Effect Of Cycle Ergometer On Gait Of Individuals With Spastic Paraplegia

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Study design

Randomized clinical trial

Background

Passive movements performed rhythmically by cycle ergometer claim to have an effect on spasticity reduction, but faster cadences have rarely been investigated for their effects on spasticity.

Purpose

To evaluate the effect of varying cadences of cycle ergometer on gait parameters i.e. walking speed, cadence, stride length and step width of spastic paraplegic subjects.

Setting

Rehabilitation department, Indian Spinal Injuries Center, New Delhi.

Subjects

20 subjects with Thoracic (T7-T10) spinal cord injury.

Material

Cycle ergometer (MOTO med exerciser), paper walkway, ink, measuring tape, and stopwatch.

Methods

Two groups A & B were formed. Group A (control group) underwent cycling for 40 min. at a cadence of 30 rounds per minute (rpm). Group B underwent cycling for 40 min at a starting cadence of 30 rpm with increasing velocities every 10 min., 45%, 55% and 65% finally reaching to 50 rpm for two weeks each day for 5days/week. Gait analysis was preformed after the gait training in both the groups.

Outcome measures

Walking velocity, cadence, stride length and step width were recorded using ink- footprint record and ambulation time.

Data analysis

A paired t-test was used to analyze the differences between gait outcomes. Correlation co-efficient analysis was done to find out the relationship between gait parameters.

Results

A Significant difference existed in all the gait parameters. Experimental group had increased velocity, stride length and step width but reduced cadence than control group. ($p < 0.05$ for each parameter). Velocity and cadence were highly correlated with one another ($r > 0.75$).

Conclusion

Passive cycling at higher speeds has positive effects on gait outcomes of spastic paraplegic individuals, which leads to attainment of functional gait and hence, better quality of life in paraplegic patients.

Key words

Spinal cord injury, paraplegia, cycle ergometer, spasticity, and gait.

Introduction

Spinal cord trauma or disease may result in an incomplete/ complete active functional inability to stand up and to walk. Walking, an important activity of daily living, is a mode of bipedal locomotion in which a period of double support, when both feet are in contact with the ground is followed by a period in which the body is supported by one lower limb while the other is swung forward. [1]

It is well established that the major functional loss following injury to the thoracic spine is the inability of the patient to independently stand up & walk in the environment encountered while engaging in normal daily activities. [2]

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Also, after the stage of spinal shock is over, there are various concomitant symptoms which provide hindrance to the spinal cord injury patient to stand and walk. They include contractures in hip, knee and ankle; the formation of heterotrophic ossification of these joints; pressure sores; spasticity; decreased cardio circulatory and pulmonary function and frequent urinary tract infection due to stagnation of fluids in the bladder. [3,4]

Of these, spasticity is the biggest hindrance for a paraplegic patient to achieve even an upright stance. Spasticity is defined as a symptom of upper motor neuron syndrome characterized by an exaggeration of stretch reflex secondary to hyper excitability of spinal reflexes. [5]

65-75% of individuals with chronic SCI have symptoms of spasticity. In individuals with thoracic spinal cord injury, 72% of those diagnosed as ASIA A and 73% of those diagnosed as ASIA B-D reported symptoms of spasticity.[6]

Spasticity has the potential to negatively affect quality of life through restricting ADLs, inhibiting effective walking and self care. Severity of spasticty is the degree to which walking is effective in functional ambulation after spinal cord injury.[8]

During the past four decades, several interventional strategies have been developed to reduce spasticity in spinal cord injury individuals. These include use of therapeutic stretching; strengthening; serial casting; Positioning; neuromuscular electrical stimulation; aquatic therapy; nerve blocks; medication and surgical intervention.

Although, all of these measures have been effective yet these have some side effects and also the positive effects are not long term. [7]

Motorized exercise cycle has been used to reduce spasticity. Passive movements, when performed rhythmically by cycle ergometer, claim to have an effect on spasticity reduction. Half an hour cycling intervention reflects a change in the reflex properties. Passive cycling has other physiological effects also such as improved blood flow to legs, better metabolic responses and improved aerobic fitness. However, pedal cadences have been limited to 25-30 rpm. Faster cadences have rarely been investigated for their effects on spasticity. It has been shown that pedaling at higher speeds resulted in decreased force output by the paretic limb. [9,10]

So, the main aim of the study was to evaluate the effect varying cadences of cycle ergometer on gait parameters i.e. walking speed, cadence, stride length and step width of spastic paraplegic subjects.

Review of Literature

There are three types of gait used- swing-to-gait, four point gait and swing-through-gait. Controlled walking is achieved only through perseverance, perfect timing, rhythm, and co-ordination. The patient is taught always to move the hands first, to walk slowly and place his feet accurately, to take the weight through the feet and to ensure that the hands can relax between each step, and to lift the body upwards and not to drag the leg forwards. For ambulation over even surfaces for four point gaits, the physical prerequisites are adequate strength in Serratus anterior, Pectoralis major, Latissimus dorsi and Triceps. Also, there must be full range of motion in elbow extension, hip extension and knee extension.[11,12]

Spasticity is a clinical triad of mass spasms, spastic hypertonus and hypereflexia. The physical therapist has a great many interventions at his/ her disposal to assist in managing the patient's hypertonicity. The foundation of spasticity management is therapeutic stretching and strengthening exercises with adjunctive modalities and functional retraining.[12]

Passive range of motion, positioning and serial/inhibitive casting assist in decreasing hypertonicity. Neuromuscular electrical stimulation will cause temporary inhibition of abnormally high tone. The electrical stimulation can be applied to either agonist/ antagonist muscles. Increased weight bearing on affected limb, rhythmic rotation and aquatic therapy have temporary inhibitive effects on hypertonicity. Botulinum toxin (botox) and Phenol nerve blocks are synergistic antispasticity intervention. Medications prescribed to treat spasticity include Dantrolene, Baclofen, Tizanidine, Clonidine and diazepam. Surgical intervention for spasticity management include orthopaedic Tenotomies as well as neurosurgical ablative and neurodestructive procedures.

Ergometer pedaling is an ideal functional exercise. The movement is significantly complex to provide a functionally relevant test for motor performance. Pedaling demands multisegmental co-ordination of bilateral reciprocal symmetrical lower extremity movements in which the muscles go through the periods of activity and subsequent passive lengthening. (13) Giuliani CA et al studied the effect of bicycle pedaling on the temporal-distance parameters and EMG characteristics of walking in hemiplegics subjects. They concluded that bicycle exercise does improve walking velocity and stride length. Potempa et al found increase in maximal oxygen consumption, work load and exercise time as well as lowering of systolic blood pressure during a sub maximal exercise.

A qualitative determination of the degree of tone is made by modified Ashworth's tone assessment scale. It is a six point ordinal scale. The scale has been shown to have high interrater reliability.[14,15]

Methodology

Number of subjects: a sample of convenience of 20 subjects with spinal cord injury.

Source of subjects: ISIC hospital, Vasant kunj, New Delhi.

The criteria for selection of subjects were:

Inclusion criteria

- Spinal cord injury subjects at least 3 months post injury with a stable spine and no significant kyphoscoliotic deformity.
- Neurological level from T7-T10.
- ASIA impairment grade A
- Spasticity grade 1+ to 3 on Ashworth's scale.
- Medically stable.
- Full Range of motion in hip, knee and ankle.

Exclusion criteria

* Any complication such as pressure sore, urinary tract infection, autonomic dysreflexia and postural hypotension.
* Visual impairment (if any, then successful use of corrective lenses)
* Any painful musculoskeletal or joint problems affecting upper limb.

Method of selection

All the spinal cord injury patients attending the rehabilitation department of ISIC hospital were evaluated and those meeting the inclusion and exclusion criteria and willing to give consent to participate in the study were included in the study.

The subjects were then randomly allocated into two groups: Group A (constant speed cycling) and group B (graded increase in speed of cycling).

Design of the study

Randomized Controlled Experimental Design.

Instrumentation for data collection

1. Cycle ergometer (MOTOmed exerciser)
2. Parallel bars with a length of 5 meters.
3. Digital stopwatch.
4. Oil paint
5. A standardized inch tape & scale.

Variables used in the study

Independent variable: Cycle ergometer

Dependent variables

1. Walking velocity
2. Cadence
3. Stride length
4. Step width

Procedure

Participants were explained about the purpose and nature of study and the informed consent was obtained from those willing to participate

Demographic details and the history like the name, age, gender, body weight, time since injury, ASIA level, functional leg length, spasticity scoring on modified Ashworth's scale for knee extensors, knee flexors and hip abductors was obtained.

Pre- experimental protocol

An initial gait assessment was performed for all the subjects within the parallel bars fitted with a 3 meter walkway. Walking velocity, cadence, stride length and step width were recorded using ink footprint record method and ambulation time.

Each subject initially walked from right to left and following a 45-60 second break for turning, walked from left to right. By noting the time spent walking to each direction, an average walking speed was obtained.

Numbers of steps per minute were counted to calculate the cadence.

Foot prints were taken with the help of oil paint applied on the sole of the patient. Stride length and step width were recorded.

Stride length was calculated by measuring the distance from heel strike of one extremity to the heel strike of same extremity again in the next step using a standardized scale. Stride length was divided by the functional leg length for each subject to normalize the differences in the patient's leg length.

Step width was calculated by measuring the linear distance between the mid point of the heel of one foot and the same point on the other foot.

Patient Seated on Cycle Ergometer.

Three trials were taken for each assessment and the mean for each outcome measure calculated.

Adequate rest periods were given in between the testing as per the patient's will.

Post experimental protocol

All the subjects were placed in their own wheelchair in front of the motorized cycle ergometer. Group A underwent cycling for 40 minutes at a cadence of 30 rpm. Group B underwent cycling for 40 min at a staring cadence of 30 rpm (rounds pre minute) with increasing velocities every 10 min. 45%, 55% and 65% finally reaching to 50 rpm.The regimen continued for two weeks (5 days/ week) gait assessment was done at the end of regime and the four parameters i.e. walking velocity, cadence, stride length and step width were recorded.

Data was recorded in the gait analysis recording form.

Data Analysis

Statistics was performed using the STATA 7.0 and SPSS software. A paired t test was used to analyze the difference between the performances of the subjects in the two groups A and B before and after the intervention, which showed a significant p- value ($p<0.05$). Correlation co-efficient analysis was done to find out the relationship between various gait parameters.

Results

Comparison of gait parameters between two groups:

There was a significant difference in velocity of both the groups before and after the intervention.

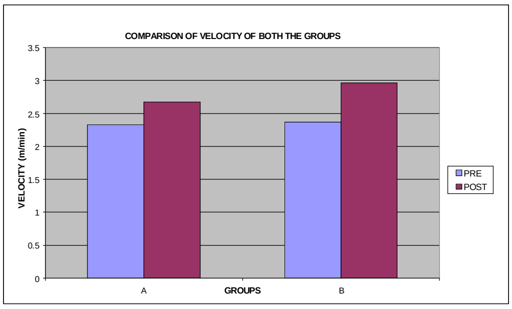
GAIT PARAMETERS OF GROUP A (CONTROL GROUP)

VARIABLE	PRE-INTERVENTION	POST INTERVENTION
VELOCITY (m/min)	2.33+- 0.30	2.37+- 0.28
CADENCE (steps/min)	11.7+- 1.05	11.9+- 1.09
SL/LEL	0.83+- 0.44	0.84+- 0.40
STEP WIDTH (cm)	3.58+- 0.79	3.62+- 0.94

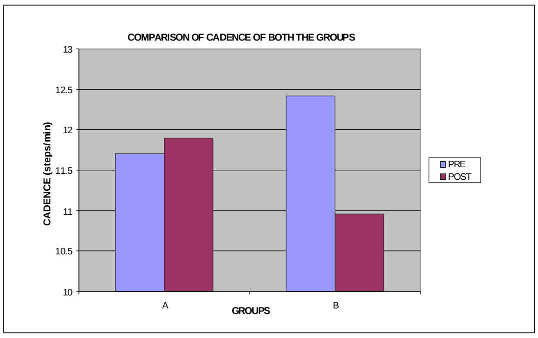
GAIT PARAMETERS OF GROUP B (EXPERIMENTAL GROUP)

VARIABLE	PRE-INTERVENTION	POST INTERVENTION
VELOCITY (m/min)	**2.67+- 0.17**	**2.96+- 0.24**
CADENCE (steps/min)	**12.42+- 0.26**	**10.96+- 0.35**
SL/LEL	**0.81+- 0.48**	**0.88+- 0.74**
STEP WIDTH (cm)	**4.1+- 0.62**	**5.1+- 0.36**

A significant positive correlation (r= 0.78) was found in group A when values of velocity were compared with values of cadence and group B (r=0.76) when values of velocity were compared with that of cadence.



GRAPH-1



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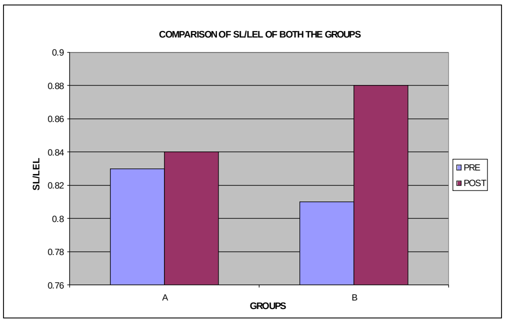

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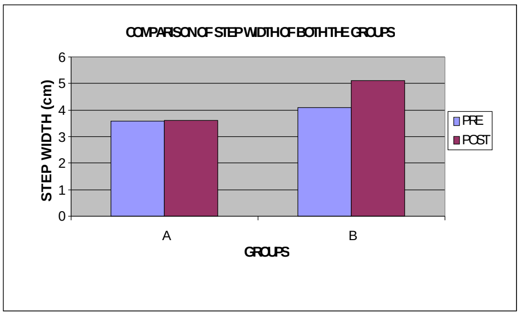

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Discussion

In our experiment, we studied the effects of rhythmic passive cycle movements of the lower limb on spasticity. There was significant difference in gait parameters of subjects in the two groups. Experimental group had increased velocity, stride length and step width but reduced cadence than control group.

Motorized exercise cycle has been used previously to treat spasticity.Reflexive responses of spasticity strongly habituated during testing.[20] A clear distinction is made between the intrinsic muscle changes and the altered reflex properties that contribute to the heightened muscle tone. First factor seems to be dependent on the muscle mechanical moments and the passive viscoelastic elements and is very stable. Also, it includes fibrosis, muscle fiber atrophy, reduction in the elastic properties, decrease in the number of sarcomeres, accumulation of connective tissues and alteration of contractile properties. In contrast, the short term variation in torque is attributed to changes in stretch reflex properties.[21,2] This would imply that a half hour cycling intervention may reflect a change in the reflex properties which were subject to testing in the experiment.

With the passive cycling device, the knee is indeed flexed and extended during half an hour. muscles were stretched during the cycling but not to the end of range of motion.

Alpha neuron excitability develops secondarily after alternation of some other segmental mechanisms in those with spasticity (e.g. increased alpha motor neuron altered interneuron activity and decreased pre- synaptic inhibition).[23,24] Initially, it is the hyperexcitability of this tonic stretch reflex that is commonly thought to result in increased muscle tone in response to passive stretch following spinal cord injury. The development of tonic stretch reflex hyper excitability could be due to a lower threshold, an increased gain of the stretch reflex or a combination of the two. The resultant increase in muscle tone is thought to be due to a combination of increased denervation hypersensitivity and changed muscle properties. Denervation leads to an initial down regulation of neuronal membrane receptors followed by an up regulation of enhanced sensitivity to neurotransmitters.[25]

Heightened muscle tone is mainly due to intrinsic muscle changes half an hour cycling intervention at single speed would lead to a change in the phasic properties pf a muscle. Different velocity cycling would have an effect on muscle mechanical moments and the passive viscoelastic elements. Also there is an increased threshold of tonic stretch reflexes in which constant cycling has no effect.[26]

Faster stepping speed resulted in greater afferent feedback to the CNS which in turn may lead to up regulation of neuronal membrane receptors and hypersensitivity to neurotransmitters.

Physical therapists have traditionally believed that faster speeds might increase unwanted muscle activation or spasticity but there appears to be little evidence for this notion.

Increasing pedaling speeds at every 10 minutes required earlier onsets of muscle activity to reach peak force which lasted only for 1 minute, after which it decrease which may result form a decrease in synaptic transmission caused by inactivation of pre- synaptic calcium channels.

It was found that a reduction in resistance during repeated passive movements without concurrent changes in EMG activity was attributable the thixotropic characteristics of a stretched muscle. Mechanical changes in musculotendinous units may also be involved.

Previous studies have shown that there is no increased amount of EMG activity at progressively faster speeds during the prolonged activation of Vastus Medialis- a speed dependent reflex effect could not be identified.[25,22]

Pedaling at faster speeds resulted in reduced force output by the paretic limb. At faster pedaling speeds, the decrease in total work done by the paretic lower extremity was primarily accounted for by increase in the resistive component. Duration of agonist burst in absolute time was actually lessened at faster speeds because the effect of muscle activity in the Vastus Medialis, Rectus Femoris and Semimembranosus occurred at similar points in the crank cycle at progressively faster speeds.

Mechanical demands of faster pedaling speeds were the major contribution factor to the reduction in force output that occurred at faster pedaling speeds. The hypothesis implies that there is no harm to the nervous system when training individuals with spasticity to pedal at fast speeds.[23,22]

Increased pedaling speeds required earlier onsets of activity to reach a peak force at appropriate points in the cycle. In addition, at faster speeds and speed- dependent interaction forces (e.g. inertial forces such as corioli's forces) decreased in magnitude.

Given these mechanical alterations at higher speeds, it is believed that nervous system must develop strategies to deal with altered mechanics. Increased inappropriate activity would involve the exacerbation of EMG timing abnormality so that greater prolonged activity e.g. in Vastus Medialis may occur. Speed dependent EMG timing alteration so that peak force generation would be delayed. In the case of pedaling, an inability to generate earlier onset of muscle activity at faster speeds will result in peak pedal forces being generated at later, less appropriate regions of the crank cycle.[27]

Consclusion

The findings support the hupothesis that passive cycling at higher speeds has positive effects on gait outcomes of spastic paraplegic individuals, which leads to attainment of functional gait and hence, better quality of life in paraplegic patients.

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