, ext = extension, flex = flexion, gait vel = gait velocity, GRF = ground reaction force, GT = gait training, HAP = Human Activity Profile, HRR = heart rate reserve, HS = hamstrings, incr = increase, LE = lower extremity, MCA = middle cerebral artery, NP = nonparetic, P = paretic, PF = plantarflexion, PRE = progressive resistance exercise, TB = thera-band, TM = treadmill, TRT = task-related training, T/t = Torque/time, StS = sit to stand, UE = upper extremity

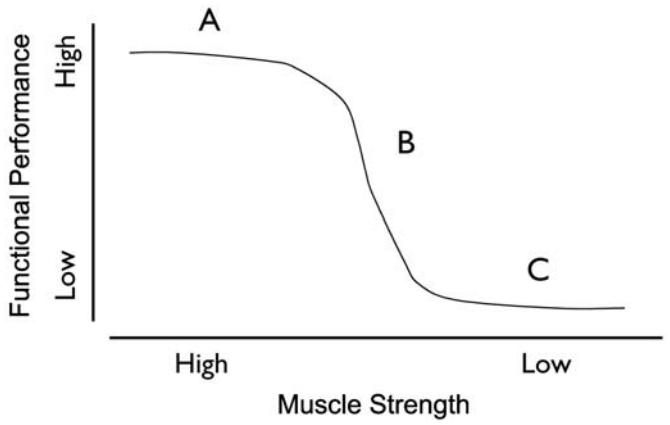

Figure.
Precipice effect depicting theoretical nonlinear relationship between muscle strength and functional performance (i.e., gait, balance, chair rise, stair climb). Effects of strengthening on function are likely to depend on initial level of poststroke weakness. In persons demonstrating relatively high strength and high performance (Region A), strength training can be regarded as preventative measure reducing risk of falling off precipice of function into disability. For hemiparetic persons falling in Region B, small improvements in strength may promote substantial improvements in function. For those in Region C, strengthening may afford physiologic benefits, but expectations for improved function should be modest. *Source:* Buchner DM, Beresford SA, Larson EB, LaCroix AZ, Wagner EH. Effects of physical activity on health status in older adults. II. Intervention studies. Annu Rev Public Health. 1992;469–88.

documented in current literature. Accordingly, it is entirely possible that failure to demonstrate consistent, straightforward benefits of resistance training (i.e., consistent magnitude of improvements, transfer of effects to function, retention of training effects) is more likely caused by heterogeneity among hemiplegic persons included in the study groups than by failure to induce significant, physiologically important adaptations. To date, studies in which subjects were stratified by hemiplegic severity have demonstrated more significant benefits of strength training in persons less severely affected by poststroke hemiplegia [80,100].

In persons demonstrating relatively high strength and high performance (Region A of the **Figure**), strength training may not produce readily measurable effects on function. Strengthening may, however, prevent decline below critical thresholds of functional capacity. Future research could explore whether strength training for such persons serves an important role in health promotion and recreation extending beyond the boundaries of the traditional rehabilitation setting.

For a specific group of stroke patients, however, a small change in strength may promote substantial improvements in function (Region B of the **Figure**). This group, which is the main focus of rehabilitation, is theoretically the most likely to benefit from strength training. Future research efforts should identify the characteristics of persons who make up this group and explore the scope of potential benefits of strength training.

Despite increases in strength, improvements in functional performance may not occur in hemiplegic persons with low strength and low performance (Region C of the **Figure**). However, even if PRT does not lead to any functional benefits, it may still play a significant role for these individuals. It is entirely possible that vigorous strength training promotes positive effects on other aspects of physiologic function in this type of at-risk population. In elders, strength training has been demonstrated to decrease depression and improve sleep patterns, influence bone mass, decrease insulin resistance (Type II diabetes), and normalize blood pressure [106]. Even without straightforward effects on functional performance, physiologic systems can have associated effects that should not go unrecognized. These physiologic effects present an additional potentially fruitful area of research.

Finally, one must recognize that PRT may not be suitable for all hemiparetic persons. In this regard, we recommend exercising prudent clinical judgment appropriate in any rehabilitation setting. High-intensity resistance training is certainly contraindicated in any case before the patient is neurologically stable. Other significant contraindications would involve postsurgical patients and persons with severe osteoporosis, acute orthopaedic, or joint injuries. While the patient or client is exercising, his or her blood pressure should be monitored, and precautions should be taken to avoid conditions leading to a valsalva maneuver.

CONCLUSION

While the number of studies is limited, emerging evidence suggests that persons with poststroke weakness can improve strength through resistance exercise in the absence of negative side effects, including exacerbation of hypertonia. Moreover, these improvements in strength appear to transfer to functional improvements. Still, many unresolved issues remain. The potential for strength training to improve the overall outcomes of rehabilitation for persons with poststroke hemiplegia warrants further investigation.

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