Original Article

Assessment of the short-term effect of antispastic positioning on spasticity

TURKAN AKBAYRAK,¹ KADRIYE ARMUTLU,¹ MINTAZE KEREM GUNEL¹ AND GULAY NURLU²

¹School of Physical Therapy and Rehabilitation, and ²Department of Neurology, Hacettepe University, Samanpazari, Ankara, Turkey

Abstract Background: This study was performed in order to investigate the effect of antispastic positioning on spasticity by using different assessment methods.

Methods: A total of 16 patients (11 males [68.75%] and five females [31.25%]), diagnosed as spastic diplegic and referred to the School of Physical Therapy and Rehabilitation, Paediatric Rehabilitation Unit for treatment, were included in this study. The mean age of patients was 6.43 ± 1.99 years (range, 4–13 years). Passive dorsiflexion movement was measured by using goniometer and intensity of spasticity was determined by using Modified Ashworth Scale (MAS) and electromyography. For the objective measurement of severity of spasticity, Hoffman reflex (H) and Hoffman reflex/motor response (H/M) ratios were used. Children were placed in an antispastic position for 20 min. The patient was placed in a sitting position, by a physiotherapist, with hips abducted at a 45° angle and externally rotated, knees extended, and ankles placed in a neutral position.

Results: The decrease in H responses, H/M ratios, MAS values and the increase in goniometric measurement values were found statistically significant after antispastic positioning (P < 0.05).

Conclusion: Our study supports that antispastic positioning can be used with neurodevelopmental treatment approaches when it is required. Antispastic positioning may help exercises to be performed more easily, and also has importance in a home exercise program to prevent muscle contractures and joint limitation in children with long-term spastic diplegia.

Key words antispastic position, cerebral palsy, reduction of spasticity.

Spasticity is characterized by the increase in tendon reflex and tonic stretch reflex because of the hyper-excitability of the stretch reflex after upper motor neuron lesion.^{1,2} Spastic cerebral palsy (CP) results from dysfunction in the corticospinal tracts leading to increased muscle tone, hyper-reflexia, and the persistence of primitive reflexes.³ Spastic diplegia is characterized by bilateral spasticity, typically greater for the legs than the arms.⁴ The ratio of occurrence of spastic diplegia is 32% in children with CP.^{5,6}

Spasticity mostly affects flexors, adductors and internal rotators of the hip, and plantar flexors of the ankle.⁷ Spasticity in the lower extremities has adverse effects on sitting posture and this leads to an impairment in the stabilization of columna vertebralis.⁸ Equine position, pes varus and spastic

Correspondence: Mintaze Kerem Gunel, PT, PhD, Associate Professor, Hacettepe University School of Physical Therapy and Rehabilitation, Samanpazari, 06100 Ankara, Turkey.

Email: mintaze@yahoo.com

Received 3 January 2002; revised 30 November 2004; accepted 9 December 2004.

pes valgus are problems frequently seen in spastic diplegic children who have spasticity of the adductor and internal rotators.⁹

Spasticity may cause abnormal posture and movement patterns, and a delay in motor development and ambulation such as sitting, crawling and walking. For this reason, inhibition of spasticity that restricts the development of the child is essential in rehabilitation.¹⁰

In addition to medical treatment and surgical interventions, physiotherapeutic methods such as electrical stimulation,¹¹ biofeedback,¹² inhibitory orthosis,¹³ Johnstone pressure splints,¹⁴ and the application of cold¹⁵ and vibration¹⁶ are also used for this purpose. Although, recently, application of oral and intrathecal Baclofen,¹⁷ injection of Botolunuim Toxin,¹⁸ and orthopaedic approaches¹⁹ are frequently used, exercise therapy²⁰ is used in addition to these methods to increase their effects. Exercise therapy includes Proprioceptive Neuromuscular Facilitation (PNF) techniques, 'handling position' of Bobath's neurodevelopment treatment approach (NDT), strengthening of the agonist muscles, and antispastic

positioning using the key points. Antispastic positioning is to keep a muscle in a position for as long as it can be tolerated. Desensitization of stretch receptors occurs as a result of prolonged and slow stretching. To manage this, orthosis and splints are used to hold the joint in a desired position and to increase function.^{20,21}

Antispastic positioning is generally used before exercise to prepare the child or to prevent contractures, limitation and deformities and is recommended in a home exercise program. This prolonged technique depends on the autogenic inhibition mechanism.^{22,23}

Different methods are used to evaluate the effects of the treatment on spasticity. These include subjective methods such as passive goniometric measurement and clinical ratio scales, and objective methods such as electrophysiologic tests.24 The Hoffman (H) reflex represents the electrical analog of the muscle stretch reflex minus the involvement of the muscle spindle. The reflex is obtained by distal stimulation of Ia afferent fibers from muscle spindles, which in turn conduct into the spinal cord, initiating the monosynaptic stretch reflex and awakening a motor response in the ankle jerk reflex, recorded from the gastrocnemius and soleus muscles. As the intensity of the stimulus increases, the motor response (M) with 5 µs latency can be recorded during mixed muscle-action potential from muscles innervated by the nerve while providing maximum stimulation of the peripheral nerve. The ratio of maximal H response is used to assess the number of motor neurons activated by monosynaptic reflexes. The amplitude ratio of H maximum/M maximum (H/M) increase on patients with hemiplegia and spinal cord trauma, and this ratio is useful for assessment of the severity of spasticity.25,26

The aim of this study is to investigate the short-term effects of antispastic positioning on spasticity intensity by using H responses, H/M ratios, the Modified Ashworth Scale (MAS), and passive goniometric measurement values.

Methods

Subjects

The study included 20 subjects selected randomly from 68 children with spastic diplegic cerebral palsy who applied to the School of Physical Therapy and Rehabilitation, Paediatric Rehabilitation Unit, Hacettepe University, Ankara, Turkey, between January 2000 and February 2002.

The inclusion criteria to the study were: (i) spastic diplegic type of CP; (ii) no limitation at ankle; (iii) severity of spasticity of gastro-soleus muscle must be 3 according to the MAS; (iv) not taking any oral or intrathecal myorelaxant drugs; and (v) not to have had any orthopaedic surgery.

A total of 20 patients fulfilled these criteria but four of them were excluded because they could not complete the application method and the tests required. Therefore, 16 patients (11 males [68.75%] and five females [31.25%]), diagnosed as spastic diplegic were included in this study. The mean age of patients was 6.43 ± 1.99 years (range, 4–11 years).

Evaluations

All of these measurements were performed at the left lower extremity.

Goniometric measurement

Passive dorsiflexion of the foot was measured by a universal goniometer when patients were sitting and their knees were supported at extension. Lateral malleolus was the pivot point. Three measurements were performed and the mean of the three was recorded for analyses.²⁴

Modified Ashworth Scale

Muscle tone was assessed using MAS (0-5 point).27

For designing a single blind study, MAS and goniometric measurement were performed by a pediatric physiotherapist with 10 years experience who was blind about the study. The same physiotherapist performed the measurements before and after antispastic positioning for improving intrarater reliability.

Electrodiagnostic tests

For the objective measurement of severity of spasticity, H reflex and H/M ratios were used. The H reflex program of 'Medelec Synergy' equipment was chosen for the study. Patients were positioned in prone lying and the foot was supported at 110-120° of plantar flexion to maintain relaxation in the gastro-soleus muscles. The cathodal, of the Ag/AgCl surface (disk) electrodes of 9 mm diameter, was placed on the musculus soleus, and the anodal one to the Achilles tendon. Filter band pass was 20 Hz-10 kHz. Nervous Tibialis posterior was stimulated progressively from popliteal fossa with square-wave pulses of 1 µs duration by bipolar stimulating electrodes. The increase was stopped when maximum amplitude (M response) was achieved. The characteristic disappearance of the H reflex as current intensity is increased to elicit a maximal M response was confirmed. Maximum H reflex amplitude was divided by amplitude of the maximum M response to yield a H/M ratio before and after the antispastic position (Fig. 1).28 The same physician performed electromyography (EMG) tests before and after the treatment.

Before all evaluations, informed consent was taken from the patients' parents.



Fig. 1 Electromyography records.

Treatment procedure

Antispastic position

Patients were supported by a physiotherapist at a straight sitting position as hips were abducted at nearly a 45° angle and externally rotated, and the knees were extended to 90° of the ankles. Patients were kept at this position for 20 min

without changing the degree of support. The head was held in a neutral position in order to prevent asymmetrical tonic neck reflex.¹⁴ During the positioning, as the parents were near the children and applications performed by the childrens' physiotherapists, no child left the 20 min application time and children were encouraged by the use of toys.

All assessments were performed before and after antispastic positioning.

Statistical analysis

The Wilcoxon signed rank test was used in order to compare the changes in H responses, H/M ratios, MAS values, and goniometric measurement values before and after the treatment. Significance level was set at 0.05.

Results

Values of H response, H/M ratio, MAS and goniometric measurement are presented in Table 1.

The decrease in H responses, H/M ratios, MAS values and the increase in goniometric measurement values were found statistically significant after antispastic positioning (P < 0.05; Table 2).

Discussion

In our study, results of the objective and subjective assessment methods indicate that intensity of spasticity was reduced by antispastic positioning.

Odean and Knutsson reported that passive stretching is effective in adult patients having paraparesis and hyper reflex activity can be reduced with antispastic positioning in a lying or sitting position.²⁹

Richards *et al.* investigated the effects of stretching plantar flexor muscles in their study. Eight children with CP were included in the study group and they were kept standing for 30 min while their foot was in the dorsi-flexion position with the help of a tilt table. The other eight children formed the control group. Intensity of spasticity was assessed using spastic locomotor disorder index and an EMG test was used to evaluate heel strike as one of the walking activities. According to the results of the study, an improvement in walking activities has been found (P < 0.01) and spasticity of the gastro-soleus muscle has decreased.²²

In one of their studies, Kerem *et al.* searched the effects of Johnstone pressure splints in combination with Bobath's NDT approaches that include antispastic positioning. They have applied both pressure splints and Bobath's NDT approaches to a study group while a control group has received only Bobath's approaches. They have evaluated

Case no.	Age (year)	Before H/M (mV/mV)	After H/M (mV/mV)	Before MAS	After MAS	Before passive ROM	After passive ROM
1	9	0.76	0.47	3	3	95	100
2	5	0.65	0.62	3	3	92	100
3	10	0.32	0.12	3	3	96	100
4	6	0.29	0.15	3	2	90	99
5	7	0.56	0.05	3	2	100	105
6	5	0.59	0.03	3	3	93	100
7	7	0.67	0.55	3	3	93	101
8	5	0.05	0.01	3	3	91	98
9	11	0.40	0.15	3	2	90	104
10	6	0.45	0.46	3	3	98	106
11	4	0.53	0.24	3	3	90	106
12	5	0.42	0.50	3	3	93	108
13	5	0.49	0.37	3	2	100	115
14	6	0.65	0.14	3	2	92	100
15	5	0.93	0.68	3	2	96	112
16	7	0.58	0.01	3	3	92	100

Table 1 Clinical and electrodiagnostic test results of cases

H, Hoffman reflex response; M, M response; MAS, Modified Ashworth Scale; ROM, range of motion (°).

 Table 2
 Comparison of electromyography results, Modified Ashworth Scale values, and range of passive dorsi-flexion before and after antispastic positioning

Parameters	Pre-treatment Mean ± SD	Post-treatment Mean ± SD	Z	<i>P</i> -value
H response (mV)	5.17 ± 3.61	2.88 ± 2.98	-3.10	< 0.05
H/M ratio (mV/mV)	0.51 ± 0.20	0.28 ± 0.23	-3.25	< 0.05
MAS	3.00 ± 0.00	2.62 ± 0.50	-2.44	< 0.05
Range of passive dorsi-flexion (°)	94.12 ± 7.89	103.37 ± 5.77	-3.53	< 0.05

H, Hoffman reflex response; M, motor response; MAS, Modified Ashwort Scale; SD, standard deviation; Z, wilcoxon signed rank test.

motor development and used MAS and passive goniometric measurement for spasticity and somatosensorial evoked potential for sensorial input assessment. As a result, both treatment methods have been found effective in inhibition of spasticity and improvement of sensorial input (P < 0.05).¹⁴

In our study, after application of antispastic positioning for 20 min, spasticity of the gastrocnemius muscle decreased significantly according to the results of the H reflex response, H/M ratio and MAS and goniometric measurement values (P < 0.05).

Time of relaxation is due to the intensity of spasticity. and there is no knowledge about the duration of inhibition in the literature. The similarity in the spasticity intensity of cases made our study original and the temporary inhibition caused by antispastic position brings out the determination of inhibition time, but the difficulty of working with children makes this third assessment impossible.³⁰ The major limitation of our study is that we were not able to do a third assessment for measuring time of inhibition except after application of the assessment. The reason was that the children did not tolerate well the electrodiagnostic tests for measuring time of inhibition, except after the application of the assessment.

Evaluation of spasticity inhibition not only provides assessment of the effectiveness of the treatment but also helps physiotherapists in planning exercise programs. Determination of success of the treatments by sensitive assessment methods is important for making use of that treatment in the literature. Although MAS and goniometric measurement are used more frequently as it is simple to perform and are well tolerated by the children, electrophysiological methods are also used as they are objective.^{31–33} For this reason, we decided to use electro-diagnostic tests in our study.

H response and H/M ratio as electrodiagnostic test parameters were used in our study as they reflect the change of the alpha motor neuron. When literature was examined, the H response and H/M ratio were found higher in spastic subjects than in the controls.

Tardieu *et al.* examined the H/M ratio in 16 spastic and 12 non-spastic children, and found that the H response was hyper-excitable in 10 children with CP, which was the same

in five spastic children as with the non-spastic children, and was hyper-excitable in one child with CP.³⁴

Futagi *et al.* evaluated the H response in 53 spastic and 56 non-spastic children that have been categorized in subgroups of 0-12 months and 1-9 years. While amplitude of the H response was found higher in non-spastic children of 0-12 months than the 1-9 years group, it was higher in spastic children in both age groups than the non-spastic children.³⁵

In another study made by Futagi *et al.*, the H response was investigated in 40 spastic and 81 non-spastic children, of 0-12 months of age. They again found that amplitude of the H reflex was higher in children with CP.³⁶

As spastic children would have higher amplitude of H response spontaneously according to the related literature, our study did not include a control group. Decrease of the H response and H/M ratio confirmed the effects of our treatment statistically (P < 0.05).

Although electrodiagnostic tests are objective and a sensitive method, they cannot be used widely for the evaluation of spasticity in clinical practice. Therefore, we wanted to use clinical tests as a goniometric measurement and MAS in addition to electrodiagnostic tests in order to see if results of these methods were parallel to the objective ones.

In our study, as passive dorsi-flexion was $90-100^{\circ}$ before handling position, this value reached $98-115^{\circ}$ parallel to the relaxation of a spastic muscle.

The change that was observed in MAS values in our study was interesting because MAS was not a sensitive method in spasticity assessment and caused a conflict in the evaluation of minimal spasticity which can reflect gross changes.³⁷ When the data was examined, it can be seen that results of MAS values were parallel to that of electrodiagnostic tests.

According to the results of clinical and electrodiagnostic tests, it can be said that handling position is an effective method in the inhibition of spasticity. Strengthening of antagonist muscles according to antispastic positioning in addition to this temporary relaxation, will cause a cumulative effect on spasticity and an effective reciprocal inhibition, and relaxation can be achieved.

This outcome is not due to a plastic change in structure of muscle but a result of increased reflex activity. We think that this study will bring new ideas to the literature. But further studies about the effectiveness of antispastic position in different intensities of spasticity and different types of CP (hemiplegia, quadriplegia) by using objective evaluation methods are necessary.

References

1 Lance JW, Burke D. Mechanism of spasticity. Arch. Phys. Med. Rehabil. 1974; 55: 332–7.

- 2 Brunstrom JE. Clinical consideration in cerebral palsy and spasticity. J. Child Neurol. 2001; 16: 10–15.
- 3 Tomlin PI. The static encephalopathies. In: Newton RW (ed). *Color Atlas of Pediatric Neurolgy*. Times-Wolfe International, London, 1995; 203–16.
- 4 Lou HC. Cerebral palsy and hypoxic-hemodynamic brain lesions in the newborn. In: Coffey CE, Brumbock RA (eds). *Textbook of Pediatric Neuropsychiatry*, American Psychiatric Association, Washington, DC, 1998; 1073–92.
- 5 Sweeney KJ. Neonates of developmental risk. In: Unphred DA (ed). *Neurological Rehabilitation*. The CV Mosby Company, St. Luis, Toronto, 1985; 137–9.
- 6 Hagberg B, Hagberg G, Olow I, vonWenndt L. The changing panorama of cerebral palsy in Sweden. V. The birth year period 1979–82. Acta. Paediatrica Scand. 1989; 78: 283–90.
- 7 Blasko PA. Pathology of cerebral palsy. In: Sussman M (ed). *The Diplegic Children Evaluation and Management*. American Academy of Orthopedic Surgeons, Park Ridge, IL, 1992; 3–44.
- 8 Sala AD, Grant AD. Prognosis for ambulation in cerebral palsy. *Dev. Med. Child. Neurol.* 1995; **37**: 1020–9.
- 9 Davies PA, Tizard JPM. Very low birthweight and subsequent neurological deficit with special reference to spastic diplegia. *Dev. Med. Child. Neurol.* 1975; **17**: 3–17.
- 10 Wilson JM. Cerebral palsy. In: Campbell SK (ed). *Pediatric Neurologic Physical Therapy*. Churchill, Livingstone, Newyork, Edinburg, London, Tokyo, 1991; 301–46.
- 11 Hazlewood ME, Brown JK, Rowe PJ, Salter PM. The therapeutic use of electrical stimulation. *Dev. Med. Child. Neurol.* 1994; **36**: 661–73.
- 12 Nash J, Neilson PD, Dwyer NJ. Reducing spasticity to control muscle contracture of children with cerebral palsy. *Dev. Med. Child. Neurol.* 1989; **31**: 471–809.
- 13 Ricks NR, Eliert RE. Effects of inhibitory casts and orthoses on bony alignment of foot and ankle during weight-bearing in children with spasticity. *Dev. Med. Child. Neurol.* 1993; 36: 11–16.
- 14 Kerem M, Livanelioğlu A, Topçu M. Effects of Johnstone Pressure splints combined with neurodevelopmental therapy on spasticity and cutaneous inputs in spastic cerebral pasly. *Dev. Med. Child. Neurol.* 2001; 43: 307–13.
- 15 Cherry DB. Review of physical therapy alternatives for reducing muscle contracture. *Phys. Ther.* 1980; 6017: 877–81.
- 16 Bishop B. Vibratory stimulation part III possible application of vibration in treatment of motor dysfunction. *Phys. Ther.* 1975; 55: 139–42.
- 17 Krach LE. Pharmacotherapy of spasticity: oral medication and intrathecal baclofen. J. Child. Neurol. 2001; 16: 31–5.
- 18 Cosgrove AP, Carry IS, Graham HK. Botilinium toxin in the management of the lower limb in cerebral palsy. *Dev. Med. Child. Neurol.* 1994; 36: 386–96.
- 19 Rattey TE, Leahey L, Hyndman DCS. Recurrence after Achilles tendon lengthening in cerebral palsy. *J. Pediatr. Orthop.* 1993; **13**: 184–7.
- 20 Bobath K, Bobath B. The neuro-developmental treatment. In: Scrutton D (ed). *Management of Motor Disorders of Children* with Cerebral Palsy. JB Lippincot, Philadelphia, 1984; 8–70.
- 21 Binder H, Eng GD. Rehabilitation management of children with cerebral palsy. *Arch. Phys. Med. Rehabil.* 1989; **70**: 482–9.
- 22 Richards CL, Maloun F, Dumas F. Effects of a single session of prolonged plantarflexor stretch on muscle activations during gait in spastic cerebral palsy. *Scand. J. Rehabil. Med.* 1991; **23**: 103–11.

- 23 Schreihber JM, Effgen SK, Palisino RJ. Effectiveness of parental collaboration on compliance with a home program. *Pediatr. Phys. Ther.* 1995; **7**: 59–64.
- 24 Moore ML. Clinical assessment of joint motion. In: Basmajian JV (ed). *Therapeutic Exercise*. Baltimore, London, 1978; 151–90.
- 25 Misiaszek JE, Pearson KG. Strech of Quadriceps Inhibits the Soleus H reflex During Locomotion in Decerebrate Cats. J. Neurophysiol. 1997; 78: 2975–84.
- 26 Katz RT, Rymer WZ. Soastic Hypertonia: mechanisms and meauserement. Arch. Phys. Rehabil. 1989; 70: 144–55.
- 27 Bohannon RW, Smith MB. Intrarater reliability of a modified asworth scale of muscle spasticity. *Phys. Ther.* 1987; **67**: 206–7.
- 28 Morelli M, Sullivan J, Seaborne DE. Comparison of human triceps surae H-reflexes obtained from mid and distal recording sites. *Electromyogr. Clin. Neurophysiol.* 1990; **30**: 181–6.
- 29 Odeon I, Knutsson E. Evaluation of the effects of muscle stretch and weight load in patients with spastic paraplegia. *Scand. J. Rehabil. Med.* 1981; **13**: 117.
- 30 Bodire-Fowler SC, Botte MJ. Muscle spasticity. In: Vernan LN (ed). Orthopedic Rehabilitation, 2nd edn. Churchill Livingstone, London, 1992; 95–308.
- 31 Malouin FM, Boiteau M, Bonneau C, Pichards L, Bravo G. Use of a handheld dynamometer for the evaluation of spasticity

in a clinical setting: a reliability study. *Physiotherapy Can.* 1989; **41**: 126–34.

- 32 Stuberg WA, Fuch RH, Miedaner JA. Reliability of goniometric measurements of children with cerebral palsy. *Dev. Med. Child. Neurol.* 1988; **30**: 657–66.
- 33 Allison SC, Abraham LD, Peterson CL. Reliability of the Modified Asworth Scale in the assessment of plantarflexor muscle spasticity in patients with traumatic brain injury. *Int. J. Rehabil. Res.* 1996; **19**: 67–78.
- 34 Tardieu C, Lacert P, Lambard M, Truscelli D, Thardeeu G. H-reflex and recovery cycle in spastic and normal children: intra and inter individual and inter groups comparisons. *Arch. Phys. Med. Rehabil.* 1997; **58**: 561–7.
- 35 Futagi Y, Abe J. H-reflex study in normal children and patients with cerebral palsy. *Brain Dev.* 1985; 7: 414–20.
- 36 Futagi Y, Abe J, Tanaka J, Okamoto N, Ikoma H. Recovery curve of the H-reflex in normal infants, central co-ordination disturbance cases and cerebral palsy patients within the first year of life. *Brain Dev.* 1988; **10**: 8–12.
- 37 Brar SP, Smith MB, Nelson LM, Franklin GM, Cobble ND. Evaluation of treatment protocols on minimal to moderate spasticity in multiple sclerosis. *Arch. Phys. Med. Rehabil.* 1991; **72**: 186–9.