Strengthening to Promote Functional Recovery Poststroke: An Evidence-Based Review

Sang Pak and Carolynn Patten

Background: Following stroke, patients/clients suffer from significant impairments. However, weakness is the predominant common denominator. Historically, strengthening or high-intensity resistance training has been excluded from neurorehabilitation programs because of the concern that high-exertion activity, including strengthening, would increase spasticity. Contemporary research studies challenge this premise. **Method:** This evidence-based review was conducted to determine whether high-intensity resistance training counteracts weakness without increasing spasticity in persons poststroke and whether resistance training is effective in improving functional outcome compared to traditional rehabilitation intervention programs. The studies selected were graded as to the strength of the recommendations and the levels of evidence. The treatment effects including control event rate (CER), experimental event rate (EER), absolute risk reduction (ARR), number needed to treat (NNT), relative benefit increase (RBI), absolute benefit increase (ABI), and relative risk (RR) were calculated when sufficient data were present. **Results:** A total of 11 studies met the criteria. The levels of evidence ranged from fair to strong (3B to 1B). **Conclusions:** Despite limited long-term follow-up data, there is evidence that resistance training produces increased strength, gait speed, and functional outcomes and improved quality of life without exacerbation of spasticity. **Key words**: *cerebrovascular accident, recovery, rehabilitation, resistance training, strength, stroke*

Stroke is the leading cause of chronic physical disability in Western industrialized nations.[1] In the United States alone, there are currently over 5.6 million people disabled from stroke. The direct and indirect costs of caring for these persons were estimated to reach $62.7 billion in 2007.[2] The incidence of stroke doubles with each decade beyond 60 years of age; thus, as the proportion of the population reaching this age milestone accelerates, the financial burden–including direct costs of medical care and lost productivity–can be expected to grow in parallel.[3]

The most prominent motor deficit after stroke is paresis of the side of the body contralateral to the cerebrovascular event. A mounting body of contemporary research evidence documents that weakness is the primary impairment in persons poststroke.[4,5] Although it can be difficult to differentiate weakness from impaired voluntary selective motor control, obligatory synergies, or hypertonia, it is critical for clinicians to specifically identify and address hemiparetic weakness. The available body of contemporary evidence suggests that incorporation of strengthening exercises into physical rehabilitation programs can serve to counteract weakness and improve function for persons poststroke. This evidence-based review was conducted to determine the effectiveness of resistance training for addressing the impairment of hemiparetic weakness and concomitantly increasing function without exacerbating spasticity.

Background

Physical disability poststroke

Significant impairments are observed in persons poststroke including weakness, generalized fatigue, loss of voluntary motor control, spasticity, and sensory and cognitive dysfunction.[5–16] It is estimated that over 65% of stroke survivors experience hemiparetic motor dysfunction up to 1-year post cerebrovascular event.[17] Between 73% and

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88% of first-time stroke survivors experience acute hemiparesis of the upper and/or lower limbs that drastically impacts performance of functional abilities.[18] One study interviewed 4,023 persons who had experienced a stroke. Two years poststroke, 10% of those interviewed stated they "always felt tired," and 29.2% stated that they were "often tired" (Riks-Stroke Questionnaires).[17]

Mechanisms of hemiparetic weakness

Weakness, formally defined as the inability of a patient to generate normal levels of muscle force under a specific set of testing conditions,[19] is one of the most common findings in the chronic phase poststroke.[20] Weakness is correlated with decreased performance of important functional activities including gait speed[21] and transfer capacity.[22] Although the correlation between strength and functional performance does not confirm a causal effect, the strength of this relationship and the accumulation of evidence suggest weakness is a major factor limiting motor performance poststroke.[20] In addition to weakness, selective control of muscles may be impaired in hemiparetic patients during voluntary, functional movements. Impaired motor control could involve spatial and temporal abnormalities in activation of the agonists, aberrant activation of the antagonist and synergist muscles, and/or impaired descending motor drive. The behavioral manifestation of these aberrant motor control mechanisms, either individually or in combination, can present as generalized weakness on the paretic side.[20,23]

Some studies report muscle weakness is more commonly found in Type II muscle.[24,25] However, in persons poststroke, the results are conflicting.[25] Typically, muscle changes are assessed by using morphologic measurements, either muscle imaging or muscle biopsy, whereas motor activation is assessed by using electromyography[24] and muscle performance is assessed using various forms of muscle testing and dynamometry.[25] Most hemiparetic persons reveal a significant reduction in force/torque production both isometrically and dynamically whether measured using isotonic or isokinetic conditions.[13] Evidence of muscle atrophy poststroke is equivocal at both the single fiber[24] and whole tissue[25,26] levels, which motivates a nonmuscular (e.g., neural) explanation of hemiparetic weakness.

In the absence of convincing evidence that structural differences in muscle contribute to hemiparetic weakness, attention turns to the neural aspects of strength and force control. The neural mechanisms controlling muscular force involve task-dependent motor unit activity–recruitment and rate coding–of already active motor units.[27] The interaction of motor unit recruitment and rate coding affords the infinite gradation of muscle forces involved in execution of functional motor tasks. 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. The consequent reduction in neural traffic at the spinal segmental level results in motor neuron loss[28] and disruption of these primary force control mechanisms. The majority of motor unit remodelling appears to occur in the subacute timeframe between 2 and 6 months poststroke.[28] Following this period, the natural history of hemiparesis describes significant muscular weakness,[29–31] which may or may not be accompanied by hyperreflexia.[32] Prevailing wisdom in clinical rehabilitation once held that both muscular weakness and spasticity resulted from disruption in the balance of activity between antagonist muscle pairs.[33] However, a body of recently performed research has established that spasticity is a phenomenon separate from antagonist co-contraction.[34–38] Accordingly, these observations suggest that muscular weakness results from failure to adequately activate motor units supplying agonist muscles.[31]

Activation impairment

It has been broadly speculated that hemiparetic weakness results from impaired agonist motor unit activation.[28,31,32,39,40] Recent studies demonstrate impaired muscle activation (i.e., paucity of electromyographic [EMG] activity) in both the upper[41] and lower extremities[42] during isolated single joint movements and also during simple reaching[43] or locomotor[44] movements. Further, EMG activity fails to modulate in response to task-specific demands.[42] This absence of EMG modulation may occur because hemiparetic individuals fully recruit

their available motor pool in an effort to perform a target task. Consistent with this premise, McCrea et al.[45] observed "saturation" of EMG activity during unrestrained (e.g., unloaded) reaching tasks. Direct evidence of activation impairment in persons poststroke has been demonstrated using a twitch superimposition technique.[46] Activation of the leg extensors was studied in 12 persons with hemiparesis at 1, 2, 3, and 6 months following stroke. Significant activation impairment was observed in both paretic and nonparetic legs throughout the entire 6-month study period, indicating a persistent disruption in the ability to volitionally produce maximal muscle force.

Weakness versus spasticity

Is weakness or spasticity the primary etiology of disability and impaired motor control in persons following stroke? Several hypotheses exist regarding the factors that contribute to spasticity. Spasticity could result from diminished agonist motor unit activation, impaired antagonist inhibition, or impaired presynaptic inhibition of spinal reflex pathways.[14,47] Weakness could contribute to spasticity in a variety of ways: reduced traffic in descending pathways responsible for voluntary movement[48,49]; muscle fiber atrophy and contracture[48,49]; changes in the spatial and temporal patterns of muscle activation causing an inefficient EMG-torque relationship[50]; loss of functional motor units and changes in the properties of remaining units; and limited voluntary range of motion in agonists with muscles producing decreased maximal force due to activation on a suboptimal portion of the force-length relationship.[51]

Intervention poststroke

The primary goals of rehabilitation are to maximize functional independence, minimize long-term disability, and increase participation in meaningful activities of daily living (ADLs). With the rising costs of health care and concomitant emphasis on attainment of benchmarks and outcomes by third party payers, it has become increasingly important to incorporate the findings of evidence-based practice into appropriate treatment plans for persons poststroke.

Contemporary stroke rehabilitation has centered around the emerging evidence of neuroplasticity.[52–56] Neural adaptation can occur in various ways including changes in synaptic strength, circulating levels of neurotransmitters, axonal sprouting, and/or formation of new synapses.[52–56] It is important to note that plasticity occurs throughout the neuraxis, not only in the brain. Although this contemporary scientific evidence should be encouraging to clinicians, convincing evidence that documents the effectiveness of different neurological treatment approaches[57,58] remains lacking. For example, one of the most commonly used methods of neurorehabilitation, neurodevelopmental treatment, is not supported by research evidence.[58,59]

Traditionally, strengthening or high-intensity resistance training has often been excluded from neurorehabilitation. This practice stemmed from the belief that high-exertion physical activities would exacerbate spasticity.[33] Historically, any therapeutic activities involving increased physical exertion were considered contraindicated to prevent exacerbation of spasticity.[5] However, many recent research studies now refute this traditional notion.[12,14,16,60]

Primary Question

This evidence-based review involved a foreground and intervention question and was conducted to answer the following questions:

- Can strengthening or high-intensity resistance training counteract weakness without increasing spasticity in persons poststroke?
- Is strength training effective for improving functional outcomes compared to traditional nonstrengthening intervention programs?

Demonstration of increased functional capacity following strengthening interventions in persons poststroke should challenge the mindset of practicing clinicians and possibly lead to a paradigm shift in neurorehabilitation.

The focus of this review was on persons with mild or moderate poststroke hemiparesis without other significant medical complications or comorbidities. The intervention of interest included high-intensity resistance or strengthening exercises alone or coupled with aerobic or endurance exercises. The comparison of interest was between

resistance training and either nonstrengthening endurance or passive protocols. Resistance training activities included free weights, elastic bands, weight machines (i.e., Keiser pneumatic devices or Universal weight stacks), or isokinetic training. The critical outcomes evaluated following resistance training included isometric and dynamic strength assessments, gait speed, gait pattern, walking endurance (6-minute timed walk), and perceived exertion. These outcomes were evaluated in conjunction with assessment scales representing all levels of the ICF model[61] and included Fugl-Meyer Motor Assessment,[6] Motor Assessment Scale (MAS),[10] Barthel Index (ADL),[62] Chedoke-McMaster Stroke Assessment (CMSA),[63] FIM™* scores,[9] and quality of life measures including the Stroke Impact Scale,[64] the Medical Outcomes Scale SF-36,[65] the Late Life Function and Disability Instrument (LLFDI),[66,67] and the Geriatric Depression Scale (GDS).[68]

Null hypotheses

For persons poststroke (subacute to chronic stage), strengthening exercises integrated into the rehabilitation program will fail to:

- Counteract muscle weakness
- Be effective in improving functional independence and other functional outcomes
- Improve outcomes compared to rehabilitation strategies without specific strengthening exercises

Strengthening exercises are expected to increase strength and function but not increase spasticity in persons in a stable phase poststroke.

Background and Search Methods

Sources for evidence-based search

The sources used for the evidence-based search included PubMed, Cochrane Library, PEDro, MEDLINE, Hooked on Evidence, Australian Physiotherapy Association, Center for Evidence-Based Physiotherapy, and reference lists from key research studies. PubMed and MEDLINE were the most frequently used sources during the search, which included studies from the last 18 years (1990–2008, inclusive).

The key words used for the search included "cerebrovascular accident," "stroke," "hemiplegia," "strengthening," "rehab," "rehabilitation," "resistance," "resistance exercise," "muscle weakness," "therapy," and "physiotherapy."

Study inclusion and exclusion criteria

The clinical studies included in this search were randomized clinical trials (RCTs), systematic reviews, and meta-analyses. The specific inclusion and exclusion criteria are summarized in **Table 1.** Initially, the search included all stages of poststroke hemiparesis, but later it excluded the acute stage (i.e., up to 30 days post cerebrovascular event) due to the limited number of clinical studies associated with resistance training in this phase of recovery. The concept of strength training was required to involve either concentric or eccentric contractions, or both, performed using the affected limb. Resistance training was demonstrated in the studies through various approaches including resistive theraband, free weights, isokinetic dynamometers such as Cybex (Cybex International, Medway, MA) or Biodex (Biodex Medical Systems, Shirley, NY), and circuit-training machines including the Keiser pneumatic devices (Keiser Corporation, Fresno, CA) and/or Universal weight stacks.

The participants included in the studies were adults between 45 and 75 years of age.[1,2] The settings for therapy ranged from acute inpatient rehabilitation to outpatient rehabilitation. Studies including participants with more than one stroke or other significant comorbidities (i.e., major orthopaedic limitations, cancer, major neurological conditions) were excluded from the search.

Results of Evidence-Based Search

Number of studies

Fifty-three studies were identified from the initial search. Eleven studies met the inclusion criteria. Out of the 11 total studies, 8 were RCTs[6–9,11–13,69] and 3 were case-control studies.[6,10,14] Only manuscripts written in English were included in the review. The levels of evidence ranged from

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Table 1. Inclusion and exclusion criteria for studies

Inclusion criteria	Exclusion criteria
• Single instance of cerebrovascular event • Subacute to chronic stage of poststroke hemiparesis (at least 1 month post cerebrovascular event) • Ability to walk at least 20 feet with assistive device or 10 feet without assistive device • Minimal to moderately impaired sensorimotor function based on Fugl-Meyer Motor Score between 40 to 90 • Near pain-free range of motion in both extremities • Strengthening protocol including resistance training exercises as part of therapy/rehab • At least one or more of the following outcomes: FIM™ scores, Fugl-Meyer Assessment, strength, quality of life, gait speed, stair climbing, gait pattern, other functional/relevant ADLs • Medically stable and no other comorbidities • Ages from 45 to 85 years • Setting: Acute rehab and outpatient	• More than one instance of cerebrovascular event • Acute stage of poststroke hemiparesis (less than 30 days post cerebrovascular event) • Unable to walk greater than 20 feet with or without assistive device • Impaired cognition as evidenced by: Mini-Mental State Exam <18, inability to understand and/or follow three-step commands. All participants screened by physician. • Painful and limited range of motion in both extremities • Rehabilitation protocol that includes constraint-induced therapy • No other functionally related outcome measures • Medically unstable significant comorbidities

1B to 3B.[70] The level of evidence for each study is summarized in **Table 2**. The strongest grade of recommendation was assigned to Kim et al.[13] (a single-blind, randomized controlled study). The earliest included study was published in 1997, and studies were collected through January 2008.

Evidence-based statistics

Table 3 provides a brief description of each of the 11 clinical trials included and summarizes the dependent variables assessed and the level of evidence. When sufficient data were presented in the study, treatment effects including the control event rate (CER), experimental event rate (EER), absolute risk reduction (ARR), number needed to treat (NNT), relative benefit increase (RBI), absolute benefit increase (ABI), and relative risk (RR) were calculated.[70] **Table 4** summarizes these evidence-based statistics calculated for the 11 studies.

Description of studies

Functional outcome measures utilized varied for each study. Overall, 8 of the 11 studies included at least one positive change in a functional outcome. Outcome measures that were used included Fugl-Meyer (upper and lower extremity portions),[71] CMSA,[63] MAS,[72] gait speed, 2- and 6-minute walking test, stair climbing, chair rise, step test, Timed Up & Go (TUG),[73,74] Berg Balance Scale (BBS),[75] SF-36,[65] Nottingham Health Profile (NHP),[76] Human Activity Profile (HAP),[77] LLFDI,[66,67] and the GDS.[68]

Muscle strength was tested at the hip, knee, or ankle, or a combination, using various equipment including handheld or isokinetic dynamometers,[11,14,60] stacked weight machines,[10] and pneumatic resistance equipment.[8,10] Studies that used weight machines or pneumatic resistance equipment measured strength through one-repetition maximum (1-RM).[78,79] Studies that used isokinetic dynamometers measured strength as peak torque at one or more criterion speeds. Five of the 11 studies included strength measures using an isokinetic dynamometer. Only one study measured both concentric and eccentric contractions.[6] Two studies used Keiser pneumatic resistance training equipment.[8,10] Three studies used self-assessment scores such as the Stroke Impact Scale,[9] the SF-36,[7] or the participant's self-perceived exertion.[12] Although all 11 studies supported the finding of significant change in strength, not all of these studies quantified the participant's self-perception using standardized measurement tools such as self-assessment scores or self-perceived exertion.

The sample sizes varied in each clinical study. The largest number of participants was included in the Moreland[12] study (N = 133); Studenski et al.[9] studied 100 participants. Both of these studies were single-blind RCTs. The fewest participants were present in the study conducted by Weiss

Table 2. Level of evidence for each study

Level of evidence	Description	Number of studies	Names of studies
1a	Systematic review with homogeneity of RTC	None	
1b	Individual RCT with narrow confidence interval	2	• Kim et al., 2001[13] • Flansbjer et al., 2008[69]
1c	All or none	None	
2a	Systematic review of cohort studies (with homogeneity)	None	
2b	Individual cohort study (including low-quality RCT; e.g. <80% follow-up)	6	• Duncan et al., 1998[7] • Teixeira-Salmela et al., 1999[60] • Moreland et al., 2003[12] • Ouellette et al., 2004[8] • Studenski et al., 2005[9] • Yang et al., 2006[11]
3a	Systematic review (with homogeneity) of case-control study	None	
3b	Individual case-control study	3	• Engardt et al., 1995[6] • Sharp & Brouwer, 1997[14] • Weiss et al., 2000[10]
4	Case series (and poor quality cohort and case-control studies)	None	
5	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	None	

Note: Levels of evidence as established by Sackett[70] described with notation of the number of studies per level in this evidence-based review and the level of each included study. RCT = randomized controlled trial.

et al.,[10] which included only seven participants and utilized a nonrandomized repeated-measures design. The average sample size for the 11 studies was 36 participants (excluding the studies conducted by Moreland et al. and Studenski et al., the average sample size was reduced to 24).

The average duration of strengthening based on all 11 studies was 8.4 weeks, with the longest duration of intervention at 12 weeks[7–10] and the shortest at 4 weeks.[11,12] The only studies that reported on a follow-up measurement posttreatment were Sharp et al.,[14] Studenski et al.,[9] Flansbjer et al.,[69] and Moreland et al.,[12] with follow-up studies conducted at 4 weeks, 5 months, and 6 months, respectively.

The majority of the participants were classified as community dwelling. All participants met preestablished inclusion criteria for gait (independent ambulation of 10 feet without assistive device or 20 feet with an assistive device). Only individuals with a single, unilateral mild to moderate ischemic stroke were included in the studies. Cognitive ability was assessed with a variety of measures (e.g., Folstein Mini-Mental State Examination[80] <18 points or able to follow basic three-step commands) without receptive aphasia.

Integration of Findings Across Studies

Gait speed

Nine of the 11 studies reported a change in gait speed as part of their functional outcome.[6–8,10–14,60] From these nine studies, five reported statistically significant changes in gait speed ($p < .01$ to $< .05$).[6,7,11,14,60] Due to the variety of outcome measures used across the studies, these changes were assessed by evaluating reported changes in self-selected walking speed, fast walking speed, and/or the 6-minute timed walk test. Where available, changes in self-selected walking speed were used. If both self-selected and fast walking speeds were reported, the average of the two gait conditions was computed and this average was used to evaluate changes posttreatment. In cases where it was the only available gait outcome, gait speed was calculated from the 6-minute timed walk. The average change in speed (across eight studies) was

Table 3. Summary of the outcomes reported: strength and function

Level of evidence	Authors	Improved strength? How measured?	Improved function? How measured?
1b	Kim et al., 2001[13]	• Yes, statistically significant changes in strength. PF, DF, and knee extension were among highest changes. • Used **Kin-Com isokinetic dynamometer**	• No, SF-36 was not statistically significant
1b	Flansbjer et al., 2008[69]	• Yes, significant improvements in dynamic and isokinetic knee extension • Used **pneumatic resistance machine** for dynamic knee extension strengthening and **Biodex Multi-Joint System 3 PRO dynamometer** for isokinetic knee strength measurement	• Yes, TUG was statistically significant • Yes, 6MW was statistically significant • Yes, participation per Stroke Impact Scale was statistically significant
2b	Duncan et al., 1998[7]	• Yes, but measured indirectly using **Physical Function Test (MOS-36)**	• Yes, Fugl-Meyer UL and LE were statistically significant • Yes, Gait speed was statistically significant • No, 6-min walk was not statistically significant • No, Berg Balance, Barthel ADL, instrumental ADL, SF-36 all were not statistically significant
2b	Teixeiria-Salmela et al., 1999[60]	• Yes, peak torque for hip & knee flex, ext. All changes significant, obtained using **Cybex II isokinetic dynamometer.**	• No avg. gait speed change 0.3 m/s (±0.3 *SD*) (nonsignificant) • Yes, stair climbing was statistically significant • Yes, self-perceived function - Nottingham Health Profile (NHP) was statistically significant • Yes, Human Activity Profile (HAP) was statistically significant
2b	Moreland et al., 2003[12]	• Yes, amount of weight that subject is able to lift **(measured using sand bag weights)**	• The only statistically significant change was 2-min walking at 6-month postdischarge – all other outcomes were not statistically significant
2b	Ouellette, et al., 2004[8]	• Yes, peak torque for hip, knee, ankle flex and ext all significant changes • 1-RM for LEs was significant (31.4%). • Used **Keiser pneumatic resistance training equipment** using 1-RM	• No, direct functional measurements (6-min walk, stair climbing, and chair rise) were not statistically significant • Yes, Late Life Function and Disability Instrument (LLFDI) was statistically significant
2b	Studenski et al., 2005[9]	• Yes, but measured indirectly using the **Stroke Impact Scale.** Self-reported strength increased for experimental group.	• Yes, gait speed was statistically significant • Yes, SF-36 was statistically significant • Yes, Stroke Impact Scale was statistically significant • Yes, FIM was statistically significant
2b	Yang et al., 2006[11]	• Yes, strength gains were significant. • Used **handheld dynamometer** (PowerTrack II)	• All functional outcomes except the step test were statistically significant
3b	Engardt et al., 1995[6]	• Yes, for both eccentric and concentric trained groups • Used **Kin-Com 500H dynamometer**	• Body weight distribution during sit to stand was statistically significant for eccentric trained group • No significant difference in gait speed
3b	Sharp & Brouwer, 1997[14]	• Yes, significant changes in peak torque throughout, greatest in quads • Hamstrings were notably difficult for patients to register a flexor torque. • Measured via **Cybex II isokinetic dynamometer**	• No, avg. gait speed change was not statistically significant • No, TUG and stair climbing were not significantly significant • Yes, statistically significant changes for self-perception of functional activities under HAP
3b	Weiss et al., 2000[10]	• Yes, 1-RM for all muscle group was significant during 4th, 8th, and 12th weeks. • Used **Keiser pneumatic training equipment** for knee • Used **stack weight training machines** for hip	• Yes chair rise was statistically significant • Yes, MAS scores were statistically significant • Incremental increase in stair climbing was not statistically significant

Note: Studies are ordered by level of evidence, per Table 2, then chronologically within each level. A challenge in synthesizing these data was the vast array of outcome measured used. These measures are briefly summarized here for reference. Biodex Multi-Joint System 3 PRO dynamometer, Biodex Medical Systems, Shirley, NY; PowerTrack II, J-Tech Medical Industries, Salt Lake City, UT; Kin-Com 500H dynamometer, Isokinetic International, Harrison, TN; Cybex II isokinetic dynamometer, Cybex International, Medway, MA; Keiser pneumatic training equipment, Keiser Corporation, Fresno, CA. PF = plantarflexion ; DF = dorsiflexion ; TUG = Timed Up & Go; 6MW = 6-minute walk; UL = upper limb; LE = lower extremity; ADL = activity of daily living; flex = flexion; ext = extension; 1-RM = one-repetition maximum; MAS = motor assessment scale.

Table 4. Evidence-based statistics: primary question

Level of evidence	Authors	Parameters	CER	EER	ARR	NNT	RBI	ABI	RR	Treatment effect size[a]	Statistical significance
1b	Kim et al., 2001[13]	Composite strength (combined avg of flex & ext of hip, knee, ankle on paretic side, %)	142	507	365	1	2.57	365	3.57	43.61	Y (p<.001)
		Self-selected walking speed (m/s)	0.09	0.04	0.05	20	0.55	0.05	0.44	0.355	N (p=.29)
		Max walking speed (m/s)	0.07	0.05	0.02	50	0.29	0.02	0.71	0.154	N (p=.65)
		Self-selected stair-climbing speed (m/s)	0.08	0	0.08	13	1	0.08	—	0.567	N (p=.17)
		Max stair-climbing speed (m/s)	0.08	0.03	0.05	20	0.63	0.05	0.38	0.373	N (p=.26)
		SF-36 (Physical Health)	–0.73	0.74	1.47	2	2.01	1.47	(1.01)	N/A	N (p=55)
1b	Flansbjer et al., 2008[69]	Timed Up & Go (TUG)	0.182	0.101	0.081	12	0.45	0.082	0.555	5.71	Y (p<.05)
		Fast walking speed (m/s)	0.144	0.11	0.034	29	0.24	0.034	0.764	1.43	N (p>.20)
		6-min walk test (s)	0.073	0.0395	0.0335	30	0.46	0.0335	0.541	2.83	Y (p<.05)
		Stroke Impact Scale participation (%)	.203	.155	.048	21	.24	.048	.764	2.15	Y (p<.05)
2b	Duncan et al., 1998[7]	Fugl-Meyer UE	0.06	0.22	0.16	7	2.67	0.16	3.67	1.02	Y (p<.02)
		Fugl-Meyer LE	0.039	0.22	0.181	6	4.64	0.181	5.64	1.26	Y (p<.01)
		Self-selected walking speed (m/s)	0.158	0.595	0.437	3	2.77	0.437	3.77	0.622	Y (p<.05)
		6-min walk (ft)	0.205	0.397	0.192	6	0.94	0.192	1.94	0.98	N (p>.02)
2b	Teixeiria-Salmela et al., 1999[60]	Self-selected walking speed (m/s)	2.5	30.4	27.9	4	11.2	27.9	12.16	1.41	Y (p<.004)
		Stair climbing	N/A	N/A	N/A	N/A	N/A	N/A	N/A	1.99	Y (p<.005)
		Human Activity Profile (HAP, %)	3.2	40.4	37.2	3	11.6	3.2	12.63	N/A	Y (p<.001)
		Peak torque (composite[b])	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.004)
2b	Moreland et al., 2003[12]	2-in walking test (% change)	56.8	67.5	10.7	9	15.9	10.7	1.19	0.382	N (p>.50)
		2-min walking test: 6 months post D/C (% change)	106	97	9	11	8.5	9	0.92	N/A	Y (p<.05)
		Disability inventory (%)	24.39	25.63	1.24	81	5.08	1.24	1.05	0.061	N (p>.50)

Table 4. Continued

Level of evidence	Authors	Parameters	CER	EER	ARR	NNT	RBI	ABI	RR	Treatment effect size[a]	Statistical significance
2b	Moreland et al., 2003[12] *continued*	Disability inventory: 6 months post D/C (%)	1.02	3.54	2.52	40	2.47	2.52	3.47	0.875	N (p>.50)
2b	Ouellette et al., 2004[8]	6-min walk test (s)	0.0624	0.101	0.039	26	0.62	0.039	1.62	0.879	N (p>.40)
		Stair climb (s)	0.015	0.045	0.03	33	0.02	0.03	3.00	0.5618	N (p>.79)
		Chair rise (s)	0.0004	0.024	0.024	43	0.98	0.024	55.58	0.22	N (p>.84)
2b	Studenski et al., 2005[9]	QOL from Barthel Index	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.05)
		FIM	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.01)
		SF-36	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.05)
		Stroke Impact Scale	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.05)
2b	Yang et al., 2006[11]	Knee extension strength (lbs)	0.0131	0.32	0.307	3	23.4	0.307	24.43	3.133	Y (p<.005)
		Knee flexion strength (lbs)	0.11	0.12	0.01	100	0.09	0.01	1.09	3.133	Y (p<.005)
		Hip extension strength (lbs)	0.006	0.173	0.167	6	27.8	0.167	28.83	2.48	Y (p<.05)
		Hip flexion strength (lbs)	0.126	0.474	0.348	3	2.76	0.348	3.76	2.48	Y (p<.01)
		Self-selected walking speed (m/s)	0.0026	0.101	0.098	10	37.9	0.098	38.85	1.57	Y (p<.001)
		Cadence (step/min)	0.0082	0.065	0.057	18	6.9	0.057	7.90	1.06	Y (p<.001)
		Stride length (m)	0.013	0.034	0.021	48	1.62	0.021	2.62	0.482	Y (p=.005)
		6-min walk test (ft)	0.019	0.115	0.096	11	5.05	0.096	6.05	1.35	Y (p=.02)
		Step test (steps/15 s)	0.175	0.034	0.141	1	0.81	0.141	0.19	1.23	N (p>.50)
		Timed Up & Go (TUG)	0.007	0.116	0.109	9	15.6	0.109	16.57	1.615	Y (p<.001)
3b	Engardt et al., 1995[6]	Maximal vol eccentric knee ext (paretic leg) (avg. 60, 120, 180 deg/s)	0.15	0.26	0.11	9	0.73	0.11	1.73	0.617	Y (p<.05)
		Maximal vol concentric knee ext (paretic leg) (avg. 60, 120, 180 deg/s)	0.283	0.259	0.024	42	0.09	0.024	0.92	0.121	Y (p<.05)
		Self-selected walking speed (m/s) Eccentric group	0.105	0.024	0.045	20	0.2	0.045	0.23	0.281	N (p>.05)
		Self selected walking speed (m/s) Concentric group	0.123	0.037	0.086	12	0.7	0.086	0.30	0.711	Y (p<.05)

continued

Table 4. Continued

Level of evidence	Authors	Parameters	CER	EER	ARR	NNT	RBI	ABI	RR	Treatment effect size[a]	Statistical significance
3b	Engardt et al., 1995[6] *continued*	Max walking speed (m/s) Eccentric group	0.1	0	0.1	10	1	0.1	—	1.053 NS	N (p>.05)
		Max walking speed (m/s) Concentric group	0.1	0.123	8.13	10	1	8.13	1.23	0.823	Y (p<.05)
3b	Sharp & Brouwer, 1997[14]	Self-selected walking speed (m/s)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.196	Y (p<.05)
		Timed Up & Go (TUG)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.0375	N (p>.91)
		Stair climbing (ascending)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.1431	N (p>.39)
		Stair climbing (descending)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.1431	N (p>.37)
		Human Activity Profile (HAP, %)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.01)
3b	Weiss et al., 2000[10]	Self-selected walking speed (m/s)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.526	N (p<.73)
		Stair climb (sec)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.717	N (p<.07)
		Repeated chair stand time (s)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.02)
		Unilateral stance (paretic side)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N (p<.69)
		Motor Assesment Scale (MAS)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.02)
		Waking subscale (from MAS)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.03)
		Berg Balance Scale	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Y (p<.003)

Note: Studies are ordered by level of evidence and then chronologically within each level. This table presents whether there was improved muscle strength and function in each study. Sixty percent of the change scores on the dependent variables were statistically significant. For 22 of the 54 dependent variables reported from 11 studies, there were no significant gains post strength training. ARR = absolute risk reduction is difference in rates of bad outcomes between experimental and control participants in a trial; ABI = absolute benefit increase is the absolute difference in rates of good outcomes between experimental and control patients in a trial; CER = control event rate is the rate at which events occur in a control group; D/C = discontinued; EER = experimental event rate is the rate at which events occur in an experimental group; ext = extension; flex = flexion; LE = lower extremity; N = no; N/A = not available; NNT = number needed to treat with the experimental treatment for one additional person to benefit; QOL = quality of life; RBI = relative benefit increase is the proportional increase in rates of good outcomes between experimental and control patients in a trial; RR = risk ratio is the ratio of the risk in the treated group to the risk in the control group; UE = upper extremity; Y = yes.

[a]Treatment effect size = effect size (t values).

[b]Composite calculated from isokinetic torque of hip extension and flexion knee extension and flexion, ankle plantar flexion and dorsi flexion, all at 60 deg/s criterion.

0.13 m/s. This mean difference was revealed by calculating the mean change in gait speed for each study and weighting the difference by the number of patients who participated in the study. When averaging the change of gait speed from only the studies reporting significant changes, the average gait speed increased to 0.15 m/s. The largest change was reported by Duncan et al.[7] where mean change of gait speed was .25 m/s ($p < .05$) after 12 weeks of treatment. Kim et al.[13] reported the smallest change in gait speed, a nonsignificant increase of 0.04 m/s ($p < .65$) after 6 weeks of treatment

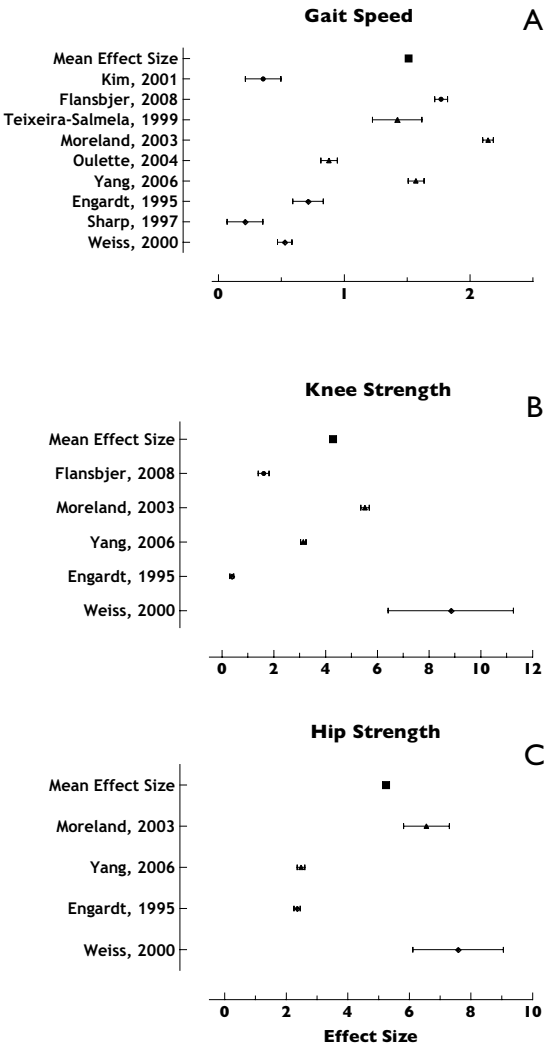

Figure 1. Mean effect sizes for gait speed and knee and hip strength. All effect sizes were considered a large difference between experiemental and control groups, per Cohen.[82] (A) Individual as well as average effective size for gait speed post strength training (in meter per second). The average effect size was 1.5. (B) Individual as well as average effective size for knee strength. The average effect size was 3.6. (C) Effect sizes for hip strength. The average effect size was 3.8.

that was similar between experimental and control groups (see **Figure 1A** for effect size). The average gait speed at the beginning of the studies was 0.617 m/s (*SD* ± 0.165 m/s) and was 0.715 m/s (*SD* ± 0.156 m/s) after interventions that included strengthening. Based on the definitions by Perry et al. (enumerated in **Table 5**), the participants in these strengthening studies would be categorized as community ambulators.[81]

The average effect size for improved gait speed was 1.5, which according to Cohen[82] is considered a "large" difference.[3*] The number needed to treat (NNT) is the number of participants needed to treat to achieve one additional favorable outcome.[83] To increase walking speed 0.13 m/s for persons poststroke, an average of 11 individuals in the chronic phase of recovery poststroke must be treated with strengthening exercises. **Figure 2** summarizes the NNT based on the outcomes of each individual study. Insufficient data were available to calculate the NNT for Sharp and Brouwer[14] and Weiss et al.[10] For Kim et al.[13] and Ouellette et al.,[8] the NNT was 35 and 26 participants, respectively. However, it is noteworthy that Kim et al.[13] reported only 39% power, which could also suggest the study was susceptible to a type II error. Moreover, in this study both the treatment and control groups produced similar improvements in gait speed.

The ABI is the difference in rates of positive outcomes between experimental and control participants in the same clinical trial.[83] The values are expressed as a percentage. The two highest absolute benefits for change in gait speed were calculated from studies by Duncan et al.[7] and Teixeira-Salmela et al.[60] with 44% and 27%, respectively. Sufficient data were not available to calculate the ABI for Weiss et al.[10] and Sharp and Brouwer.[14] With the exception of Duncan et al.[7] and Teixeira-Salmela et al.,[60] the remaining studies revealed a relatively low percentage of benefit in gait speed. Over nine studies, the ABI averaged 11%.

*Effect size is a term used to express the magnitude of a treatment effect. The strength of this approach lies in its independence from sample size. Cohen[82] established putative benchmarks helpful for clinical interpretation where an effect size of 0.2 is considered small, 0.5 is considered medium, and 0.8 is considered large. In terms of percentiles, an effect size of 0.0 indicates that the mean of the treated group is at the 50th percentile of the untreated group while 0.8 indicates that the mean of the treated group is at the 79th percentile of the untreated group. An effect size of 1.5 indicates that the mean of the treated group is at the 93.3rd percentile of the untreated group with 70.7% nonoverlap in observations between groups.

Table 5. Functional walking categories

Physiological ambulator	Limited household ambulator	Unlimited household ambulator	Most limited community ambulator	Least limited community ambulator	Community ambulator
0.1 m/s	0.23 m/s	0.27 m/s	0.40 m/s	0.58 m/s	0.80 m/s

Note: Functional walking categories as outlined by Perry et al.[81] Unlimited community ambulation is associated with self-selected walking speed of at least 0.80 m/s. The majority of participants included in the studies reviewed achieved this speed.

Muscle strength

Nine studies directly measured muscle strength using either a handheld dynamometer[6–13,69] or weight machines. Strength was assessed at the hip, knee, and ankle joints, but not all studies measured each joint. For example, Engardt et al.[6] and Sharp and Brouwer[14] measured only knee strength (see **Table 6** and **Figures 1B & C**). The percentage of change varied from 22% to 288%. The greatest apparent change in strength was reported by Moreland et al.,[12] where patients exercised using free weights or sand bag weights, emphasizing resistive type of work. This study also had the largest sample size (i.e., 133 participants), and the participants were drawn from acute inpatient rehabilitation. Either or both of these factors may help explain such remarkable gains in strength.[12] It is important to note, however, that we report post-treatment changes as a percentage change relative to baseline. In the case of Moreland et al., the absolute increase in weight resistance at the knee was only 1.0 to 2.1 kg compared to the lowest baseline measurement of 0.4 kg, which serves to inflate the percentage change in strength.[12] If the data from Moreland et al. are excluded, the overall percentage change in knee strength in the remaining eight studies is 39.2%.

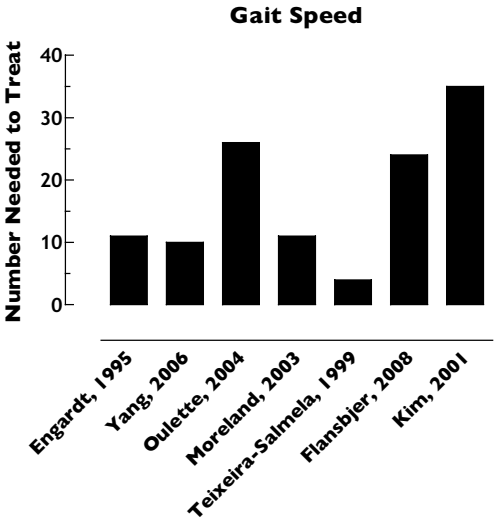

Figure 2. Number needed to treat for gait speed change. This graph shows number of participants needed to treat with strengthening exercises for one additional participant to make an average gain in the speed of their gait by 0.13 m/s. The zero values for Sharp and Brouwer[14] and Weiss et al.[10] are the result of insufficient data for calculation.

The greatest relative change in hip strength was observed by Moreland et al.[12] (122%). However, it is important to reiterate that these are small absolute changes equating to approximately 1.7 kg increases in the weight resistance at the hip. Teixeira-Salmela et al.[60] computed a composite strength score across the hip, knee, and ankle joints that averaged a 42.3% improvement. When averaged across the nine studies that reported quantitative data, the greatest percentage increase in strength was observed in the ankle (83%), followed by the knee (72.3%) and hip (51.3%). However, when treated as weighted averages to account for the considerable differences in sample size across studies, the largest effect was revealed at the knee (142%). Here it is important to note that knee strength was measured in all nine studies, whereas hip and ankle strength were tested in only five studies. In a relatively large cross-sectional study, Adams and colleagues investigated the residual strength for persons with moderate to severe hemiplegia noting that weakness appeared to be more pronounced distally than proximally,[84] with residual strength ranging from 37% in the plantarflexors to 68% in the hip flexors.[84,85] The cumulative data from this evidence-based review

Table 6. Summary of strength changes: hip, knee, and ankle

Authors	Number of participants	Hip	Knee	Ankle
Kim et al., 2001[13]	20	15.00%	85.00%	150.00%
Flansbjer et al., 2008[69]	24	Not measured	49%	Not measured
Teixeira-Salmela et al., 1998[60]	13	42.30%	42.30%	42.30%
Moreland et al., 2003[12]	133	122.00%	288.00%	137.00%
Ouellette et al., 2004[8]	42	Not measured	31.40%	51.10%
Yang et al., 2006[11]	48	32%	22%	34.50%
Engardt et al., 1995[6]	20	Not measured	29%	Not measured
Sharp & Brouwer, 1997[14]	15	Not measured	37%	Not measured
Weiss et al., 2000[10]	7	45%	67%	Not measured
Average	Mean: 36 Median: 20	51.26%	72.33%	82.98%
Weighted average		58.78%	141.97%	79.42%

Note: Strength changes are presented as the percentage change between baseline and postintervention in individual joints: hip, knee, and ankle. Strength was most consistently measured at the knee, producing a 72.3% improvement across all studies. Strength gains ranged from 15% to 150% across the hip, knee, and ankle, respectively. Several studies constructed composite strength scores obviating the ability to extract individual joint changes. Three of seven studies did not measure strength at the hip or ankle. Sample sizes are provided. Changes are expressed as percent, relative to baseline. Weighted averages adjust strength changes by number of participants per individual study.

suggest that the weakest muscles were associated with the greatest improvements in strength (see **Table 6**).

The effect sizes for gains in knee and hip strength (**Figures 1B & C**, respectively) varied by study. Due to lack of sufficient data reporting baseline scores and variance, these calculations could not be computed for the studies by Ouellette et al.,[8] Teixeira-Salmela et al.,[60] and Sharp and Brouwer.[14] Effect size was used to document the magnitude of the treatment effect. For both knee and hip strength, the effect sizes from Weiss et al.[10] were 8.84 and 7.58, respectively. As discussed previously, these are remarkable effect sizes confirming the presence of a positive treatment effect. It is interesting that Weiss et al.[10] had the smallest sample size (seven participants) and did not have a control group. The average effect size over all included studies was large for both knee (3.6) and hip (3.8).

Functional outcomes

Nine of the 11 included studies[7–9,12–14,60,69] measured functional activities and quality of life using self-assessment tools, such as SF-36,[65] MAS,[72] HAP,[77] NHP,[76] LLFDI,[66,67] CMSA,[63] or the Barthel Index.[62] From those nine studies, six[7–9,14,60,69] reported statistically significant changes from at least one of the self-assessment outcome tools (see **Table 3**). Ouellette et al.[8] reported no significant changes for performance-based functional measures such as 6-minute walk or stair climbing, yet participants' self-perceived changes in disability, as determined using the LLFDI, revealed statistically significant improvements. More specifically, the experimental group reported less self-perceived limitation in performing life tasks at home and in the community.[8] Although gait speed was not significantly improved in the pilot study conducted by Weiss et al., the quality of gait improved according to the gait subscale of MAS.[10] Overall, six studies reported decreases in self-reported limitations in performing tasks as well as improvements in quality of life.[7–9,14,60,69]

Adverse events

Previously reported studies of general physical fitness activities for stroke patients reported no deaths or symptoms related to vascular events, fractures, or other injuries.[86] However, in this evidence-based review, there were four adverse events reported from the study conducted by Ouellette et al.[8] These adverse events ranged from coronary artery stent placement, inguinal hernia, and ECG abnormalities to symptoms consistent with coronary artery disease.[8] The authors did not specify whether the adverse events were directly related to the strengthening program. There were no other

Table 7. Incremental cost-effectiveness

Authors	Cost of strength treatment (exp)	Cost of nonstrength treatment (con)	Cost difference exp vs. con	Incremental cost-effectiveness therapy/ treatment	Length of intervention (based on $100/ treatment)	Notes
Kim et al., 2001[13]	$1,350	$1,350	$0	$0	2x/wk for 10 wks	Control group same number of interventions
Flansbjer et al., 2008[69]	$2,000	$2,000	$0	$0	2x/wk for 10 wks	Control group same number of interventions
Duncan et al., 1998[7]	$2,400	$400	$2,000	$60,000	3x/wk for 8 wks	Control group follow-up every 2 wks (for 8 wks)
Teixeira-Salmela et al., 1999[60]	$3,000	$0	$3,000	$66,667	3x/wk for 10 wks	Control group had no intervention
Ouellette et al., 2004[8]	$3,600	$3,600	$0	$0	3x/wk for 12 wks	Control group same number of interventions
Yang et al., 2006[11]	$1,200	$0	$1,200	$70,588	2x/wk for 6 wks	Control group had no intervention
Weiss et al., 2000[10]	N/A	N/A	N/A	N/A	3x/wk for 10 wks	No control group available
Sharp et al., 1997[14]	N/A	N/A	N/A	N/A	N/A	No control group available
Engardt et al., 1995[6]	N/A	N/A	N/A	N/A	2x/wk for 6 wks	Unable to calculate
Total incremental cost				$31,017		

Note: This table presents incremental cost-effectiveness for each study that provided sufficient data. The average incremental cost between strengthening and nonstrengthening interventions is reported in conjunction with the total cost of treatment. Strengthening appears to be cost-effective as a neurorehabilitation intervention. Exp = experimental; Con = control; N/A = not applicable.

reported adverse events in the remainder of the studies.[6–14,69]

Cost-effectiveness

The incremental cost-effectiveness ratio (ICER) was used to assess the incremental benefits of producing an average change in gait speed of 0.13 m/s resulting from rehabilitation interventions that involved strengthening as compared to nonstrengthening interventions. The ICER quantifies the added cost incurred to produce one additional benefit from the target intervention.[86] Here benefit refers to a mean increase in gait speed of 0.13 m/s. Implicit in this calculation is the absence of adverse events and, per the parameters of this evidence-based review, exacerbation of spasticity. Costs for both experimental and control interventions were based on a rate of $100 per physical therapy session. The data are summarized in **Table 7**. Only nine studies were included in the calculation, because those reported by Sharp and Brouwer[14] and Weiss et al.[10] did not provide sufficient data for calculation of the ICER. Based on the nine studies included, the additional cost involved in producing a 0.13 m/s change in walking speed in a person poststroke would be $31,017. The minimal detectable change for gait speed in persons poststroke ranges from 0.07 to 0.36 m/s.[87] Thus, the mean change of 0.13 m/s produced across the nine strengthening interventions considered for this calculation can be considered both clinically important and, using Perry's functional walking categories, functionally significant[81] (see **Table 5**). According to various sources, when performing cost utility studies a threshold value of $50,000[88] is considered a benchmark that justifies treatment.[89] Evaluated against this benchmark, the incremental cost of $31,017 involved in producing a clinically and functionally significant improvement in gait speed can be considered cost-effective.

Spasticity

The final element of our primary question was to determine whether strengthening exacerbates spasticity in persons poststroke. As mentioned previously, there is some evidence that neither high-exertion activities nor strengthening exercises increase spasticity, yet the issue remains prominent in clinical thinking.[90] Four of the 11 studies formally measured spasticity using either the Pendulum test[91] or

the modified Ashworth scale.[92] Both tests are used to quantify spastic hypertonia, and reliability has been established.[92,93] Consistent with our expected findings, Teixeira-Salmela et al.[60] reported no increased spasticity as measured by the Pendulum test.[60] In addition, Sharp and Brouwer,[14] Moreland et al.,[12] and Flansbjer et al.[69] reported no increased spasticity during or post strength training using the modified Ashworth scale.[12,14]

Discussion

This evidence-based review was conducted to determine whether strengthening exercise is beneficial for persons poststroke. The studies meeting the evidence-based criteria consistently provided good evidence that lower extremity muscle strength was increased after strengthening. However, significant variability was revealed in the relative strength gains reported (15% to 288%).[6–14,60,69] This variability may be explained, in part, by both the heterogeneity of clinical presentation, including the severity and chronicity of poststroke hemiparesis, and the lack of consistent inclusion criteria. An additional parameter that contributes to the tremendous variability of outcomes is the variety of strengthening approaches utilized across studies. Despite these inconsistencies, measurable gains in lower extremity strength were revealed in adults poststroke; these gains were associated with functional improvements and occurred without adverse effects.

Primary question

Is there evidence that high-exertion activity exacerbates spasticity?

The clinical belief that strengthening persons poststroke exacerbates spasticity was not confirmed by the studies included in this evidence-based review. Indeed, none of the 11 clinical studies reported increased spasticity or hypertonia during or after a course of treatment.[6–14,69] Other investigations of neuromotor function poststroke have addressed the issue of how significantly spasticity contributes to motor dysfunction. Ada and colleagues assessed the level of spasticity (e.g., stretch reflex threshold) during self-paced walking for persons with poststroke hemiplegia as compared to neurologically normal participants.[34] Stretch reflex thresholds observed during walking were similar in magnitude between hemiplegic and control participants, thus providing no evidence of increased resistance to dorsiflexion range of motion attributable to hyperreflexia. Furthermore, other studies have failed to report an increase in spasticity during or following high-exertion physical activities.[86,90]

Is there evidence for significant functional outcomes following strengthening?

Prevailing clinical thought argues that functional improvements emerge only from task-specific training approaches.[94,95] Despite the variability of methods for both gait assessment and actual changes in walking function, the 11 studies reviewed revealed an average change of 0.13 m/s in gait speed following strengthening. Although apparently small in absolute terms, based on categories developed by Perry[81] a change of 0.13 m/s could potentially advance a hemiparetic person to a higher level of walking function (see **Table 5**). This is especially the case for individuals in the lower range of functional walking categories (i.e., physiologic to unlimited household ambulators), where apparently small absolute gains in walking speed represent functional gains that advance an individual from one category to the next. For example, an increase in gait speed from 0.23 m/s to 0.27m/s suggests that a limited household ambulator could advance to unlimited household ambulation.[81] Similarly, an increase of 0.13 m/s would afford a limited community ambulator enhanced function for unlimited community ambulation. Here it is important to recognize that our calculations of gait speed changes are quite conservative in that they are derived from post hoc analysis and draw from various approaches to assessment of gait speed. In all likelihood, larger improvements in gait speed exist, and thus functional improvements apply not only to individuals at the low end of the continuum. In future studies, careful attention to the method of gait assessment will expand our understanding of how, specifically, improved strength enhances locomotor function. Moreover, studies using instrumented gait analysis will eluci-

date both neural and biomechanical mechanisms that contribute to improved walking function.

Is there evidence that lower extremity strengthening improves participation and quality of life?

Self-assessment functional scores from SF-36, GDS, and Sickness Impact Profile revealed statistically significant improvements following strength training.[7,9] Other self-assessed functional outcome including the HAP and NHP improved from baseline as result of strength training.[14] It is possible that strengthening may also decrease depression and encourage positive self-perspective.[6–14] Other positive outcomes include improved sleep patterns, influence on bone mass, decreased insulin resistance (for type 2 diabetes mellitus), and normalized blood pressure.[96–98]

Limitations of this evidence-based review

Many of the studies discussed in this evidence-based review had limitations in the research design affecting both internal and external validity. For example, Moreland et al.[12] reported a 20% dropout rate and Weiss et al.[10] reported two of nine participants dropped out due to personal reasons resulting in an unusually small sample size, even for a pilot study. Despite these limitations, the mean and median sample sizes were 36 and 20 participants, respectively, across the 11 studies. Small sample size limits both the power of a study to detect changes and the generalizability of the findings. Sample bias must also be taken into account. The majority of the patients were recruited on a volunteer basis. Persons who volunteer may be more motivated to improve but certainly differ from those who do not volunteer, thus affecting the generalizability of this finding to the greater population of persons poststroke.

An important limitation of this evidence-based review is the absence of follow-up (e.g., retention) measurements in the majority of the studies reviewed. Only four of the studies (Moreland,[12] Studenski,[9] Sharp,[14] and Flansbjer et al.[69]) included follow-up measurements over a period ranging from 6 to 16 weeks following treatment. Moreover, not all studies included a control group to monitor for effects of natural history of recovery and/or to provide comparison between strengthening and nonstrengthening interventions (Weiss,[10] Sharp,[14] and Engardt et al.[6]). To elevate the research evidence, the design of future studies should include long-term (i.e., 6 and 12 months postintervention) follow-up studies to monitor for retention of treatment effects, control groups receiving dose-equivalent treatment with nonstrengthening intervention, and blinding of evaluators.

Maximal strength capacity monitored using 1-RM contractions or other means was not measured in all studies included in this evidence-based review. For example, Moreland et al.[12] used self-perceived exertion in response to unspecified amount of ankle weights to establish and adjust the training stimulus. Other variations included Studenski et al.[9] who used theraband to assess strength. The use of nonstandard measurement methods and noncalibrated loading compromise the ability to estimate objective changes in performance and objectively evaluate the benefits of strengthening for neurologic populations.

Finally, a variety of self-assessments of functional outcomes were reported across the 11 studies. These included HAP, NHP, CMSA, Barthel Index, and SF-36. Future studies would benefit not only from careful attention to study design but also from selection of appropriate outcome measures that facilitate comparisons across studies.

Gaps in research

Training parameters

A variety of strengthening protocols was prescribed among the clinical studies reviewed. One approach involved training the participants with adequate resistance based on 60%–80% of 1-RM.[99,100] This approach is well documented and straightforward but is difficult to apply when using sand bags or elastic bands rather than dynamometers or weight stacks. For example, Moreland et al.[12] and Studenski et al.[9] used the patient's self-report of effort using elastic theraband or arbitrary weights and, further, failed to specify the number of repetitions in the strengthening protocol. This approach brings to question whether the intensity was sufficient to produce a training stimulus and challenge the individuals' maximum capacity. Few

studies in the rehabilitation literature assess these critical training parameters.

Another gap in the research was the type of strength training exercises included in some of the rehabilitation protocols. Only one study (i.e., Engardt et al.[6]) separated treatment groups into eccentric and concentric loading. Although both types of exercises produced significant strength gains, the functional gains (e.g., symmetrical body weight distribution during sit to stand) appeared greater for the eccentric trained group (i.e., Engardt et al.[6]). Such differential effects of training mode warrant further investigation to better understand their prevalence, persistence, and generalizability to functional performance.

Type of lesions and stage poststroke

Subject-specific characteristics including age, gender, premorbid condition, significant comorbidities, and time poststroke were poorly controlled across studies. Indeed, significant variations in baseline functional levels and chronicity were noted both within and between the reviewed studies. One additional variable, lesion burden, was wholly unaccounted for in any of the 11 studies. Because the mechanism of injury, lesion location, lesion size, and extent strongly influence the capacity for recovery, future studies that include this information and control for lesion characteristics between subject groups will greatly advance the literature.

Quantifying muscle strength

The 1-RM is the gold standard to objectively quantify strength performance[79,101] yet is rarely, if ever, used clinically. An alternate approach to strength assessment is dynamometry. Handheld dynamometers (e.g., myometers) can be used in regular clinical practice to augment manual muscle testing techniques yielding objective and reliable[102,103] indicators of isometric force production. Isokinetic dyamometers (i.e., Biodex, Cybex, Kin-Com) allow assessment of dynamic force production either isotonically (constant load) or isokinetically (constant velocity). Further, isokinetic dynamometers provide means to constrain movement speed and assess dynamic force production through the full range of motion (e.g., muscle lengths). The reliability,[104–106] validity,[41,105] and minimal detectable changes[104,105] of dynamic strength measurements obtained through dynamometry have been established in persons poststroke. Comprehensive, accurate measurement of force production capacity involves testing through the full range of motion across a range of movement speeds (i.e., 30 deg/s to 210 deg/s).[105] Among three muscle strength measures (peak torque, total work, average power) coupled with different angular velocity, Hsu and colleagues reported that normalized peak torque and total work appeared to be more reliable and quantifiable than the normalized average measurement ($p < .05$ for hip flexion and $p < .01$ for knee extension and ankle plantarflexion).[107] In contrast, some studies in this review quantified muscle strength by the color of the theraband, the amount of sand bag weights that could be lifted, or by self-report.[7,9,12] It is not surprising that these noncontrolled techniques yield less convincing information regarding the effectiveness of strengthening to either produce strength gains or functional benefits of strengthening. Advancement of this area of research and elevation of the evidence require utilization of the most objective and sensitive measurement techniques.

Incorporating various speeds to strength training

Translation of strength gains to functional performance requires an enhanced capacity to produce force in the dynamic conditions required of the functional task. Thus, exposing the affected limb to different movement speeds appears to be an important component to effective resistance training for persons poststroke. Many functional activities require the limbs to generate increased forces over a range of movement speeds.[106] For example, functional reaching tasks involve elbow extension at speeds of up to 400 deg/s in healthy adults and about 210 deg/s for persons poststroke.[106] Thus, effective resistance training should incorporate both an element of loading (i.e., intensity) and an element of speed (i.e., specificity).

Implications for current clinical practice

Taken together, the literature to date provides no evidence that strengthening exacerbates spasticity.

Thus, the primary rationale that historically proscribed high-exertion activity in neurorehabilitation can be reconsidered.[33] Further, the available evidence indicates that strengthening is indeed effective for counteracting muscle weakness and enhancing gait function in persons in the chronic phase poststroke. Thus, there appears to be a link between enhanced strength and improved functional performance. These positive functional outcomes are accompanied by improved self-perception and enhanced quality of life, again without increasing hypertonicity. In summary, the recommendation for this evidence-based review would be somewhere between grade 2-3B.

Even though the highest quality research evidence is not yet available, the few early findings highlighted in this evidence-based review are sufficiently strong to reconsider the traditional barriers to strengthening in neurologically impaired populations. The results of these studies further motivate the design of larger clinical trials that will produce definitive level 1 evidence. There is also a need to systematically test specific parameters of resistance training in an effort to identify the optimal approach to promoting recovery of motor function effectively and efficiently. Finally, due to limited long-term follow-up studies to date, the persistence of resistance training effects remains unclear. These questions will guide future research in neurorehabilitation.

Table 8. Recommended parameters for resistance training

Parameters	
Level of resistance	1-RM at 60%–80%
Number of repetitions	Max of 12 repetitions per set
Number of reps	3 sets each, 8 to 10 exercises
Number of times per week	3 sessions
Total training interval	Minimum period of 6 to 12 weeks before adjustment of program
Contraindications	• Non-neurologically stable patients • Postsurgical patients • Severe osteoporosis • Acute orthopedic or joint injuries • Hemophilia or other blood disorders • Severely limited ROM
Precautions	• Valsalva • Monitor vital signs, especially blood pressure
Type of machines	• Biodex, Isokinetic, circuit-training, or weight machines
Frequency of reassessment	• 1-RM (e.g., maximal strength) to be tested every 2 weeks
Incorporate to which type of activates?	• Functional task-specific activities
Stage of stroke	• Subacute or chronic and medically stable
How to reassess strength	• Recommend dynamometer or weight stack apparatus • If unavailable, use handheld dynamometer (but cannot quantify dynamic strength)

Note: Strength training is recommended for patients with hemiparetic weakness. The recommended parameters for strength training are summarized. These can be considered guidelines for implementation in current clinical practice. 1-RM = one-repetition maximum; ROM = range of motion.

Recommendations for neurorehabilitation practice

There is currently no gold standard to guide the development of strengthening protocols for persons poststroke. However, based on the clinical studies reviewed, the basic parameters recommended for implementation in clinical practice are listed in **Table 8**. These guidelines can be augmented using principles well established in able-bodied populations[79,108] and complemented by sound clinical judgment. Hypertonia/hyperreflexia, if prominent, is a precaution rather than a frank contraindication to strengthening and should be monitored during evaluation and treatment. There is no evidence that resistance training will worsen hypertonia. Other recommended components of resistance training include using various criterion speeds (i.e., 30, 90, and 120 deg/s) to challenge the capacity to produce force under varying dynamic conditions. Systematic baseline strength assessment using either the 1-RM approach or dynamometry is strongly recommended. Equally important is careful reassessment of strength at regular intervals, at least every 2 weeks, to monitor and ensure intensity of the training stimulus. Finally, incorporation of functional activities is recommended in both assessment and treatment, as their improvement is the ultimate goal of therapeutic intervention.

Moreland et al.[12] reported that persons more severely impaired poststroke, as defined by the motor recovery component of CMSA,[63] revealed poorer results from strength training. More specifically, those who presented with lower extremity motor recovery less than Stage 4 consistently demonstrated poorer

functional results compared to those who achieved at least Stage 4. Therefore, those individuals with more limited motor recovery may not immediately gain from strengthening. Studies have not been conducted to determine whether significant improvements may be revealed in these more severely impaired individuals in the longer term (e.g., weeks to months). The timing of intervention with strengthening is also relevant. There is some observation that strengthening in the periacute and acute rehabilitation stages poststroke is less effective, in part because there are myriad medical issues to resolve and tolerance to physical activity is limited.[15] Such topics remain to be addressed in ongoing and future investigations.

Its Not About the Muscle!

Especially in neurorehabilitation, it is important to see beyond the trivial intention of resistance training, that is exercising to induce physiological changes in muscle (i.e., hypertrophy) and mechanical effects such as increased joint stability. It has long been established that strength and strengthening involve neural and muscular factors.[109] The neural aspects of strengthening are often overlooked in the focus on the mechanical and metabolic aspects of muscle. In this light, it is significant that weakness is among the most prominent characteristics of poststroke hemiparesis, yet evidence of muscle atrophy is equivocal, at best, even in chronic hemiplegia.[26] Clearly, hemiparetic weakness is more complex than muscular factors alone, which underscores the need to delineate the rationale for strengthening interventions. Here it is important to recognize that effective resistance training in neurorehabilitation capitalizes on the capacity to induce significant neural adaptations, such as improved central motor activation, that elevate the capacity of the neuro-musculo-skeletal system to perform movement tasks.[15] The challenge in clinical application is to develop resistance training paradigms that optimize these neural adaptations.

Rather than the historical perspective which questioned *whether* strengthening interventions are appropriate in neurorehabilitation, the contemporary focus has shifted to understand *why* resistance training is effective and how to improve its effects across a broad range of persons poststroke. Definitive conclusions regarding the optimal timing, frequency, and duration of strengthening programs have not been clearly defined from the reviewed studies. However, there is evidence in nondisabled individuals that changes in cortical excitability occur rapidly, as early as following 4 weeks of strengthening.[110] Carroll and colleagues demonstrated that after resistance training to the hand muscles, the magnitude of the evoked response to transcranial stimulation was reduced at a given level of torque when compared to the baseline.[111] This phenomenon suggests that fewer motor neurons are activated posttraining due to enhanced efficacy of synapses between corticospinal neurons and spinal motorneurons, which ultimately alters the input-output properties of the corticospinal pathways and supplements changes in processing at the cortical level.[111] Furthermore, changes in the pattern of motor unit recruitment and increases in the neural drive may be inclusive factors after strength training.[112] Thus, based on the principles of neuroplasticity, strength or resistance training plays an important role in improving motor unit recruitment,[6] because the capacity to produce muscular force is primarily a neural phenomenon with task-specific regulation of neural activity.[15] Despite this evidence of neuroplastic adaptation, the severity of supraspinal lesion may render 4 weeks of strength training insufficient for individuals poststroke. Thus, longer treatment duration may be warranted, which remains a topic for future research studies.

Conclusion

The recommendations from this current review draw from studies ranging only from levels 1B to 3B,[70] yet there is sufficient evidence to support a change in clinical practice. For persons in a stable phase of recovery poststroke, this current review based on recent clinical studies confirms that strengthening can positively increase strength, promote functional improvement, and potentially change quality of life without increasing spasticity. The primary mechanisms for force production and force control reside supraspinally and in spinal segmental centers. Even though the outward manifestations may share similarities among nondisabled, orthopaedic, and neurologic populations, resistance training constitutes a potent form of

neuromotor training. Ongoing efforts are needed in both neurorehabilitation practice and research to better understand effective means to optimize neural adaptations.

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