

Original Article

The effect of passive cycling movements on spasticity after spinal cord injury: preliminary results

TH Kakebeeke^{*1}, HE Lechner¹ and PA Knapp²

¹Institute for Clinical Research, Swiss Paraplegic Centre, Nottwil, Switzerland; ²ALEA Solutions GmbH, Zürich, Switzerland

Objective: To investigate the influence of rhythmic passive movements of the legs on the reduction of spasticity after spinal cord injury (SCI).

Setting: Swiss Paraplegic Centre Nottwil, Switzerland.

Methods: A total of 10 subjects with motor complete SCI were treated with a cycling device for half an hour. Before and after cycling their spasticity was tested with an isokinetic dynamometer. The subjects were tested one week later by exactly the same procedure with a half an hour break instead of the cycling. Subjects were asked about their spasticity before and after the cycling and break.

Results: There was no significant difference in elicited peak torque either before and after the cycling, or before and after the break (MANOVA, $P < 0.05$). Six out of 10 subjects estimated their spasticity as less after the cycling.

Conclusion: With the isokinetic dynamometer, it was not possible to show an effect of passive cycling on spasticity reduction. However, six out of 10 of the subjects estimated their spasticity to be less after cycling. This positive effect might be attributed to a reduced spasticity in the trunk and/or to the attention the subjects perceived during the intervention.

Spinal Cord (2005) 43, 483–488. doi:10.1038/sj.sc.3101747; published online 12 April 2005

Keywords: spasticity; passive cycling; spinal cord injury

Introduction

One of the concomitant symptoms of spinal cord injury (SCI) above T12 is spasticity. Out of a studied population of 354 SCI subjects, 65% report spasticity and about one-quarter of these subjects uses medication for alleviation.¹ The National Spinal Cord Injury Statistical Center (Birmingham, USA) claims that 42.7% of individuals with SCI are treated for spasticity in the first year after injury. At 10 years after injury, 35% are still treated for spasticity. Severe spasticity is mostly treated with medication.^{2,3} The side-effects of antispastic medication are sometimes very strong and subjects frequently complain of dizziness and drowsiness.⁴ Moreover, since the life expectancy of SCI subjects has increased considerably, this implies a dependence on drugs for decades.⁵

It is for this reason that many patients are in search of conservative measures that may influence spasticity. The main conservative means of reducing spasticity is physiotherapy. A physiotherapeutic session consists

mainly of active exercises, which are performed by the patient and passive movements for those exercises, which the subject cannot perform him- or herself.^{6,7} The main aim of passive movements of the legs is to preserve full range of motion over the joints that are immobilized.

Passive movements of the legs, which are performed rhythmically, are a part of certain therapies that claim to have an effect on spasticity reduction.⁸ Resistance against passive movement should diminish after repeated motion.⁹ According to the Bobath therapy, a failing reciprocal innervation is one of the fundamental problems in spastic patients. To get sensory cues is important in the therapies of Rood, Kabat, Brunnström and Johnstone.¹⁰ However, an underlying physiologic paradigm that explains why rhythmic, passive movements have an influence on spasticity in patients with a lesion of the upper motor neuron is equivocal.⁹

Currently, there is equipment on the market with which the legs can be passively moved in a standardized way for long periods of time. One of the effects is supposed to be a reduction of spasticity. We are

*Correspondence: TH Kakebeeke, Institute for Clinical Research, Swiss Paraplegic Centre, CH-6207 Nottwil, Switzerland

interested in the effect of these devices on the elicited torques in the legs before and after subjects perform rhythmic, passive movements of the legs.

Recently, the assessment of spasticity was performed by using isokinetic dynamometry in patients with SCI.^{11–13} With this technique, it is possible to test the resistance to passive movement in the joints with preset speeds at different lengths of the same muscle. As one of the definitions of spasticity is a velocity-dependent increase in muscle tone to passive stretching,¹⁴ isokinetic dynamometry seems to be a suitable technique suited for this purpose. Although the use of the Ashworth and Modified Ashworth Scale is widespread, clinically valuable and fairly reliable,^{15,16} these scales are still subjective and therefore less suitable for objective measurements. With isokinetic dynamometry, we hope to gather an objective measure of torque differences before and after a therapeutic intervention.

However, we also want to know whether the subjects think that this device has an influence on their spasticity. As SCI subjects are a potential market for motorized exercise-cycles, it is important to know what kind of effect these passive movements have on them. It is clear that this will be a subjective measure.

With the two approaches (objective and subjective measures), we hope to disentangle the pure physical (objective) effect from the subjective effect of a therapeutic session in which rhythmic, passive cycle-movements are performed.

Methods

In all, 10 subjects with motor complete SCI (ASIA A, except one with ASIA B) for at least 12 months were studied (out of 16 that were recruited). Subjects who were included were known to have spasticity and had a sufficient range of motion of hip, knee and ankle to perform the test and the intervention. Those subjects were excluded who suffered from joint problems, pain, severe osteoporosis, urinary tract infection, and skin pressure sores. Median age of the subjects was 42 years (range, 23–60) and median time postinjury was 2 years (range, 1–25). Level of injury ranged from C6 to T12 (Table 1). Before testing began, subjects were informed about the purpose of the study. A consent form was signed by each subject and the subjects were free to withdraw from the experiment at any time. A complete experimental set-up was located in the Swiss Paraplegic Centre (Nottwil, Switzerland) where all testing took place. Before testing, each subject was asked to empty the bladder.

Subjects were tested twice, on two separate days, so that they served as their own controls. Firstly, subjects were tested for spasticity according to the same protocol as described in an earlier work of the same group.¹⁷

For spasticity testing, an isokinetic dynamometer (Cybex Norm II, Lumex Inc. Ronkomkoma, NY, USA) was used. In the sitting and lying position, the lower limb of the right leg was moved at two different speeds (10° and 120° per s). The range of motion (ROM) was

Table 1 Subjects characteristics

Subject	Neuro-logical level	ASIA impairment scale	Sex	Age	Years post injury
1	C6	B	M	31 y 7 m	2 y 9 m
2	T5	A	M	44 y 4 m	2 y 4 m
3	C8	A	F	44 y 9 m	23 y 2 m
4	T5	A	M	42 y 1 m	1 y 4 m
5	T11	A	M	60 y 3 m	2 y 2 m
6	T3	A	M	50 y 5 m	8 y 8 m
7	T4	A	M	23 y 4 m	3 y 2 m
8	T3	A	M	45 y 6 m	25 y 0 m
9	T12	A	M	23 y 10 m	10 y 7 m
10	T10	A	M	38 y 1 m	1 y 7 m

y = years; m = months

equally defined for both positions so that we could test extensors and flexors of the knee at different lengths. As was shown in an earlier work of the same group,¹⁷ the knee extensors are prestretched in the lying position and therefore spasticity is easily elicited. On the other hand, the knee flexors are prestretched in the sitting position and therefore in this position more excitable. The knee was flexed twice with 10° per s to record the passive resistance at the end of the ROM. Then the speed was increased to 120° per s to elicit spasticity. With this speed, the knee was flexed four times.

Following the spasticity measurements, the subjects were placed in their own wheelchair in front of a motorized cycle (THERA-vital, Medica Medizin GmbH, Hochdorf, Germany). With the THERA-vital, the legs can be passively moved in a standardized way for long periods of time. The subjects cycled for 30 min at a cadence of 40 rounds per minute (rpm). The ROM of the flexion/extension movement in the knee was at least 50° to 65° depending on the flexibility and the length of the legs of the subject. Immediately after cycling, the subjects were transferred back to the isokinetic dynamometer and were tested again with the same protocol.

The second series of experiments took place at the same time of day exactly one week later without the cycling intervention. The time between the first and second measurement was spent in the same room with the same experimenters and the subject was also transferred to his own wheelchair for a break of 30 min. An overview of the protocol is given in Table 2.

We recorded torque, angle, direction and speed with 3000 Hz. Measurement and data analysis were performed with SOLEASY (ALEA Solutions GmbH, Zürich, Switzerland). This is a software package based on LabView, National Instruments. We analysed peak torque and the sum of the four consecutive torques (Σ torque) during all movements.

For the statistical analysis, we used a MANOVA (repeated measures analysis of variance). For each person, peak torque before and after cycling and before and after the break of half an hour were analysed.

This was performed for the lying and sitting position. For the Σ torque, the same procedure (MANOVA) was performed. We chose P -values <0.05 as level of significance.

Moreover, all subjects were asked the following question, after cycling and after the break, but prior to the second dynamometer test: 'Do you think you are more, less or equally spastic?'

Results

From the original 16 enrolled patients, we were able to elicit spasticity from 10 subjects in the lying position. Out of these 10, six showed spasticity of the hamstrings in the sitting position. We analysed the peak torque of

the 10 subjects in the lying position and of the six subjects in the sitting position. The peak torque and Σ torques provoked by the faster movements (120° per s) are presented in Tables 3 and 4 for the lying and sitting position, respectively. There was no significant difference in peak torque or in Σ torque before and after the cycling intervention or the half-hour break.

The torques at slow speed (10° per s) were not used for the analysis of the spasticity reduction as they only represented the passive forces at the end of the ROM.¹⁷

As far as the subjective feelings of the subjects are concerned, six out of 10 indicated they would be less spastic after cycling and, three subjects out of 10 indicated they would be less spastic after the break of half an hour (Table 5).

Table 2 Test protocol

Test 1	Spasticity testing	Transfer in wheelchair	Cycling	Transfer on dynamometer	Spasticity testing
Test 2	Spasticity testing	Transfer in wheelchair	Break while sitting in wheelchair	Transfer on dynamometer	Spasticity testing

Table 3 Knee extensor PT and Σ PT in the lying position

Subject	PT before cycling	PT after cycling	PT before break	PT after break	Σ PT before cycling	Σ PT after cycling	Σ PT before break	Σ PT after break
1	31.3	30.7	36.5	30.4	79.3	86.2	88.7	104.5
2	37.7	38.6	39.3	34.3	132.1	137.9	142.3	122.1
3	15.7	11.9	17.5	16.2	26.8	21.5	29.8	27.3
4	4.0	2.1	3.6	1.5	4.8	3.5	4.4	2.7
5	46.1	43.0	45.3	46.7	170.0	162.1	166.2	154.6
6	14.2	21.6	16.9	9.9	44.0	71.5	50.3	9.9
7	4.2	2.2	10.1	14.1	4.2	4.3	15.3	20.6
8	24.3	18.7	25.5	18.8	78.3	55.5	66.5	66.8
9	6.7	12.8	12.0	8.5	25.9	46.6	40.3	32.5
10	14.1	9.9	4.3	9.3	36.8	23.5	7.2	12.8
Mean (SD)	19.8 (14.6)	19.2 (14.3)	21.1 (14.9)	19.0 (14.0)	60.2 (55.0)	61.3 (54.4)	61.1 (55.9)	55.4 (53.8)

PT = peak torque; Σ PT = sum of peak torques

Table 4 Knee flexor PT and Σ PT in the sitting position

Subject	PT before cycling	PT after cycling	PT before break	PT after break	Σ PT before cycling	Σ PT after cycling	Σ PT before break	Σ PT after break
3	21.1	13.5	12.8	11.7	84.3	53.9	51.1	46.9
4	8.4	11.5	28.3	18.3	33.4	46.2	113.1	73.2
5	26.5	25.0	25.2	31.3	106.1	100.0	100.7	125.2
6	10.3	32.9	37.0	12.2	117.8	100.1	164.2	120.2
8	11.1	7.8	10.8	10.1	44.3	31.1	43.3	40.4
9	15.7	4.7	13.3	12.8	62.9	18.9	53.1	51.2
Mean (SD)	15.5 (7.1)	15.9 (10.8)	21.2 (10.6)	16.1 (8.0)	74.8 (33.8)	58.4 (34.5)	87.6 (47.3)	76.2 (37.7)

PT = peak torque; Σ PT = sum of peak torques

Table 5 Impression of spasticity

Subject	After cycling	After break
1	Less	Equal
2	Equal	Equal
3	Less	Equal
4	Less	Equal
5	Less	Equal
6	More	Less
7	Less	Less
8	Less	More
9	More	Equal
10	Equal	Less

Discussion

In this experiment, we studied the effect of rhythmic, passive cycle-movements of the lower limbs on spasticity. We found no significant difference in evoked peak torque before and after cycling. This was the case for peak torque and Σ torques of both knee extensors and flexors.

In a study by Rösche *et al*⁸ a motorized exercise-cycle was also used to treat spasticity. However, this was carried out on subjects who suffered predominantly from multiple sclerosis. In this study, the motor neuron excitability was investigated by means of the mean F-wave amplitude, mean F-wave/M-response ratio and maximum F-wave/M-response ratio. The article focuses on how to parameterize the motor neuron excitability in multiple sclerosis and not so much on the reduction of spasticity after the cycling intervention. It was concluded that the antispastic effect of the treatment can be best documented with the recording of the F-wave-amplitude and that there was a slight significant decrease of this amplitude after treatment. As this study was performed only on multiple sclerosis patients and showed equivocal results, we were interested to study the effect of rhythmic, passive movements in SCI subjects.

Repeated passive movements in the knee were also studied in an article from Nuyens and her group.¹⁸ In this experiment, 10 flexion and extension movements were imposed on the knee at different speeds (60°, 180° and 300° per s). The highest decline in resistive torque took place during the first movements of the test repetitions, indicating that the reflexive response strongly habituated during testing. In our experiment, we investigated four repetitions after the intervention. However, it took at least 10 min after the cycling to transfer the subject back to the dynamometer and reposition him as before the intervention. The possible effect of the rhythmic movements may therefore already have been eliminated before we were able to test the subject.

In a review article by Katz and Rymer¹⁹ on spastic hypertonia, a clear distinction is made between the

intrinsic muscle changes and the altered reflex properties that contribute to heightened muscle tone. The first factor seems to be dependent on the muscle mechanical moments and the passive viscoelastic elements, and is very stable. In contrast, the short-term variations in torque are attributed to changes in stretch reflex properties.¹⁹ This would imply that a half-hour cycling intervention may reflect a change in the reflex properties, which were subject to testing in this experiment. With the passive cycling device, the knee is indeed flexed and extended during half an hour; however, we were not able to show an effect of these rhythmic movements of the legs on the elicited torque. It should be stressed that the muscles were stretched during the cycling but NOT to the end of ROM. We used a commercial device that enabled us to move the knee within a ROM of 50–65°. This was not real stretching for any of our subjects, it was merely a rhythmic flexion/extension movement within a segment of their ROM. This may have been why we were not able to see an effect of stretching as it is understood by the physiotherapist, when the joint of the subject is moved to the end of ROM and the muscles are really stretched.^{20,21}

We deduced from the cycling behaviour of our subjects that we did not elicit spasticity. The security break that stops the cycling when spasticity occurs, did not come into operation in any of the subjects. This may be explained by the speed with which our subjects cycled. With 40 rpm they developed a speed of 60–86° per s (with a ROM of 50–65°). This was well under the speed with which we elicited spasticity during the test. We cannot exclude the effect of a higher speed as was performed with this cycle on spasticity reduction; we only tested one speed.

As our subjects, apart from one, were all ASIA A, we asked ourselves, how they might 'feel' that they were less spastic. Finally, ASIA A implies no sensory function below the lesion. We presume that those subjects who felt a change, actually may have felt a change in the spasticity of the trunk. Severe trunk spasticity has an influence on breathing and posture and gives the subject indirect information regarding how strong his spasticity is. We asked the subjects about their spasticity in general, although nine out of 10 were only able to feel the indirect responses of spasticity of the trunk. From the spasticity in the legs they had no actual feelings. In this study, we tested spasticity in the legs, and spasticity of the trunk was not a topic.

The subjective feelings of the subjects concerning the cycling intervention were in contrast to what we gathered with the isokinetic dynamometer. Although we could not prove that the cycling produced a reduction in spasticity of the lower limbs, six subjects were convinced that they would be less spastic after the cycling. Obviously, the belief the subjects had in this apparatus, combined with the attention they had experienced for their paralyzed limbs, gave them the impression to be less spastic. Therefore, the cycling intervention was suggested to have a good influence on their perception of spasticity reduction.

As the rhythmic, passive movements of the legs are applied mostly as a part of a physiotherapy session, it is difficult to prove the effect of only this aspect of the therapy on the patient's spasticity. Moreover, rhythmic, passive movements are difficult to perform in a standardized way, as they are individually performed by hand by the physiotherapist. The effectiveness of spasticity reduction in the legs by a physiotherapeutic routine is in this way difficult to prove. Other very important factors that may contribute to a reduction in spasticity, such as belief in the therapy and attention from the therapist, are even more difficult to control. For this reason, it is not easy to perform experimental studies that control all therapeutic aspects of one therapy in order to study one part of it. Most studies in the therapeutic field therefore concentrate on the comparison of data before and immediately after therapeutic intervention. The effect is then attributed to the entire therapy session and not to a certain aspect of the therapy.

We think this is a very important aspect of any therapeutic device. In the case of this cycling apparatus, the paraplegic subject sees his limbs moving as he is cycling. This may have an effect on how he will judge the spasticity of his lower limbs. The most frequently heard statement from our subjects was: 'my limbs have been moved for 30 min, so therefore they have to be less spastic' and so they answered that they 'felt' less spastic (all but one were ASIA A). Also, three out of 10 felt less spastic after the half an hour break in which they just talked to the experimenter.

Therapeutic interventions are difficult to study double blinded, randomized and prospective, and impossible if movement is involved. During our intervention and control experiment (the half-hour break), the subjects receive attention, make transfers from and back to their chairs and invest time to reduce their spasticity. It is very difficult to attribute positive feelings the subject may have after half an hour of any therapy to only the cycling as so many things are involved. It may have been the attention the subjects have given to their legs that gave them the feeling they were less spastic.

In this context, we would like to refer to the study on the social placebo effect, first reported in 1924, also known as the 'Hawthorne' effect named after the Hawthorne plant where these studies were performed.²² In this plant, the workers were informed that they were participating in a scientific experiment (the intervention and the controls), and productivity increased in both groups. The fact that the workers had the feeling that they were taken seriously and were being observed already made them more motivated and therefore they worked harder. This effect cannot be excluded in therapeutic interventions where social contact is involved.

Conclusions

In this experiment, we measured the spasticity of the knee flexors and extensors after rhythmic passive

movements of the legs in SCI subjects. We could not show any reduction in spasticity after the intervention with an objective measure. However, subjectively, the patients profited from the cycling. They had the feeling they were less spastic after the cycling. We do not know whether this is caused by the movements of the legs and slight movement of the pelvis, the attention of the physiotherapist or the knowledge of doing something to control spasticity. The combination of all these factors together gave the subjects the feeling they were less spastic after the cycling.

References

- Sköld C, Levi R, Seiger A. Spasticity after traumatic spinal cord injury: nature, severity, and location. *Arch Phys Med Rehabil* 1999; **80**: 1548–1557.
- Penn RD *et al*. Intrathecal baclofen for severe spinal spasticity. *N Engl J Med* 1989; **320**: 1517–1521.
- Priebe MM, Sherwood AM, Thornby JI, Kharas NF, Markowski J. Clinical assessment of spasticity in spinal cord injury: a multidimensional problem. *Arch Phys Med Rehabil* 1996; **77**: 713–716.
- Kirshblum S. Treatment alternatives for spinal cord injury related spasticity. *J Spinal Cord Med* 1999; **22**: 199–217.
- Whiteneck GG *et al*. Mortality, morbidity, and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia* 1992; **30**: 617–630.
- Buck M, Beckers D. *Rehabilitation bei Querschnittlähmung*. Springer-Verlag: Berlin, Heidelberg 1993.
- Bromley I. *Tetraplegia and Paraplegia* 5th edn. Churchill Livingstone: Edinburgh, London, New York, Madrid, Philadelphia, San Francisco, Sydney, Toronto 1998.
- Rosche J, Paulus C, Maisch U, Kaspar A, Mauch E, Kornhuber HH. The effects of therapy on spasticity utilizing a motorized exercise-cycle. *Spinal Cord* 1997; **35**: 176–178.
- Katz RT. Management of spastic hypertonia after stroke. *J Neuro Rehab* 1991; **5**: S5–S12.
- Partridge C. Physiotherapy approaches to the treatment of neurological conditions – an historical perspective. In: Edwards S (ed). *Neurological Physiotherapy: a Problem-solving Approach*. Churchill Livingstone: New York 1996 pp 3–14.
- Franzoi AC, Castro C, Cardone C. Isokinetic assessment of spasticity in subjects with traumatic spinal cord injury (ASIA A). *Spinal Cord* 1999; **37**: 416–420.
- Perell K, Scremin A, Scremin O, Kunkel C. Quantifying muscle tone in spinal cord injury patients using isokinetic dynamometric techniques. *Paraplegia* 1996; **34**: 46–53.
- Akman MN, Bengi R, Karatas M, Kilinc S, Sözyay S, Özker R. Assessment of spasticity using isokinetic dynamometry in patients with spinal cord injury. *Spinal Cord* 1999; **37**: 638–643.
- Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. *Neurology* 1980; **30**: 1303–1313.
- Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner* 1964; **192**: 540–542.
- Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth Scale of Muscle Spasticity. *Phys Ther* 1987; **67**: 206–207.
- Kakebeeke TH, Lechner H, Baumberger M, Denoth J, Michel D, Knecht H. The importance of posture on the

- isokinetic assessment of spasticity. *Spinal Cord* 2002; **40**: 236–243.
- 18 Nuyens GE, De Weerd WJ, Spaepen Jr AJ, Kiekens C, Feys HM. Reduction of spastic hypertonia during repeated passive knee movements in stroke patients. *Arch Phys Med Rehabil* 2002; **83**: 930–935.
 - 19 Katz RT, Rymer WZ. Spastic hypertonia: mechanisms and measurement. *Arch Phys Med Rehabil* 1989; **70**: 144–155.
 - 20 Guissard N, Duchateau J, Hainaut K. Muscle stretching and motoneuron excitability. *Eur J Appl Physiol Occup Physiol* 1988; **58**: 47–52.
 - 21 Odeen I. Reduction of muscular hypertonus by long-term muscle stretch. *Scand J Rehabil Med* 1981; **13**: 93–99.
 - 22 Wickstrom G, Bendix T. The ‘Hawthorne effect’ – what did the original Hawthorne studies actually show? *Scand J Work Environ Health* 2000; **26**: 363–367.