RESEARCH ARTICLE

EFFECT OF LOWER LIMB PASSIVE CYCLING MOVEMENT ON SPASTICITY IN PERSONS WITH STROKE

Jyoti Ranjan Sahoo, Mrs. Monalisa Pattnaik and Dr. Patitapaban Mohanty
Department of Physiotherapy, SVNIRTAR, Olatpur, P O Bairoi, Cuttack, Orissa 754010, India.

Abstract:

**Introduction:** One of the contributors to the post-stroke functional impairment is spasticity. It was estimated that 2/3rd of patients would develop post-stroke spasticity. The prevalence of spasticity was found to be 40% one year after stroke which leads to functional impairment in activity of daily living. However no study evaluated the effect of lower limb passive cycling movement on spasticity in persons with stroke. **Objectives:** The objective of this study is to evaluate how post stroke spasticity behaves with lower limb passive cycling movement. **Methodology:** This was a randomized control trial. Thirty subjects with stroke (n=30) were randomly divided into two groups, Group A (Experimental group: Passive cycling and conventional physical therapy exercises; n=15) and Group B (Control Group: Conventional physical therapy exercise; n=15). Pre test measurement of all dependent variables – spasticity by MMAS (modified modified Ashworth scale), ROM by goniometer and 10 meter walk test were made on recruitment for the study and post test measurement was made after completion of therapy for 4 weeks, (5 days a week). **Data analysis:** The Dependent variables for spasticity were analysed using Man Whitney U test. Straight leg raising, dorsiflexion range of motion and 10 metre walk test were analysed using a 2x2 ANOVA. All pair wise post – hoc comparisons were analyzed using a 0.05 level of significance. **Results:** The reduction in spasticity from pre to post was significantly more in experimental group. The study shows that there is statistically significant improvement in dorsiflexion range of motion, SLR as well as improvement in walking function in experimental group but not in control group. **Conclusion:** Passive cycling was more effective in reducing spasticity of lower limb, improve the range of motion and walking function than those in subjects with conventional exercises in persons with stroke.

**Key Words:** Passive cycling, Spasticity, Stroke

**Introduction**

Stroke is defined as the rapidly developing clinical signs of focal or global disturbance of cerebral function, with symptoms lasting 24 hrs or longer or leading to death, with no apparent cause other than that of vascular origin (WHO MONICA, 1988). It is the 3rd most common cause of death following heart disease and cancer, (Wolf PA et. al., 1983). According to Lance (1990) one of the contributors to the post-stroke functional impairment is spasticity. It was estimated that 2/3rd of patients with stroke would develop post-stroke spasticity. Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of stretch reflex as one component of the upper motor neuron (UMN) syndrome. Spastic hypertonia is a major source of disability after stroke. Both reflex and nonreflex changes with spastic hypertonia substantially affect the functional performance of stroke patients. The term ‘hypertonia’ is usually reserved for a perceived resistance to lengthening of muscles to passive movement, (Rymer WZ et. al., 1994). The non reflex contributions include the dynamic component of viscous damping (dashpot-like property with resistance proportional to velocity) and static component of elastic stiffness (spring-like property with resistance proportional to displacement), and reflex changes may have both phasic (dynamic) and tonic (static) components (Meinders M et. al., 1996). Increased resistance to passive movement after stroke is possibly a result of another phenomenon in muscle called “thixotropy” (Nuyens GE et. al., 2002). The term thixotropy refers to the property of certain systems becoming less viscous converted from gel to sol when shaken (Lamontagne et. al., 1989). In muscles, thixotropic changes may occur as a consequence of motion by tearing the cross-bridges between the actin and myosin filaments (Walsh EG et. al., 1992).
Electrical passive pedaling systems (EPPS) are among these innovated rehabilitation technologies by which the physical impairment patients practice physical exercises independently (Therese et. al., 2009). According to Kakebeeke et. al., 2005 the passive movements of the legs preserve full range of motion over the joints that are immobilized and have an effect on spasticity. The mechanism for reduction in spasticity due to the passive cycling results from a decrease in synaptic transmission caused by inactivation of presynaptic calcium channel. Thixotropic changes may occur as a consequence of motion by tearing cross bridges between actin and myosin filament. Mechanical changes in the musculotendinous unit may change (Nuyens GE et. al., 2002). Muscle extensibility and tonic reflex hyperactivity appear to be closely linked, it has been seen that the gradual development of stretch reflex hyperactivity influenced by increasing muscle stiffness and contracture in patients who are relatively immobile; i.e. hyperreflexia may occur as an adaptive response to non-functional, contracted and stiff muscle (Gracies et. al., 1997). ROM at a joint is restricted due to increased stiffness in spastic muscles. Increased stiffness in parietic muscle group is reported by patients 2 months post stroke (Malouin et. al., 1997).

Spasticity results in decreased muscle length and increased muscle stiffness which will hamper person’s ability to exercise, train and regain effective performance of motor actions. Loss of extensibility of calf muscles in particular can potentially have negative effects on the ability to balance in standing, on walking, particularly up and downstairs, and on standing up (Vandervoort et. al., 1999), and is said to predispose the elderly to falls (Studenski et. al., 1991).

No significant experimental studies have been done to know the effect of passive cycling effect on spasticity, range of motion and walking velocity in stroke patient in lower limb except single case study on knee movement on spastic hypertonia in CP and stroke. Thus it was proposed to find out the effect of lower limb passive cycling on spasticity, range of motion and walking velocity in stroke patients.

Materials and Methodology

Selection Criteria: Persons with hemiplegia due to single stroke attack, age group 40-60 years (mean age 46.76 ± 10.09), duration of stroke > 3 month, able to walk 10 meter with or without walking aids.

Exclusion Criteria: Lower extremity traumatic fracture or orthopaedic surgery, with in 3 month of stroke, hemiparesis resulting from non-stroke causes, subjects with hip, knee and plantar flexor contracture and with ankle-foot orthosis.

Sampling Methods: By random sampling subjects were divided into 2 Groups with 15 subjects each

Variables: Independent Variable: Passive electrical cycling, conventional Physical therapy exercises, Dependent Variables: Spasticity by Modified Modified Asworth scale, Range of motion measurement by goniometer, 10 Metre Walk test

Procedure

The whole procedure was explained to each subject and informed consent was taken. These subjects were randomly allocated to either of two groups. Group A (Experimental group: passive cycling and conventional physical therapy exercises; n=15) and Group B (Control Group: conventional physical therapy exercise; n=15). The Spasticity of hip flexor-extensor, knee flexor-extensor and ankle plantarflexors was measured by Modified modified Asworth Scale (MMAS) (Noureddin Nakhostin Ansaria et. al., 2009). The joint range of motion was measured by Goniometer (Brosseau L et. al., 2001) and the walking velocity was measured by 10 meter Walk Test (Gurucharri, Renee Robertson 1999).

Group A Experimental group: subject seated in an adjustable chair in front of Reck Motomed Viva-2 electric cycle and performed passive cycling at 40 RPM for 30 minutes, 5 times/week for 4 weeks. Conventional exercises were given to both the groups, such as unilateral bridging, hamstring stretching, tendoachillis stretching, stepping up onto a step with weight on the hemiplegic leg to facilitate weight bearing on the affected limb.

Pre test measurements were taken prior to recruitment for study and post test measurements were taken at the end of 4 weeks.

Data analysis

The spasticity of hip flexors, hip extensors, knee flexors, knee extensors and ankle plantar flexors were analysed using Man Whitney U test. P value was set at 0.05. Straight leg raising, dorsi flexion range of motion and 10 meter walk test were analysed using a 2x2 ANOVA. There was one between factor (group) with two levels (Experimental
and control) and one within factor (time) with two levels (Pre and Post). All pair wise post–hoc comparisons were analyzed using a 0.05 level of significance. Statistical analysis was performed using SPSS version 16.0.

Results

The results of this study as shown in table-1 indicates better reduction in spasticity in hip flexors, hip extensors, knee flexors, knee extensors and ankle plantar flexors group of muscles from pre to post in experimental group (graph-1). There is statistically significant improvement in dorsiflexion range of motion, SLR as well as walking function in the experimental group but not in control group, over the 4 weeks period. The change in hip flexors spasticity as determined by Man Whitney U test (U value is 44.500 and p value is 0.001), that of hip extensors (U = 60.000 and p = 0.006), knee flexors (U value is 55.500 and p value is 0.010), knee extensors (U value is 62.500 and p value is 0.013), and ankle plantar flexors (U value is 4.500 and p value is 0.000) indicated a significant difference in change of score between groups.

![Graph 1]

Table 1:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Experimental (mean and SD)</th>
<th>Control (mean and SD)</th>
<th>Significance p&lt;0.05</th>
</tr>
</thead>
<tbody>
<tr>
<td>DF</td>
<td>41.33±9.0</td>
<td>31±8.90</td>
<td>0.048</td>
</tr>
<tr>
<td>SLR</td>
<td>68.33±8.38</td>
<td>60.33±11.25</td>
<td>0.318</td>
</tr>
<tr>
<td>TMWT</td>
<td>0.69±0.16</td>
<td>0.55±0.26</td>
<td>0.397</td>
</tr>
<tr>
<td>Spasticity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HF</td>
<td>20.03</td>
<td>10.97</td>
<td>0.001</td>
</tr>
<tr>
<td>HE</td>
<td>19.00</td>
<td>12.00</td>
<td>0.006</td>
</tr>
<tr>
<td>KE</td>
<td>19.30</td>
<td>11.70</td>
<td>0.010</td>
</tr>
<tr>
<td>KE</td>
<td>18.83</td>
<td>12.17</td>
<td>0.013</td>
</tr>
<tr>
<td>PF</td>
<td>22.7</td>
<td>8.30</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Graph-2 shows improvement in dorsiflexion range in experimental group from pre to post test measurement occurred in a greater extent than the control group.

The 2X2 ANOVA revealed that there was a main effect for time \((F = 46.215, \text{df} = 1, p = 0.000)\) and group \((F = 4.270, \text{df} = 1, p = 0.048)\), the main effect also qualified to the interaction of time \(\times\) group in dorsiflexion ROM. The main effect also qualified to interaction of time \(\times\) group: \(F = 11.554, \text{df} = 1, p = 0.002\). Tukey’s HSD post hoc analysis reveals statistically significant improvement in experimental group but not in control group over 4 weeks period. After 4\textsuperscript{th} week they are significantly different from each other.

Graph-3 shows improvement in SLR occurs in a greater extent in experimental group than the control group from pre to post test measurement.

For SLR, there was a main effect for time \(F=101.314, \text{df} = 1, p = 0.000\). There was a main effect also for group, \(F = 1.033, \text{df} = 1, p = 0.318\). The main effect also qualified to interaction of time \(\times\) group: \(F = 36.709, \text{df} = 1, p = 0.000\). Tukey’s HSD post hoc analysis reveals statistically significant improvement in both experimental group as well as control group over 4 weeks period. After 4\textsuperscript{th} week the improvement seen in experimental group is significantly more than control.
Graph-4 shows improvement in 10 meter walking test from pre to post test measurement occurred to a greater extent in experimental group than the control group.

For 10 meter walk test, there was a main effect for time $F = 238.463$, df = 1, $p=0.000$. There was a main effect also for group ,$F = 0.739$, df =1 , $p =0.397$. The main effect also qualified to interaction of time x group : $F = 114.111$, df =1, $p= 0.000$ Tukey’s HSD Post hoc analysis reveals statistically significant improvement in experimental group but not in control group over 4 weeks.

Discussion

The results of the present study showed that there was statistically significant reduction in spasticity in hip flexors, hip extensors, knee flexors, knee extensors and ankle plantar flexors group of muscles from pre to post measurement in experimental group as compared to control group. The study also showed statistically significant improvement in dorsiflexion range of motion, SLR as well as walking function in experimental group.

The change in spasticity in control group can be attributed to therapy comprising of task related training (Carr and Shephered, 1997) including weight bearing, stretching and stepping exercises. According to Merritt (1981) self-stretching, regular physiotherapy and physical activities affect spasticity. Passive movement and stretching represent proprioceptive neuromuscular facilitation, that help in the reduction in spasticity (Bobath 1990). Odeen and Knutsson Kunkel et. al., (1981) reported reduction in spasticity associated with regular periods of passive standing. Reduction in spasticity in experimental group may be due to repetitive movement which reduces resistance of muscle. Clinical evidence has shown that regular mobilizations help prevent contractures and can reduce the severity of spastic tone for several hours. Habitation of reflex activity to repeated stretch may result from a decrease in synaptic transmission caused by inactivation of presynaptic calcium channels. Lamontagne et. al., (1998) found that a decrease in resistance during repeated passive movements without concurrent changes in electromyographic activity was attributable to thixotropic characteristics of the stretched tissues. According to Katz (1991) resistance against passive movement should diminish after repeated motion. Dietz V, Berger W (1983) and O’Dwyer NJ (1996) concluded the mechanical changes in the musculotendinous unit may also be involved. According to Katz (1991) half-hour cycling intervention may reflect a change in the reflex properties. In his study on spastic hypertonia, a clear distinction is made between the intrinsic muscle changes and the altered reflex properties that contribute to heightened muscle tone. MMAS score improve after 30 minutes of passive cycling. In addition, Rosche et. al., (1997) reported that the mean F-wave amplitude, the mean F-wave/M-response ratio, and the maximum F-wave/M-response ratio were significantly lower after leg training with a motorized exercise-bicycle, documenting a decrease in the reflexive component of spasticity. CPM reduces the H-reflex amplitude immediately after the intervention (Chang et. al., 2007).

After 4 weeks of intervention it was found that there was significant improvement in range of motion in terms of dorsiflexion of ankle in experimental group but not in control group. However straight leg raising (SLR) was significantly improved in both the groups. After 4th week, the improvement in experimental group was significantly
more as compared to control group. As the spasticity increases resistance to manual stretch, it leads to diminished passive ROM. So more the stiffness or more the spasticity, the more diminished will be ROM. Therefore, passive cycling was given to reduce the spasticity and increase the passive ROM. Increased ROM after stretching may involve biomechanical, neurophysiologic and molecular mechanisms. Certain basic biomechanical properties influence the muscle-tendon unit’s response to stretch (Taylor et. al., 1990). Tension in muscle comprises passive and active components. Stretch can affect the active component by altering neural activity and the passive mechanical component by affecting viscous and elastic properties of muscle. The mechanisms of increasing muscle length and ROM may be due to the cellular and adaptive mechanisms of a muscle fibre (De Dyne 2001). One characteristic of viscoelasticity is that the tissues respond to stretch and to being held at a constant length with a decrease in tension, i.e., the stress or force produced by the tissues at that length gradually declines, called ‘stress relaxation’ (Duong et. al., 2001). During stretch, soft tissues undergo progressive deformation and they can be progressively extended with a constant force which is called ‘creep’. In this way, finally ROM is increased. Although passive stretching may change the length of the muscle, it is the repetitive stretching during passive cycling that affects the dynamic ROM. The improvement in SLR in control group can be attributed to stretching exercise which improves the range of motion. Manual stretching can increase range of movements, reduce spasticity, or improve walking efficiency (Tamis Pin, 2006). It was seen that sustained stretching of longer duration was preferable to improve range of movements. Static stretching training, performed only three times per week, was sufficient to induce an increase in hamstring flexibility (D.Gallon, A.L.F. Rodacki, 2011). However clinical significance was not observed in the control group.

There was statistically significant improvement in walking velocity (meter/sec) in 10 Meter Walk Test after intervention, in the experimental group as compared to control group at the end of 4 weeks. Reduction in spasticity and increase in range of motion contribute to increase walking speed. The increased plantarflexor spasticity during stance phase and excessive plantarflexion at pre swing phase results in difficulty in foot clearance and therefore increased the swing time. Soleus and gastrocnemius spasticity lead to persistent ankle plantar flexion. Heel strike then could not possible (Shumway-Cook 2002), hence in this way it reduces step length in affected limb. Progression is also hampered by the persistent knee flexion that follows hamstring spasticity limiting the effectiveness of terminal swing (Perry J, 1992). Knee flexion is minimum in the presence of quadriceps spasticity. Hip flexor spasticity similarly (Shumway-Cook 2002) restricts progression in mid and terminal stance, where hip extends normally. According to Bobath this method consists on trying to inhibit increased muscle tone (spasticity) by passive mobilization associated with tactile and proprioceptive stimuli. Ankle plantar flexor contributed to faster comfortable gait velocity, the less the spasticity, the faster the comfortable gait velocity. (An-Lun Hsu, Pei-Fang Tang, 2003) The use of a neuro-developmental approach, focusing on normalizing tone and movement patterns, is widespread and claims that inhibition of spasticity results in an improved motor function (Lennon S., 1980) improvement in walking speed in post-stroke spastic hemiplegia (Gerard E. Francisco 2003).

Conclusion
The results of the study shows that passive cycling was more effective in reducing spasticity of lower limb, improve the range of motion and walking function than any conventional exercises in persons with stroke.

References
24. Perry J, 1992 125