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Strength Training in Individuals with Stroke

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Abstract

Purpose—This paper reviews the mechanisms underlying the inability to generate force in individuals with stroke and summarizes the effects of strength training in these individuals. In addition, a systematic review of studies that have incorporated progressive strengthening interventions in individuals with stroke is presented.

Summary of Key Points—Central (e.g., motor recruitment) and peripheral (e.g., muscle atrophy) sources may alter muscle strength in individuals with stroke and further investigations are needed to partition and quantify their effects. As to the effect of strength training interventions in individuals with stroke, the majority of studies (albeit with small samples) that evaluated muscle strength as an outcome demonstrated improvements. With regard to the effect of strength training on functional outcomes in individuals with stroke, positive outcomes were found in less rigorous pre-test/post-test studies, but more conflicting results with controlled trials.

Conclusions—Although there is some suggestion that strength training alone can improve muscle strength, further research is required to optimize strength training and the transfer of these strength gains to functional tasks in individuals with stroke.

Keywords

cerebrovascular accident; resistance; exercise; muscle

INTRODUCTION

Stroke is the leading cause of serious, long-term adult disability;[1] the absolute numbers of individuals with stroke are increasing as a result of an aging adult population, coupled with an ever improving survival rate following stroke.[2]

A cerebrovascular accident or stroke is caused by an interruption of the flow of blood to the brain or by a rupture of blood vessels in the brain. The clinical consequences of a stroke depend upon the anatomical regions of the brain affected, as well as the volume of tissue damage. Acute manifestations from the stroke, in addition to chronic musculoskeletal adaptations (e.g., contractures) contribute to resulting weakness on the side contralateral to the brain injury (i.e., hemiparesis).

In the past, strength training in persons with spasticity has been controversial. Bobath[3] advocated that decreased muscle strength was due not to weakness but to the opposition of

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spastic antagonists and that strenuous activity would increase spasticity and reinforce abnormal movement. Thus, unlike intensive cardiac rehabilitation and orthopaedic programs, individuals with stroke have historically undertaken moderate exercise programs, with much of the treatment aimed at inhibition of abnormal muscle tone and facilitation of normal movement patterns.[3] However, stroke rehabilitation is currently undergoing a major re-evaluation based on recent physiological and clinical evidence. For example, Sharp and Brouwer[4] found that individuals with stroke could undertake intensive muscle strengthening without any increase in spasticity, as measured by the leg pendulum test and simultaneous EMG recordings. In addition, Ada et al.[5] measured ankle stretch reflexes under simulated walking conditions and concluded that it is unlikely that spasticity of the gastrocnemius muscle contributes to the walking problems common in ambulatory persons with stroke.

Increasing recognition of the importance of muscle strength in stroke recovery is based, in part, on studies that have demonstrated a relationship between muscle strength and function in persons with stroke. Paretic muscle strength is related to a number of activities of daily living in individuals with stroke, including bringing the hand to the mouth,[6] balance,[7] walking speed,[8–10] ability to rise from a chair[11] and stair climbing.[8] Muscle strength of the involved side of the body is also inversely related to falls[12] and the inpatient stroke rehabilitation length of stay.[13] Significant relationships also exist between non-paretic lower extremity (LE) muscle strength and functions such as gait and stair climbing.[8] Reports of non-paretic limb weakness[14] suggest that muscle strength of both limbs should be considered. Although correlations do not imply causation, they do generate theories and hypotheses which can be tested through clinical trials.

This paper reviews 1) mechanisms underlying muscle strength following stroke; 2) effects of strength training on muscle and functional outcomes; and 3) safety and monitoring issues relevant to strength training in individuals with stroke.

EFFECTS OF STROKE ON MUSCLE STRENGTH

Stroke results in a reduction in muscle strength (ability to generate force or torque) predominantly on the paretic side, with mild weakness on the ipsilateral, non-paretic side when compared to healthy individuals. The effects on the non-paretic side are attributed to the small percentage of descending cortical tracts that originate from the lesion site and remain ipsilateral.[15] The sedentary lifestyle that often ensues following stroke may also contribute to the diminished muscle strength of the person with chronic stroke compared to healthy age-matched controls.

Patterns of Muscle Strength Deficits

Changes in muscle strength resulting from a stroke depend on the location and volume of the brain injury, as well as the time since stroke (e.g., acute versus chronic). Such factors may contribute to discrepancies as to which muscles are more affected than others following a stroke. Although there is little evidence that either flexors or extensors are selectively affected following a stroke, there is evidence to support the clinical observation that distal muscles of the paretic side have greater strength deficits (relative to the non-paretic side) compared with proximal muscles.[16,17] This evidence concurs with observations in healthy

subjects of greater facilitation of the corticospinal system for distal muscles compared to proximal muscles.[18,19] Thus, greater deficits would be expected to occur in distal muscles following disruption of these pathways. The cortical control of muscle activity can be assessed with the functional coupling (coherence) between the cortical electroencephalogram (EEG) and muscle electromyogram (EMG). Following stroke, there is a smaller EEG-EMG coherence for distal upper extremity (UE) muscles, but not for more proximal muscles;[20] this finding corresponds to the observation of greater central deficits to distal muscles. Although a retrospective review of medical records by Andrews and Bohannon[21] did not support this proximal-distal gradient in individuals with stroke, prospective studies have supported it.[16,17]

Recovery of muscle following stroke may also be influenced by whether that recovery involves the UE versus the LE. The magnitude of motor impairments (as assessed by the Fugl-Meyer scale) appear to be similar between the UE and LE during the rehabilitation phase of recovery,[22,23] although Desrosier et al.[22] reported that the rate of motor recovery was greater for the UE following hospital discharge.

Muscle Atrophy

What are the causes underlying the inability to generate force in individuals with stroke? Electrophysiological evaluations of motor unit recruitment have estimated that the number of functioning motor units following a stroke is reduced. For example, McComas et al.[24] reported no changes for subjects less than two months post-stroke, but a 50% reduction in functioning motor units of the paretic extensor digitorum brevis muscle compared to its non-paretic counterpart by 6 months following stroke. Hara et al.[25] reported that the paretic abductor pollicis brevis had only about 60% of the functional motor units of the non-paretic side in individuals 2 to 7 months following a stroke. Theses changes were attributed to transynaptic degeneration of the alpha motorneurons resulting from a lack of descending input. In addition, histochemical analyses of muscle biopsies following a stroke have found selective atrophy of type II muscle fibers and an increase in percentage of type I fibres. However, the exact nature of the atrophy may be dependent on the specific muscles tested, duration of stroke and level of physical activity.[26–29] Dual-energy X-ray absorptiometry and computerized tomography scans also have indicated reductions of lean tissue mass and increased intramuscular fat deposition within the hemiparetic limb of persons with chronic stroke.[30]

More recently, Landau and Sahrmann[31] attempted to quantify the peripheral and central regulation of the tibialis anterior muscle. In 17 individuals with acute stroke, they reported a significant mean reduction in the ability to voluntarily generate torque using the paretic tibialis anterior, but an increase in torque when the paretic tibialis muscle was electrically stimulated when compared to the non-paretic side. Furthermore, the torque response was similar between sides in response to electrical stimulation for 14 individuals with chronic stroke and was within the range of healthy controls, but the voluntary torque of the paretic side was again lower when compared to the non-paretic side. The finding that individuals with acute and chronic stroke can produce normal levels of torque when electrically stimulated, suggests that contractile capacity may not differ between sides and central

regulation (e.g., motor unit recruitment and firing rate) may be the predominant factor contributing to muscle weakness following stroke. The discrepancies between the Landau and Sahrmann[31] paper and documented muscle atrophy in individuals with stroke[26–30] arise, in part, from our lack of understanding of the contribution of observed changes in motor unit number and fibre composition to overall muscle strength. Morphological changes (e.g., muscle atrophy) have not been found to relate to sensory impairment,[26] site of lesion,[26] duration of injury,[32] or ambulation status,[32] although low levels of daily physical activity were found to relate to muscle atrophy following a stroke.[33] Further studies that partition the central and peripheral regulation of muscle in individuals with stroke are needed to confirm the findings of Landau and Sahrmann[31] and to determine whether these findings can be generalized to other muscle groups.

Motor Unit Function

There is ample evidence that motor recruitment and discharge firing rate are altered following a stroke and that this will contribute to an inability to generate force. Motor units of the paretic side are recruited at lower levels of absolute force compared to the non-paretic side[34] and there is a reduced ability to increase motor unit discharge rate during voluntary force increases.[28,34,35]

Abnormalities in motor recruitment and discharge rate are further complicated by the interactions of different muscles and joint positions during voluntary muscle contractions. Ada et al.[36] reported changes to the torque-angle relationship following stroke that resulted in decreased production of force at short muscle lengths. Bourbonnais et al.[37] found that the magnitude and contribution by elbow muscles was different between the paretic and non-paretic sides. In the paretic UE, maximum activation took place in a plane outside of the elbow flexion-extension motion and towards external humeral rotation and shoulder girdle elevation. In addition, increased co-activation of agonists/antagonists during dynamic functional movements (e.g., gait[38]) may limit agonist force production. Furthermore, individuals with stroke also demonstrate a delay in the initiation and termination of muscle contraction as measured by EMG activity.[39]

Passive Mechanical Properties

The mechanical properties of muscle and connective tissues also influence the ability to generate force through a range of motion. For example, hypertonia is the increase in joint resistance to passive movement and may result from spasticity (i.e., hyperactivity of the stretch reflex), and from viscoelastic changes in muscle and connective tissues.[40] Increasing evidence suggests that altered mechanical properties in muscle and connective tissue following brain injury are more important than influences from altered reflexes.[41–43] According to O'Dwyer et al.[42], spasticity did not relate to muscle weakness, leading these authors to suggest that the amount of attention directed to increased tonic stretch reflexes associated with spasticity exceeds its actual effects. In an *in vitro* study, Friden and Lieber[44] found that when tension was removed from spastic sarcomeres (biopsied from UE muscles from persons with cerebral palsy), the sarcomeres at resting length were shorter and stiffer than healthy sarcomeres. The same scientists[45] also demonstrated *in vivo* that these sarcomeres were abnormally elongated at all wrist angles, suggesting that stretching the

stiffer sarcomeres contributed to increased passive mechanical stiffness. Because sarcomeres generate their greatest contractile force at maximal overlap between the cross-bridges, stretched spastic sarcomeres would lead to a reduced ability to generate force.

Central and peripheral sources that may alter muscle strength in persons with stroke have been identified. Future research should quantify the contributions and interactions of these components to the resulting muscle strength and to the performance of natural functional movement.

MEASUREMENT OF STRENGTH

The most common research measure used to assess strength in persons with stroke is peak or average torque during isometric or isokinetic contractions. These measures have been shown to be reliable.[46,47] One small study[48] (n=10) reported poor test-retest reliability for the knee flexor torque at 60 degrees/second in individuals with mild and moderate stroke which may have resulted from the selected angular velocity. In a similar group of subjects, 13 of 20 participants could complete knee movements at 30 degrees/second, but not at 60 degrees/second.[47] Ordinal muscle strength grades (0 to 5) are commonly used in the clinical setting. Unfortunately, their sensitivity to small, but clinically relevant changes is questionable.[49] Hand-held dynamometers are an inexpensive alternative in which reliability has been established for individuals with stroke.[46]

Other parameters of muscle strength may also be important but need further validation. For example, McCrea et al.[50] found that the time to generate force and time to reduce force were longer for UE paretic muscles compared to those on the non-paretic side. In addition, the time to generate force was greater for the non-paretic side of persons with stroke compared to healthy controls. A fatigue test (holding a sustained isometric contraction) may also provide complementary information about the integrity of the muscle. However, Riley and Bilodeau[51] tested elbow flexion fatigue in persons with stroke and they found that increasing compensations at other joints (e.g., increased shoulder torques), in addition to the lower level of voluntary activation that they measured during a fatigue test in these individuals, could confound this measurement.

EVIDENCE OF CLINICAL TRIALS WHICH USE GRADED MUSCLE STRENGTHENING

Methods Used for the Systematic Review

MEDLINE (from 1966 to March 2004) and CINAHL (from 1982 to March 2004) database searches were performed using combinations of the key words stroke, cerebrovascular accident, rehabilitation, muscle, strength, exercise and clinical trials. Articles identified from this process were then reviewed for additional references. Neither theses or conference proceedings were included. Studies which evaluated the effect of graded muscle strengthening on muscle strength or functional outcomes were identified but those which included treadmill training, balance and functional tasks, walking practice, electrical modalities (e.g., functional electrical stimulation), endurance exercise (e.g., arm or bicycle ergometer) and constraint-induced therapy were excluded. In these cases, one would not be

able to attribute the improved functional outcomes solely to muscle strengthening. However, studies which examined the effect or added effect of a graded strengthening program, compared to uni- or multi-dimensional programs, were included. The methodological rigor of the study was indicated by the level of evidence (Level I to V) supported by each study design as described by Sackett[52] (Table 1). Although a newer, revised version of the levels of evidence now includes the categories of systematic reviews with homogeneity and additional sub-categories[53], the original version[52] was utilized because the literature in this dataset did not include these new categories. Given the lack of standardization and consensus in applying grades of recommendations,[54,55] (e.g., A, B, C and I, II, III), the descriptor "Good" was used to describe evidence supported by at least one Level I study, "Fair" evidence was supported by at least one Level II study, and "Insufficient/Poor" evidence was supported by Level III, IV, or V studies.

Studies that included a control group comparison were also evaluated by the PEDro scale which is an 11-item scale to assess the quality of clinical trials in physical therapy.[56] Item one assesses external validity and is given a YES or NO, and the other ten items assess internal validity and are each given one point if the criterion is satisfied for a maximum score of 10. The PEDro score for each controlled clinical trial was searched within the Physiotherapy Evidence Database (PED) (www.pedro.fhs.usyd.edu.au). The PED has established PEDro scores which have been verified by two independent persons. For those studies that did not have existing PEDro scores, two individuals independently scored the remaining clinical trials, with a third person if consensus was not reached.

Results of the Systematic Review

The search resulted in 12 studies[4,57–67] with the majority involving the LEs (Table 2). Nine of the studies[57–59, 61–64,66–67] were controlled trials and their PEDro scores ranged from two to seven (Table 3). The small number of studies that evaluated the effect of muscle strength in persons with stroke reflects the highly integrative and eclectic rehabilitation process, including working towards functional tasks, strengthening exercises, mobility goals, electrotherapeutic modalities, exercise on stationary bicycles or treadmills, and movement facilitation. However, the paucity of studies that examined muscle strength versus a control or sham group makes it difficult to ascertain the specific contributions of strength training to the overall improvements of the client.

Three Level I or II RCT studies[57–59] evaluated the effect of LE muscle strengthening (in addition to regular physical therapy) during the inpatient stroke rehabilitation phase. Two of the studies[57,59] found no group differences for functional walking measures, while the third[58] found greater strength and better activities of daily living immediately following 4 weeks of a strength program. However, these effects were not retained compared to the conventional group at a 2 month post-treatment assessment. Results of these studies fail to show substantial improvements in the functional outcomes measured in these settings. The multi-disciplinary, full-day treatments in which patients receive numerous physical activities including physical therapy, nursing care (practice of self-care, transfers), occupational therapy and recreational therapy, make it difficult to isolate treatment effects of strength training. Hence, the impact of an additional 15–20 minutes of exercise to an already

intensive schedule of physical activities may not be sufficient to produce clinically significant changes, particularly in light of the subject variability and natural recovery. In addition, much of today's physical therapy regimens include functional and dynamic movements against gravity (e.g., repetitive rise from a chair) which can also influence muscle strength.

In persons with chronic stroke, only three studies[61–63] have measured the effects of muscle strengthening when compared to a control group. Unfortunately, one of these studies[62] had major methodological flaws (Table 2). A control group is important because the activity involved in commuting to a centre three times a week (e.g., getting in and out of the car or bus, walking from the parking lot to the gym) is already an exercise stimulus. In addition, attention from a clinician, increased focus on health or simply the familiarization of testing procedures could lead to improvements in the outcome measures. In a small RCT, Kim et al.[61] found that a 6-week isokinetic strengthening program for the ankle, knee and hip resulted in a trend ($p < 0.06$) towards improvement of the strength training group for muscle torque, but walking speed increased for both the strengthening and passive exercise groups. Kim et al.[61] suggested that strength training should be complemented by functional training to take advantage of any gain in strength and to transfer gains of strength to functional tasks. Bourbonnais et al.[63] reported that a LE strength program, providing feedback on the force generated, resulted in 55% improvement in strength and 25% improvement in gait speed compared to an UE strength program (control condition). Positive effects of strength training in persons with chronic stroke have also been demonstrated by studies[4,64,65] without control groups, but results must be interpreted with caution given the lack of control comparisons.

Only three studies have applied graded muscle strengthening for the UE. In persons with mild impairments following stroke, Bütefisch and colleagues[66] used an isotonic wrist/hand strengthening program in conjunction with an inpatient rehabilitation program. Improvements were noted in muscle torque, hand kinematics, and UE function. In contrast, Trombly et al.[67] found no changes in finger or hand function with a similar population of patients. Although their protocol maximally recruited the muscles,[68] subjects performed only one set of 10 repetitions and the sample size was small (5 subjects per group). Using an UE isometric strengthening protocol in persons with chronic stroke, Bourbonnais et al.[63] found a 35% improvement in generating isometric forces but no improvements in dexterity or UE coordination.

The majority of studies that evaluated muscle strength as an outcome demonstrated improvements (with Levels of evidence from II to V).[4,58,60–66] Consequently, a fair recommendation can be given to the effect of strength training on increasing muscle strength in persons with stroke. There is poor or insufficient evidence for the effect of strength training on functional outcomes in persons with stroke. Positive outcomes resulted from less rigorous pre-test/post-test studies with conflicting results in the controlled trials. In fact, the two studies[57, 61] that had the highest PEDro scores (7 out of 10) did not show significant differences between the strength training and control group for functional outcomes. In addition, the small sample sizes (and therefore low power) used in many of these studies may contribute to the non-significant findings for the controlled trials. The reduced rigor of

the pre-test/post-test interventions lessens the certainty that the improvements resulted from the intervention.

The review of the evidence should consider not only the quality of the evidence (e.g., research design, sample size), which is captured by the level of evidence assigned to each study, but also elements such as the effect size and confidence intervals.[54] Morris et al.[69] reported large effect sizes analyzed from five studies for the use of progressive muscle strengthening in improving muscle strength[4,64–66] and activity limitations.[4,63–65] One additional paper (a thesis) was included in their analyses but was excluded in this review.

CENTRAL AND PERIPHERAL ADAPTATIONS OF STRENGTH TRAINING

The fact that strength training can result in improvements in the ability to generate force in individuals with stroke is not surprising, given the impairments in central regulation of muscle force in persons with stroke and the known neural adaptations that can occur with exercise. In healthy young male adults, Akima et al.[70] found a greater percentage of the quadriceps femoris cross-sectional area was activated following 2 weeks of isokinetic training, suggesting an increase in motor unit recruitment. Motor units are also capable of increasing their discharge rate with strength training. The exact timing of this increase and effects of the person's age are not known. However, Patten et al.[71] reported an immediate increase in motor unit discharge rate on commencing a 6-week isometric resistance program, which remained elevated for young adults, but was not maintained for older adults over the 6 weeks. Studies which characterize the motor unit responses to strength training in individuals with stroke are needed to determine whether similar neural changes occur. Repetitive muscle activation could also cause cortical reorganization, as seen with forced use of the paretic UE through constraint-induced therapy.[72] In animal models, exercise has been shown to activate molecular and cellular mechanisms, i.e., an increase in brain-derived neurotrophic factor and nerve growth factor expression that are likely mediators of activity-dependent changes in the central nervous system.[73,74]

Strength training can potentially reduce muscle atrophy, although the literature is not clear as to the importance of these peripheral changes to muscle strength in persons with stroke.[31,32] In addition, strength training has the potential to alter the passive viscoelastic properties of muscle and tendon,[75,76] which could influence the hypertonia observed in stroke.

STRENGTH TRAINING VERSUS FUNCTIONAL TRAINING

Although more controlled trials are needed, the preliminary evidence from this systematic review suggests that the strength protocols in these studies may not be sufficient to transfer the strength gains to functional tasks without complementary task-specific practice. However, many of these studies did not match the strength training protocol to the requirements of the functional tasks (e.g., ranges of motion, speeds of contraction, magnitude and type of contraction) which would enhance the specificity of the training. In addition, other outcomes not yet evaluated, such as bone density, may benefit from strength training alone. Individuals with stroke are at increased risk of developing osteoporosis on the paretic side[77] and, coupled with poor balance arising from motor, sensory, visual and

perceptual deficits, they have a seven-fold increase in fracture risk within the first year after hospitalization for stroke.[78]

Clinical trials with older adults have demonstrated that resistance training alone can improve muscle strength, as well as mobility (e.g., sit-to-stand, gait and stair climbing).[79,80] However, the altered motor coordination following stroke likely requires task-specific practice to make use of any gains in strength. A number of task-specific training interventions that include components of graded muscle strength or muscle strengthening during functional tasks (e.g., treadmill training, repetitive sit-to-stand, circuit training[81–84]) have been shown to be effective in improving functional performance. In addition, a few task-based programs have demonstrated improvements in muscle strength. For example, LE muscle strength in persons with chronic stroke was improved with a treadmill training program[85] and with a shallow water walking/running program.[86] Clinical trials which compare strength training programs, task-based programs and combined strengthening/task-based programs could quantify the contributions and interactions of the strength components and task-specific components.

PRECAUTIONS AND SAFETY

A review of the evidence should consider the beneficial effects, in light of any possible adverse effects, reported for any of the studies (from RCTs to case reports).[54] In the majority of the reviewed studies, it appeared that the protocol was tolerated well and minimal drop-outs occurred. Moreland et al.[57] did report more adverse occurrences (e.g., pain, stiffness) in their sub-acute patients who performed resistive exercises (compared to conventional therapy); however, these differences were not statistically significant. They also undertook a post-hoc subgroup analysis of patients with severe stroke (less than stage 4 Chedoke-McMaster leg score), and found that these patients performed consistently worse on the outcome measures compared to the conventional therapy group; however, these differences were not statistically significant. Although they suggested that patients with severe stroke may be detrimentally affected by a strengthening program, none of the other studies undertaken with a sub-acute population[58–60] found worse results in the strength training group. In fact, Inaba et al.[58] only assessed non-ambulatory individuals with stroke (indicative of severe stroke) and found improvements in the resistive group compared to those subjects undergoing conventional treatments. Weiss et al.[65] reported that two subjects experienced minor back discomfort during strength training, and one subject experienced some discomfort with her non-paretic knee.

With the high incidence of hypertension (75%) and cardiac disease (70%) in individuals with stroke,[87,88] factors that affect the hemodynamic responses during strength training and how they can be influenced by exercise prescription must be considered. Extreme blood pressure (BP) responses have been reported with high intensity strengthening exercise. For example, a 30-second isometric double-leg press at 87.5% of maximum voluntary contraction can cause BP values up to 282 mm Hg systolic and 181 mm Hg diastolic in healthy individuals.[89]

What intensity is required to obtain muscle strengthening effects for individuals with stroke? Some studies on strength training in individuals with stroke have used high intensity protocols (maximal effort).[4,61] MacDougall et al.[89] reported that a Valsalva manuever (which would further raise BP) was not avoidable if the force was greater than 80% of maximum voluntary contraction for healthy individuals using a leg press task. Badics et al.[60] found improvements of at least 30% for LE and UE strength using a low intensity strengthening protocol (30–50% MVC) in persons with stroke. However, there was no control group in this study. Future controlled studies should determine whether submaximal protocols are effective in increasing muscle strength in individuals with stroke, as this would likely increase the pool of candidates who could safely participate in these programs. In addition, it is possible that the intensity needed for training effects for individuals with stroke need not be as high as in healthy individuals due to the lower baseline levels; for example, older adults appear to benefit from muscle strengthening programs that differ widely in intensity and frequency.[90]

Several modifications and adaptations can be incorporated to reduce adverse events. A gentle warm-up and cool-down has been found to reduce extreme changes in BP in healthy individuals.[91,92] An upright body posture may minimize increases in BP during dynamic LE exercises compared to a supine body posture.[93] Discouraging breath holding and ensuring rhythmical breathing coordinated with lower intensities of resistance may help to avoid the Valsalva manoeuvre.

The type of muscle contraction will also influence the hemodynamic responses. For example, at 70% or 90% of maximum voluntary contraction, mean BP is greatest and absolute force lowest, for isometric contractions compared to concentric or eccentric contractions.[89] Furthermore, eccentric exercise may induce less cardiovascular stress, as measured by smaller heart rate and BP increases, compared to concentric exercise at matched force output.[89,94]

Bed rest, inactivity, disuse atrophy, and muscle weakness are commonly found in individuals with stroke and may increase their vulnerability for skeletal muscle injury during strenuous or unaccustomed exercise.[95] Heavy-resistance strength training (55 total repetitions of 5 repetitions maximum of unilateral knee-extension, three times per week for 9 weeks), has been reported to cause higher levels of muscle damage (assessed via electron microscopy) in older adults than in young individuals.[96] Muscle injury is more likely to occur during eccentric exercise[97] and particularly after inactivity. Ploutz-Snyder et al.[98] reported quadriceps muscle injury after the introduction of moderate eccentric exercises (65% of maximal contraction) after 5 weeks in the nonweight-bearing limb, but not in the weight-bearing limb in young adults.

Although eccentric exercise has greater potential for muscle injury, it may result in less cardiovascular stress.[94] To reduce the risk of muscle injury, a gradual increment in intensity, variety in exercises, rest between exercises and monitoring of muscle soreness, should be implemented.[95]

Safety guidelines based on physiological responses of healthy individuals are not sufficient for exercise prescription for individuals with stroke. Clinical trials on muscle strengthening need to be complemented by studies which examine the physiological responses of exercise in persons with stroke.

CONCLUSIONS

Although there is evidence that strength training alone can improve muscle strength, further evidence is needed to determine the carry-over effects of strength training to functional tasks in people with stroke. Many research questions remain to be answered to optimize strength training in persons with stroke, particularly in identifying the types of muscle contraction (e.g., eccentric versus concentric), optimal training intensities, complementary role of other rehabilitation interventions (e.g., functional electrical stimulation, treadmill training) and effects on bone density.

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Table 1

Levels of evidence (adapted from Sackett[52])

Levels of evidence	
Level I	Large randomized controlled trial (RCT), low error risk
Level II	Small randomized trial, moderate to high error risk
Level III	Nonrandomized concurrent cohort comparisons between subjects who did and did not receive the intervention
Level IV	Nonrandomized, historical cohort comparisons between current subjects who received the intervention and former subjects who did not receive the intervention
Level V	Case series, no control

Table 2

Clinical trials investigating graded muscle strengthening programs in individuals with stroke

Authors, Study Design, and Evidence Level	Subjects	Intervention	Results
LOWER EXREMITY			
INPATIENT REHAB			
Moreland et al,[57] 2003 RCT Level I	133 subjects (multi-centre), < 6 months post-stroke	Inpatient conventional physical therapy versus conventional + resistive exercises with weights, 30 minutes, 3X/week.	No difference in 2 minute walk test or Disability Inventory
Inaba et al,[58] 1973 RCT Level II (high error risk, large number of drop-outs)	77 subjects, < 4 months post-stroke, non-ambulatory	1 to 2 months, daily 1. functional training and stretching (n=26) (control) 2. control activities plus active exercise (n=23) 3. control activities plus resistive exercise (n=28)	Greater improvement of 10 Repetition Maximum strength and activities of daily living for group 3 (resistive group) after 1 month. No group differences after 2 months training, but it does not appear that all subjects underwent 2 months training.
Glasser,[59] 1986 Small RCT Level II	20 subjects, 3–6 months post-stroke	5 week inpatient physical therapy (5X week, 2 one hour sessions/day) (n=10) versus physical therapy plus kinetron isokinetic LE exercise (n=10) (resisted reciprocal hip/knee flexion during semi-sitting posture progressed from 10–30 minutes over the 5 weeks).	No differences in functional ambulation profile (includes temporal and distance gait variables)
Badics et al,[60] 2002 Pre-test/post-test Level V	56 subjects, 3 weeks to 10 years post-stroke	Residential rehabilitation which included leg extensor presses, arm presses (triceps) at 30–50% MVC, 3–5 sets of 20 repetitions. Other activities not documented. 20 subjects did not do the arm program due to severe arm conditions.	31%↑ in LE strength and 37%↑ in UE strength.
OUTPATIENT OR COMMUNITY			
Kim et al,[61] 2001 Small RCT Level II	20 ambulatory subjects, > 1 year post-stroke	6 week passive exercise (n=10) versus isokinetic paretic strengthening (hip, knee, ankle) (n=10).	Trend, $p < 0.06$ for ↑ isokinetic torque (of strength group over control No group differences in gait or stair speed.
Carr and Jones,[62] 2003 RCT Level II (high error risk, e.g., 200% baseline group differences for some strength measures and no co-variates considered. Drop-outs occurred, but their numbers not reported)	40 subjects, > 6 months post-stroke	16 week aerobic (recumbent bike with arm ergometer) versus aerobic + arm/leg graded strength training (free weights and isokinetic machines), 3X week.	Both groups ↑ knee flexion torque and shoulder extension, but not knee extension. Only strength +aerobic group↑ shoulder flexion.
Bourbonnais et al,[63] 2002 Small RCT Pre-test/post-test/8 week retention Level II	25 subjects with chronic stroke	6 week, 3X week, visual feedback of multi-joint, multi-directional isometric force generation (coordination exercises). UE (n=13): varying combinations of shoulder, elbow, and grip forces (progress from 20–60% MVC). LE (n=12): varying combinations of hip, knee and ankle forces (40–90% MVC).	UE group had 35% ↑ in UE isometric force. No group difference for UE Fugl-Meyer, dexterity, finger-to-nose test. LE group had 55% ↑ in LE isometric force. LE group had 25% greater improvement in gait speed over UE. No change in Timed up and Go Test for either group.
Engardt et al,[64] 1995 Stratified non-random allocation Level III	20 ambulatory subjects with chronic stroke	6 week, 2X week, isokinetic eccentric (n=10) or concentric (n=10) paretic knee extensor strengthening.	Both groups ↑ knee eccentric and concentric torque but no group differences. Eccentric has ↑ in eccentric and concentric torque relative to the non-paretic leg, but concentric did not.

Authors, Study Design, and Evidence Level	Subjects	Intervention	Results
Note: no actual control for effect of strength and could be considered Level V for this variable			↑ symmetrical forces during rise up from chair with eccentric training only. No group differences for gait speed. ↑ Antagonist EMG activity with concentric but not eccentric training.
Weiss et al,[65] 2000 Pre-test/post-test Level V	7 subjects with chronic stroke	12 week, 2X/week resistance training for both LEs (leg press, knee extension, hip motions) at 70% 1 Repetition Maximum.	68% ↑ on paretic and 48% ↑ on non-paretic for five leg muscle groups. 21%↓ on rise from chair time. No change gait speed. ↑ Motor Assessment Scale (lower limb score). ↑ Berg balance score by 5 points.
Sharp and Brouwer,[4] 1997 Pre-test, post-test, 1 month retention Level V	15 ambulatory subjects, > 6 months post-stroke	6 week, 3X/week, 40 minutes/day isokinetic paretic knee strengthening.	15–20% ↑ paretic knee extension and 37–39%↑ flexor torque post-intervention. Only 1 of 6 muscle tests (30 degrees/second, quads) were significant at retention. 5.3% ↑ gait velocity post-test and 6.8% ↑ at retention. No change Timed up and Go or stair ability ↑ level of physical activity (Human Activity Profile).
UPPER EXTREMITY			
Bütefisch et al,[66] 1995 Multiple baseline RCT Level II	27 subjects 3–19 weeks post-stroke with minimal to mild UE deficits 1–4 week intervention	Group 1: Strength group, 2X day, 15 min each session, in addition to standard inpatient therapy (n=12) (grip exercises, isotonic wrist extension against weights, resisted finger extension. Group 2: 2 weeks TENS group (n=15), followed by strength program	↑ grip strength, isometric hand extension force, acceleration of hand extension and Rivermead Motor Assessment (arm section) for both groups following strength training period.
Trombly et al,[67] 1986 Level II	20 subjects, average 6 weeks post-stroke and could grasp a 2.5 cm cylinder	All received typical rehabilitation (including occupational therapy) plus the following treatments (one set of 10 repetitions) daily for a max of 20 sessions: Group 1: Control (n=5) Group 2: Resisted finger extensions (n=5) Group 3: Ballistic finger extensions (n=5) Group 4: Resisted grasp (n=5)	No group differences in finger or hand function. Note: underpowered sample with baseline group differences in hand function.
Bourbonnais et al,[63] 2002 (see above in LE section)			

Table 3

PEDro Scale: Criteria Assessing Quality of Study

Criterion	1	2	3	4	5	6	7	8	9	10	11	
Scale Item	**Eligibility Criteria Specified**	**Random Allocation**	**Concealed Allocation**	**Groups Similar at Baseline**	**Subject Blinding**	**Therapist Blinding**	**Assessor Blinding**	**Adequate Follow-up**	**Intent-To-Treat Analysis**	**Between-Group Statistics**	**Point Estimates and Variability Data**	**Total Score**
Moreland et al,[57] 2003	Yes	1	1	0	0	0	1	1	1	1	1	7
Kim et al,[61] 2001	Yes	1	0	1	1	0	1	1	0	1	1	7
Engardt et al,[64] 1995	Yes	1	0	1	0	0	0	1	0	1	1	5*
Bourbonnais et al,[63] 2002	Yes	1	0	1	0	0	0	1	0	1	1	5
Trombly et al,[67] 1986	Yes	1	0	0	0	0	0	1	0	1	1	4*
Bütefisch et al,[66] 1995	No	1	0	1	0	0	0	1	0	0	1	4*
Glasser,[59] 1986	No	1	0	1	0	0	0	0	0	1	1	4*
Inaba et al,[58] 1973	Yes	1	1	1	0	0	0	0	0	1	0	4*
Carr and Jones,[62] 2003	No	1	0	0	0	0	0	0	0	0	1	2

*Studies in which the PEDro scores were taken from the Physiotherapy Evidence Database