Bangladesh Journal of Neuroscience 2013; Vol. 29 (2) : 124-129

REVIEW ARTICLE

Role of Strength Training on Poststroke Hemiparesis, a Review on Recent Developments.

MD SHAHIDUR RAHMAN[1], AKM SALEK[2]

Abstract:

Stroke is the leading cause of activity limitation and participation restriction all over the world and weakness presents a more serious compromise to movement and function in poststroke hemiplegia than spasticity. In this article we have gone through publications on functional consequences of post-stroke paresis and effect of resistance training in effective stroke rehabilitation and reviewed the clinical and functional phenomena of paresis in stroke survivors, effects of high-resistance training on improvement of strength and functional capacity. By examining the various facets of weakness, we can understand the specific nature of motor impairment and can identify potential strategies to mitigate its effects, functional improvement and participation in activities. The specific aims were to emphasis the importance of strength training in the functional improvement of patients suffering from stroke with paralysis.

Key words: *stroke, strength training, functional improvement*

Introduction:

Stroke is a leading cause of long-term disability in the Western world, with a prevalence of approximately 900 per 100,000 persons. Because of remarkable development in the acute management of stroke, the majority of patients now survives and recovers, experiencing only a modest decrease in life expectancy [1] . In Bangladesh we do not know the actual prevalence and impact of stroke but it may approximate or exceed the statistics of the western world.

During the last decade, the number of stroke survivors has increased 30 percent worldwide. As a result considerable number of stroke survivors present with partial to complete loss of function and social burden. Though the incidence of stroke doubles with each decade beyond 60 years of age, the incidence of stroke has increased dramatically in younger individuals in the recent years [2]. This changing landscape for persons surviving stroke underscores the critical importance of providing effective rehabilitation with the potential to optimize recovery of function, minimize long-term disability, and enable functional independence so that quality of life and employment is ensured. In a poor country like Bangladesh the socioeconomic burden of disabled stroke survivors is much higher. We need to bring these young disabled into mainstream of development.

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

Traditional perspectives: Spasticity presented the most significant limitation to recovery of normal motor function. Physical exertion was clinically observed to exacerbate spasticity. Therapeutic activities using forceful contractions became restricted for persons with nervous system injury. One prominent approach to treatment of adult

hemiplegia thus centered on the concept of managing muscle hypertonia. The general goal of neurorehabilitation treatment is to focus on improving control and especially the quality of movement. Meta-analyses that examined the effects of commonly used interventions for rehabilitation of both the upper and lower limb in poststroke hemiplegia reported a lack of compelling evidence that any of the existing approaches to neurorehabilitation have demonstrated superior efficacy for promoting recovery of motor function [4, 5].

Resistance training: Currently emerging evidence suggests that paresis may be directly responsible for compromised motor function [6-8]. Positive effects of resistance exercise have been demonstrated in persons with poststroke hemiplegia, and in some cases, concomitant influences on performance of functional tasks have been observed. High-intensity activities, including resistance training, could form an important component of rehabilitation programs for persons with poststroke hemiplegia.

Muscle weakness is a common impairment after stroke. However, facilitation treatment models have often emphasized the management of spasticity without addressing underlying muscle weakness. Another common intervention focus is functional training, sometimes without addressing the contributing impairments. Lower-extremity muscle strength has been correlated with gait speed in stroke patients[9]. Additionally, lower-extremity muscle strength on admission to rehabilitation is a predictor of function at discharge[10]. Lower-extremity strength has also been inversely correlated with a risk of falling in elderly individuals. The recommendation for including strengthening in the acute rehabilitation of patients with muscle weakness after stroke is based on Working Group Consensus, considering the positive relationship between muscle strength, function and prevention of falls. Researchers in strength training of post-stroke patients have studied subjects after acute rehabilitation had been completed (greater than 6 months after stroke) and demonstrated improvement in muscle strength and function with training[11, 12]. There is a lack of research on specific strength training during acute rehabilitation[13].

Role of agonists and antagonists in functional improvement

Weakness following stroke is referred to as either hemiparesis (mild to moderate degree of weakness) or hemiplegia (severe or complete loss of motor function) on one side of the body. However, evidence is now emerging that weakness also occurs on the ipsilesional side (traditionally termed the "nonparetic"), within a short time frame post-acute stroke[14]. In the literature, poststroke weakness has been described not only as impaired force magnitude[15], but also as a more broadly defined phenomenon, including slowness to produce force[16, 17], a rapid onset of fatigue[18], an excessive sense of effort [19], and difficulty with producing force effectively within the context of a task[20]. Throughout this review, we use the term "poststroke weakness" to include all aspects of weakness following stroke.

Co-contraction of antagonist muscles has also been found to interfere with force magnitude, rate of force production and intersegment coordination by acting as an "antagonist restraint" [21] . Significant impairment of agonist activation has been demonstrated in the paretic limb[7, 22]. Such observations lead predictably to questions of whether and how agonist activation can be improved and whether such improvement in physiologic function leads to clinically and functionally important differences in motor performance.

Post-stroke paresis and functional impairment

Post stroke hemiplegia is associated with significant impairments of motor function that are believed to compromise activity of daily living (ADL) performances and lead to loss of independence. However, a direct causal relationship between strength or weakness and motor function has not been established. Traditionally, a strong bias has existed against quantifying strength in hemiplegic persons. As a result, the majority of clinical research in this population has focused on outcome measures at the activity and participation levels[23]. Bohannon and Andrews observed that gait performance in 17 hemiparetic persons was significantly correlated with knee extensor torque but not with spasticity and knee extension muscle performance measured either isometrically or isokinetically correlated significantly with gait

velocity[24]. Nakamura and coworkers also observed that spasticity was unrelated to locomotors impairments[25, 26]. Isokinetic knee extension strength in the paretic limb was strongly associated with self-selected walking speed (SSWS). Lindmark and Hamrin observed a moderate relationship between SSWS and either motor scores or knee extension torque, which improved in predictive power when examined in a multivariate statistical model [27]. Pohl and coworkers observed that the combination of peak isometric knee extension force and rate of force acquisition explained a significant 12 percent of variance related to gait speed in hemi paretic adults[28]. When plantar flexion strength was added to the model, its explanatory power increased such that it became possible to predict maximal gait speed[29]. Each of the investigations just described focused on isolated muscle groups or actions. However, functional movement involves simultaneous activation and coordination of multiple muscles. This disparity may contribute in part to failure to demonstrate a direct relationship between strength and function[30].

Strengthening Exercise Induces Neurological Recovery.

Because poststroke weakness involves both neural and muscular changes, it seems appealing to suggest an analogy with other physiological conditions, such as aging, for which very clear benefits of strength training have been demonstrated[31]. Currently, available evidence regarding strengthening in hemiplegia indicates that significant strength gains are attainable in persons with poststroke hemiparesis at acute, subacute, and chronic stages of recovery [32, 33 34]. However, the physiological mechanisms responsible for these therapeutically induced improvements have not been demonstrated. Strengthening exercises may influence neural drive at either the supraspinal or spinal level.

Power reaching or skilled reaching training

Recent efforts for stroke rehabilitation have been directed toward functional and task-specific therapies that focus primarily on ADL and on grossly related precursor activities [35, 36]. A common element to these more recent approaches is substantially increased therapeutic intensity relative to traditional approaches. Increased intensity is defined by a substantially increased volume of therapeutic participation[37] an increased amount of direct participation in therapeutic activities, or performance of activities at a higher level of the subject's functional capacity[38]. Exercise dose in a stroke unit is variable and can be predicted by age and disability. Increased exercise dose is associated with improved mobility outcomes[39].

Current controversy thus centers on whether the critical variable for therapeutic efficacy is the task specificity or the intensity of effort involved in therapeutic activities. Thus, while skill-based, task-specific interventions clearly promote important use-dependent cortical reorganization, resistance training apparently can promote additional, beneficial plasticity elsewhere in the neuraxis. In all likelihood, the most effective therapeutic intervention involves a combination of elements.

The Ultimate Goal of Rehabilitation

The ultimate goal of rehabilitation following stroke is to promote improvements in function, activities, and participation. Collective efforts are thus required to design effective and efficient rehabilitation interventions. Weakness is not the only impairment in poststroke hemiplegia but weakness plays a significant contributory role to motor disability. By examining the various facets of weakness such as low force production, fatigability, excessive sense of effort, ineffective task-dependent force production etc. we can understand the specific nature of motor impairment and can identify potential strategies to mitigate its effects, functional improvement and participation in activities.

Future research:

Important area for future research is developing a greater understanding of the mechanisms underlying poststroke weakness. Without this information, we are restricted in our efforts to design appropriate rehabilitation interventions to counteract compromised function associated with poststroke weakness. Recent research evidence indicates that "task-specific" therapy[40-43] produces superior outcomes as compared to traditional therapeutic approaches[44-47]. In elders, strength training has

been demonstrated to decrease depression and improve sleep patterns, influence bone mass, decrease insulin resistance (Type II diabetes), and normalize blood pressure[48]. However, there is also evidence that increased intensity of therapy leads to more significant functional outcome. There is a need to establish the effectiveness of strength training in relation to task-specific therapies because it may be the case that strength training is an efficient means for delivering high-intensity therapy. To define and implement suitable protocols of strength training into stroke rehabilitation programs, future research should explore the specific factors such as the types of exercise, the frequency, intensity and time spent in strength training, and the number of specific exercises. Moreover, the long-term effects, both long-term training and retention of training, need to be understood. Finally, once gains in strength have been achieved we need to understand how they translate to functional gains and how they are best maintained.

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

Conclusion:

While the number of studies is limited, emerging evidence suggests that persons with poststroke paresis can improve strength through resistance exercise in the absence of negative side effects, including exacerbation of hypertonia. Moreover, these improvements in strength appear to transfer to functional improvements. Despite increases in strength, improvements in functional performance may not occur in hemiplegic persons with low strength and low performance. It is entirely possible that vigorous strength training promotes positive effects on other aspects of physiologic function in this type of at-risk population. Still, many unresolved issues remain. The potential for strength training to improve the overall outcomes of rehabilitation for persons with poststroke hemiplegia warrants further investigation.

References:

1. Lopez-Yunez AM. The management of stroke patients by neurologists: common questions and new observations. Semin Neurol. 2002; 22(1): 53-61.
2. Zorowitz RD, Gross E, Polinski DM. The stroke survivor. Disabil Rehabil. 2002; 24(13): 666-79.
3. Katz RT, Rovai GP, Brait C, Rymer WZ. Objective quantification of spastic hypertonia: Correlation with clinical findings. Arch Phys Med Rehabil. 1992; 73:339-47.
4. Wagenaar RC, Meijer OG. Effects of stroke rehabilitation (1). J Rehabil Sci. 1991; 4(3): 61-73.
5. Wagenaar RC, Meijer OG. Effects of stroke rehabilitation (2). J Rehabil Sci. 1991; 4(4): 97-109.
6. Bohannon RW. Selected determinants of ambulatory capacity in patients with hemiplegia. Clin Rehabil. 1989; 3: 47-53.
7. Canning CG, Ada L, O'Dwyer NJ. Abnormal muscle activation characteristics associated with loss of dexterity after stroke. J Neurol Sci. 2000; 176:45-56.
8. Nadeau S, Arsenault AB, Gravel D, Bourbonnais D. Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. Am J Phys Med Rehabil. 1999; 78(2):123-30.
9. Bohannon RW, Walsh S. Nature, reliability, and predictive value of muscle performance measures in patients with hemiparesis following stroke. *Arch Phys Med Rehabil. 1992*; *73*: *721*–25.
10. Andrews AW, Bohannon RW. Discharge function and length of stay for patients with

stroke are predicted by lower extremity muscle force on admission to rehabilitation. *Neurorehabil Neural Repair. 2001*; *15*: *93*–97.

11. Rimmer JH, Riley B, Creviston T, Nicola T. Exercise training in a predominantly African-American group of stroke survivors. *Med Sci Sports Exerc. 2000*; *32*: *1990*–96.
12. Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Muscle strengthening and physical conditioning to reduce impairment and disability in chronic stroke survivors. *Arch Phys Med Rehabil. 1999*; *80*: *1211*–8.
13. Duncan P, Zorowitz R, Bates B, Choi JY, Glasberg JJ, Glenn D et al Management of Adult Stroke Rehabilitation Care. A Clinical Practice Guideline. Stroke*. 2005; 36: e100-e143*,
14. Harris ML, Polkey MI, Bath PM, Moxham J. Quadriceps muscle weakness following acute hemiplegic stroke. Clin Rehabil. 2001; 15(3): 274-81.
15. Adams RW, Gandevia SC, Skuse NF. The distribution of muscle weakness in upper motor neuron lesions affecting the lower limb. Brain. 1990; 113: 1459-76.
16. Canning CG, Ada L, O'Dwyer NJ. Slowness to develop force contributes to weakness after stroke. Arch Phys Med Rehabil. 1999; 80: 66-70.
17. Bohannon RW. Walking after stroke: comfortable versus maximum safe speed. Int J Rehabil Res. 1992;15(3): 246-48.
18. Ingles JL, Eskes GA, Phillips SJ. Fatigue after stroke. Arch Phys Med Rehabil. 1999; 80: 173-78.
19. Gandevia SC. The perception of motor commands or effort during muscular paralysis. Brain. 1982;105: 151-9.
20. Beer RF, Given JD, Dewald JP. Task-dependent weakness at the elbow in patients with hemiparesis. Arch Phys Med Rehabil. 1999; 80(7): 766-72.
21. Gowland C, DeBruin H, Basmajian JV, Plews N, Burcea I. Agonist and antagonist activity during voluntary upper-limb movement in patients with stroke. Phys Ther. 1992; 72(9): 624-33.
22. Davies JM, Mayston MJ, Newham DJ. Electrical and mechanical output of the knee muscles during isometric and isokinetic activity in stroke and healthy adults. Disabil Rehabil. 1996; 18(2): 83-90.
23. WHO. International classification of functioning, disability and health. Geneva: World Health Organization; 2002.
24. Bohannon RW, Andrews AW. Correlation of knee extensor muscle torque and spasticity with gait speed in patients with stroke. Arch Phys Med Rehabil. 1990; 71(5): 330-3.
25. Nakamura R, Hosokawa T, Tsuji I. Relationship of muscle strength for knee extension to walking capacity in patients with spastic hemiparesis. Tohoku J Exp Med. 1985; 145: 335-40.
26. Nakamura R, Watanabe S, Handa T, Morohashi I. The relationship between walking speed and muscle strength for knee extension in hemiparetic stroke patients: a follow-up study. Tohoku J Exp Med. 1988;154: 111-3.
27. Lindmark B, Hamrin E. Relation between gait speed, knee muscle torque and motor scores in post-stroke patients. Scand J Caring Sci. 1995; 9:195-202.
28. Pohl PS, Duncan P, Perera S, Long J, Liu W, Zhou J, et al. Rate of isometric knee extension strength development and walking speed after stroke. J Rehabil Res Dev. 2002; 39(6): 651-58.
29. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clin Biomech (Bristol, Avon). 1999; 14(2):125-35.
30. Conelly DM, Vandervoort AA. Improvements in knee extensor strength of institutionalized elderly women after exercise with ankle weights. Physiother Can. 1995; 47(1): 15-23.

31. Latham N, Anderson C, Bennett D, Stretton C. Progressive resistance strength training for physical disability in older people. Cochrane Database Syst Rev. 2003(2): CD002759.

32. Winstein CJ, Rose DK, Tan SM, Lewthwaite R, Chui HC, Azen SP. A randomized controlled comparison of upper-extremity rehabilitation strategies in acute stroke: A pilot study of immediate and long-term outcomes. Arch Phys Med Rehabil. 2004; 85:620-28.

33. Inaba M, Edberg E, Montgomery J, Gillis MK. Effectiveness of functional training, active exercise, and resistive exercise for patients with hemiplegia. Phys Ther. 1973; 53(1): 28-35.

34. Kim CM, Eng JJ, Macintyre DL, Dawson AS. Effects of isokinetic strength training on walking in persons with stroke: a double-blind controlled pilot study. J Stroke Cerbrovasc Dis. 2001; 10(6): 265-73.

35. Dean CM, Shepherd RB. Task-related training improves performance of seated reaching tasks after stroke. Stroke. 1997; 28(4):722-28.

36. Malouin F, Potvin M, Prevost J, Richards CL, Wood-Dauphinee S. Use of an intensive task-oriented gait training program in a series of patients with acute cerebrovascular accidents. Phys Ther. 1992; 72(11): 781-93.

37. Taub E. Constraint-induced movement therapy and massed practice. Stroke 2000; 31(4): 986-8.

38. Kwakkel G, Wagenaar RC, Koelman TW, Lankhorst GJ, Koetsier JC. Effects of intensity of rehabilitation after stroke. Stroke. 1997; 28:1550-6.

39. Katharine Scrivener, Catherine Sherrington, and Karl Schurr, Exercise dose and outcome in a comprehensive stroke unit : description and prediction from a prospective cohort study J Rehabil Med 2012; 44: 824–9

40. Kwakkel G, Wagenaar RC, Twisk JWR, Lankhorst GJ, Koetsier JC. Intensity of leg and arm training after primary middle-cerebral artery stroke: a randomised trial. The Lancet. 1999; 354: 191-6.

41. Duncan P, Richards L, Wallace D, Stoker-Yates J, Pohl P, Luchies C, et al. A randomized, controlled pilot study of a home-based exercise program for individuals with mild and moderate stroke. Stroke. 1998; 29: 2055-60.

42. Smith GV, Silver KH, Goldberg AP, Macko RF. "Task-oriented" exercise improves hamstring strength and spastic reflexes in chronic stroke patients. Stroke. 1999; 30(10): 2112-8.

43. Richards CL, Malouin F, Wood-Dauphinee S, Williams JI, Bouchard J-P, Brunet D. Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Arch Phys Med Rehabil. 1993; 74: 612-20.

44. Dean CM, Richards CL, Malouin F. Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. Arch Phys Med Rehabil. 2000;81(4): 409-17.

45. Silver KH, Macko RF, Forrester LW, Goldberg AP, Smith GV. Effects of aerobic treadmill training on gait velocity, cadence, and gait symmetry in chronic hemiparetic stroke: a preliminary report. Neurorehabil Neural Repair. 2000; 14(1): 65-71.

46. Miltner WH, Bauder H, Sommer M, Dettmers C, Taub E. Effects of constraint-induced movement therapy on patients with chronic motor deficits after stroke: a replication. Stroke. 1999; 30(3): 586-92.

47. Taub E, Miller NE, Novack TA, Cook EW, Fleming WC, Nepomuceno CS, et al. Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehabil. 1993; 74(4):347-54.

48. Hurley BF, Roth SM. Strength training in the elderly: effects on risk factors for age-related diseases. Sports Med. 2000;30(4): 249-68.