

Impact of Seasonal Variations on Spasticity Assessment and Treatment

Martha Miller, Lisa Martin, Farooq Ismail, Chris Boulias, Lisa Lyons, Chetan P Phadke

Martha Miller, Lisa Martin, Farooq Ismail, Chris Boulias, Chetan P. Phadke, Faculty of Medicine, University of Toronto, Toronto, Canada

Farooq Ismail, Chris Boulias, Lisa Lyons, Chetan P. Phadke, West Park Healthcare Centre, Toronto, Canada

Chetan P. Phadke, Faculty of Health, York University, Toronto, Canada

Correspondence to: Chetan P. Phadke, Spasticity Research Program, West Park Healthcare Centre, 82 Buttonwood Ave., Toronto, Canada

Email: chetan.phadke@westpark.org

Telephone: +1-416-243-3600

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Seasonal temperature variations appear to have no effect on spasticity or botulinum toxin dosage. Performance of spasticity measurements in warm indoor environment may have negated the impact of seasonal temperature differences. Future studies should perform spasticity measurements in temperature controlled rooms to mimic seasonal variations.

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Key words: Muscle spasticity, Botulinum toxin, Stroke, Multiple sclerosis, Cerebral palsy, Cold temperature, Hot temperature

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ABSTRACT

BACKGROUND: Spasticity is treated using intramuscular botulinum toxin injections. Previous reports of worsening of spasticity in cold weather suggest that spasticity levels and botulinum toxin dose may both be influenced by seasonal temperature changes.

AIMI: The aim of this study was to determine the effect of seasonal temperature variation on spasticity and botulinum toxin dosage.

METHODS: Medical charts of 132 participants in 3 diagnostic groups: (1) stroke ($n=60$); (2) multiple sclerosis ($n=34$); and (3) cerebral palsy ($n=38$) receiving treatment in an outpatient spasticity clinic were reviewed. Age, sex, diagnosis, number of muscles injected, botulinum toxin dose, and spasticity (Modified Ashworth Scale) were collected for summer and winter. Spasticity and botulinum toxin dose were compared between summer and winter using separate paired t-tests for each diagnostic group.

RESULTS: The spasticity level, botulinum toxin dosage, and total number of muscles in summer versus winter months were not significantly different ($p>0.05$) in any diagnostic group. Conclusions:

INTRODUCTION

Spasticity is a velocity-dependent increase in muscle tone, and is associated with a variety of upper motor neuron lesions^[1]. Although the exact physiological mechanisms of spasticity are not fully understood, it is thought to be caused by hyperexcitability of the stretch reflex, which occurs after an upper motor neuron lesion^[2]. Spasticity can result in reduced range of motion, pain, stiffness, and loss of function, all of which may decrease one's ability to participate in activities of daily living^[3]. Spasticity is commonly seen in a number of diagnostic groups such as cerebral palsy (CP), multiple sclerosis (MS), stroke, brain injuries, and spinal cord injuries^[2].

A diverse range of internal and environmental factors can contribute to an increase in spasticity^[4]. One such factor known to worsen spasticity is cold, outdoor (weather) and indoor (air-conditioning), which is especially relevant in geographic areas of the globe with extreme seasonal temperature variation^[5]. Much of

our current understanding on the effects of seasonal temperature variations on spasticity are the result of anecdotal reports, with little objective evidence^[4,6]. While these anecdotal reports provide the basis of our understanding of the relationship between temperature and spasticity, they are biased because of self-reporting, which by nature is subjective and open to interpretation.

To date, there is little research that objectively examines the relationship between temperature and spasticity. A study by Nilsagard *et al* (2006) examined the effect of a cooling vest on spasticity in heat-sensitive patients with MS, and found no statistically significant difference^[7]. However, the use of a cooling vest is problematic because individuals typically experience spasticity in their extremities, which would be unaffected by the vest. A study by Chiara *et al* (1998) found that immersion in a cold bath increased spasticity in participants with MS^[8]; however, the bath water was 24 degrees Celsius, and, therefore, not comparable to the cold temperatures reflected in other studies. The above studies lack ecological validity since use of cooling vests and cold baths to simulate the effects of a cold environment might vary from what a person experiences in the real world where ambient temperatures can fall down to and below freezing.

Spasticity can affect both upper and lower limbs and one of the available treatment is localized intramuscular injections of botulinum toxin (BoNTA)^[3]. From a physiological perspective, BoNTA works to relieve spasticity by inhibiting the release of acetylcholine at the neuromuscular junction, thereby producing paralytic effects on the injected muscle to decrease tone and to allow stretching of the muscle fibers^[3]. Treatment effects of BoNTA are not permanent, and, therefore, injections are typically required every 3-4 months^[3]. As such, patients will often receive injections in both winter and summer months. Typically, higher doses of BoNTA are associated with higher levels of spasticity. Thus, if spasticity levels are higher in winter, it is possible for the BoNTA dosage to be higher in winter as well.

Given the lack of objective data, there is a gap in the literature regarding the relationship between temperature, spasticity, and BoNTA dosage. There is a need to systematically examine the relationship between spasticity levels and related BoNTA dosage in winter versus summer months. Thus, this study carried out a retrospective chart review to objectively compare the differences in spasticity levels, as measured by the Modified Ashworth Scale (MAS), and related BoNTA dosage in winter versus summer months. The objective of this retrospective study was to compare the differences in spasticity levels (MAS) and BoNTA dosage in winter versus summer months.

METHODS

This was a quantitative, single site, retrospective chart review. In compliance with Helsinki Declaration, ethical approval for this study was provided by the West Park Healthcare Centre and University of Toronto Research Ethics Boards. Since only patient past health records were accessed and no patients directly observed in this study, the ethics board did not require us to obtain informed consent from participants. Data was obtained from the participants' health records to objectively compare spasticity level and BoNTA dosage in winter versus summer months. Because spasticity is most commonly associated with stroke, MS, and CP^[1], participants were selected from these three diagnostic groups. Non-random, purposive sampling was used to obtain 132 medical charts from adult patients age 18 and over with stroke ($n=60$), MS ($n=34$), or CP ($n=38$) who were receiving, or had previously received, treatment with BoNTA from an outpatient spasticity clinic. This was a convenience sample since it was only

feasible for students to collect data from up to 132 patient medical charts.

The toxin dose may be varied by physicians within the first three treatment cycles to determine the optimal dosage of BoNTA required to best manage a client's spasticity. Thus, participants were excluded from the study if they had received three or fewer treatments of BoNTA injections. All patients were treated with two physicians with experience >10 years of working together in spasticity management and BoNTA injections and similar practice patterns. Participants were also excluded if they had not received BoNTA injections in consecutive summer and winter months. Summer months were considered June 21 to September 21 (\pm two weeks), and winter months were considered December 21 to March 21 (\pm two weeks). Maximum and minimum temperature ($^{\circ}$ Celsius) data on the day of spasticity assessments was gathered using climate data from Government of Canada^[9] and The Weather Network^[10]. All medical data was collected from participants' electronic and paper medical records at an outpatient spasticity clinic in Toronto, Canada. The following pieces of data were collected from participant charts: total BoNTA dosage, injection location, number of injections, MAS scores, age, sex, and body weight.

Modified Ashworth Scale (MAS)

The MAS is an assessment of the clinician's perception of resistance after moving the affected joint through passive range of motion^[11]. Craven and Morris (2010) found the intra-rater reliability for the MAS to be fair-to-perfect ($0.20 < k < 1.0$), and inter-rater reliability was found to be poor-to-moderate ($k < 0.60$)^[12-14]. Although measures of reliability are low, there is evidence to suggest that a change in MAS scores indicates a change in muscle spindle excitability^[15] and level of excitation at the spinal cord neurons^[16]. MAS remains the primary choice for clinicians to measure spasticity. MAS in this study was performed by clinicians with experience in spasticity assessment of >5 years. All spasticity assessments were performed by the same two clinicians (upper limb – occupational therapists and lower limb – physiotherapist) in supine position.

Data Analysis

Since MAS is an ordinal scale, we chose to use non-parametric tests (Wilcoxon signed rank test) to make statistical comparisons for data in summer versus winter months ($p < 0.05$). Rather than assessing spasticity levels for each individual muscle, specific muscle groups were chosen; namely, wrist flexors, elbow flexors, and ankle plantar flexors, as these groups have been found to be most commonly treated for spasticity^[1]. The MAS scores for each muscle group, the sum of BoNTA dosage, and the total number of muscles injected were compared in summer versus winter months using paired t-tests. The above analyses were carried out for the whole group, as well as separately for each of the three diagnostic groups using IBM SPSS software version 22 (IBM Corporation, New York). Graphs were prepared using a Microsoft Excel add-on (XL Toolbox[®] 2008-2011 Daniel Kraus, Würzburg, Germany).

RESULTS

A total of 132 participants were recruited with a diagnosis of either stroke ($n=60$), MS ($n=34$), or CP ($n=38$). The age range of participants was 19-88 years (Mean=55.6, SD=17.1). The male:female ratio was 64:68 (total participant group), 22:16 (stroke), 10:24 (MS), and 32:28 (CP) in our participants. For other demographic data, see table 1. Average total number of muscles

injected was 6 muscles in summer and 6 muscles in winter. Average summer temperatures (° Celsius) on the day of testing were 26±6°C (maximum) and 16±4°C (minimum) and average winter temperatures were -2 ±6°C (maximum) and -7 ±6°C (minimum).

MAS score and BoNTA in summer versus winter

Spasticity levels and BoNTA dosage in summer versus winter months were not significantly different ($p>0.05$) for any of the diagnostic groups, as well as for the group as a whole (Figures 1 and 2). There was no significant difference between total number of muscles injected in summer versus winter for any of the diagnostic groups, as well as for the group as a whole ($p>0.05$).

Not all muscles were injected in all patients; however, data from individual muscles did not show a significant change in either BoNTA dosage (Table 2) or spasticity measured using MAS (Table 3) between summer and winter. Only in the gastrocnemius muscle in the MS group 37 units more were injected in winter vs. summer; however this number is unlikely to be significant because of high standard deviation (100 units; see Table 2). Finger flexors and soleus muscle in the stroke group showed a slightly higher median MAS score in winter (MAS=2) compared to summer (MAS=1.5 or 1+;

$p>0.05$); see Table 3. Similarly, the hip adductors in the MS group showed higher median MAS in winter than summer.

DISCUSSION

In this study, seasonal temperature variations although significant, appear to have no effect on spasticity level, BoNTA dosage, or total number of muscles injected. These findings are inconsistent with previous research^[5], demonstrating that patients perceive that cold weather worsens their spasticity. A study by Shirado *et al* (1995) investigated outdoor winter activities of spinal cord-injured patients, and found that 17% of participants who were able to go outside in the winter reported experiencing increased spasticity in their upper and lower extremities^[6]. Additionally, all of these participants believed that their spasticity worsened with direct exposure to cold weather^[6]. A similar study conducted by Fleuren *et al* (2009), which also explored the perceptions of spasticity in patients with spinal cord injury, found that 58% of participants identified other factors that contributed to spasticity, which were not listed on the questionnaire provided^[17]. Temperature/climate (both hot and cold) was among the other factors that were listed.

Table 1 Demographic characteristics.

	Stroke	MS	CP	All Participants
Number of Participants	60	34	38	132
Age range	27-88	28-79	19-69	19-88
Mean age	66.6	56.88	37.05	55.59
% Female	46.67%	70.59%	42.11%	51.52%
% Male	53.33%	29.41%	57.89%	48.48%
Average body weight - Summer (Kg)	76.3	64.78	67.02	71.29
Average body weight - Winter (Kg)	79.76	71.42	59.42	71.48
Average total # muscles injected - Summer	6.36	5.85	5.95	6.11
Average total # muscles injected - Winter	6.43	5.72	5.66	6.03

Table 2 Average BoNTA dose in summer vs winter.

Patient type	Muscle Injected >10 patients	# of patients	BoNTA dose (units)		Difference (W-S)
			S	W	
Stroke	PM	23	92 ±22	93 ±21	1
	BB	12	50 ±19	52 ±23	2
	B	32	51 ±13	53 ±11	2
	BR	31	49 ±14	52 ±14	3
	PT	27	54 ±18	53 ±16	-1
	L	12	50 ±00	50 ±00	0
	FDS	39	51 ±10	54 ±11	3
	FDP	22	49 ±12	50 ±13	1
	FPL	15	25 ±00	27 ± 07	2
	FCR	21	51 ±19	54 ±18	3
	FCU	18	43 ±14	45 ±14	2
	G	29	95 ±38	94 ±38	-1
	S	21	62 ±20	63 ±23	1
	TP	20	79 ±25	79 ±22	0
MS	FDL	12	52 ±23	50 ±20	-2
	AM	14	109 ±36	107 ±33	-2
	AL	13	113 ±38	104 ±38	-9
CP	G	11	91 ±30	128 ±100	37
	FCU	10	39 ±13	42 ±12	3
	G	14	75 ±26	77 ±22	2

W: Winter; S: Summer; PM: Pectoralis Major; BB: Biceps Brachii; B: Brachialis; BR: Brachioradialis; PT: Pronator Teres; L: Lumbricals; FDS: Flexor Digitorum Superficialis; FDP: Flexor Digitorum Profundus; FPL: Flexor Pollicis Longus; FCR: Flexor Carpi Radialis; FCU: Flexor Carpi Ulnaris; G: Gastrocnemius; S: Soleus; TP: Tibialis Posterior; FDL: Flexor Digitorum Longus; AM: Adductor Magnus; AL: Adductor Longus; G: Gastrocnemius.

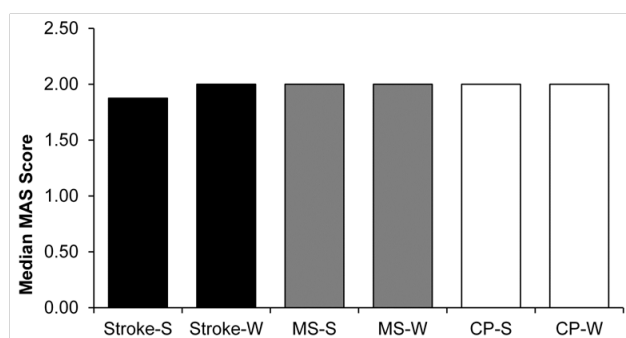


Figure 1 Median MAS score in winter vs summer. S: summer; W: winter; MS: multiple sclerosis; CP: cerebral palsy.

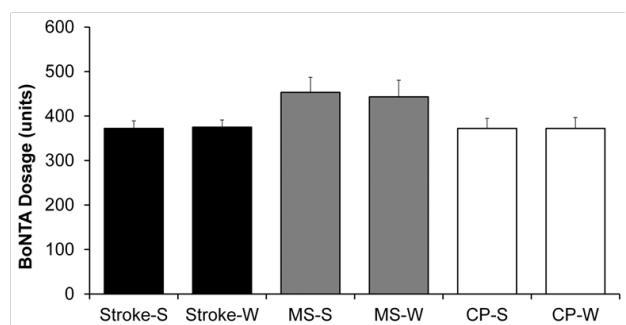


Figure 2 Average BoNTA dosage in summer vs winter. Standard error bars are shown.

Table 3 Median MAS score in muscle groups tested in winter and Summer.

Patient type	Muscle group Injected >10 patients	# of patients	Median MAS (max 4)		Difference (W-S)
			S	W	
Stroke	Shoulder Adductors	19	2	2	0
	Elbow Flexors	36	2	2	0
	Pronators	22	2	2	0
	Wrist Flexors	12	2	2	0
	Finger Flexors	33	1.5	2	0.5
	G	23	2	2	0
	S	11	1.5	2	0.5
MS	Hip Adductors	10	2	2.3	0.3
	Knee Extensors	10	3	3	0
CP	G	12	2	2	0
	G	10	3	2	-1

W: Winter; S: Summer; G: Gastrocnemius; S: Soleus.

Although the exact mechanism is unclear, there are several potential pathways that can explain the worsening of spasticity in cold weather. One potential mechanism is the hypersensitivity of the neurons in the spinal cord in patients with spasticity as seen by testing the stretch reflexes. It is possible that on top of this reflex hypersensitivity, cold exposure may further amplify the shivering response designed to regulate body temperature^[18,19]. It is also possible that blood flow to the extremities when exposed to cold is redirected to the body core in order to preserve heat and minimize heat loss inadvertently amplifying the effect of cold exposure^[19].

A potential explanation as to why there was no significant change in MAS scores in winter versus summer could be due to the fact that participants were seated in an indoor waiting room prior to administering the MAS. Because participants were seated in a temperature controlled room (heated in the winter and air-conditioned in the summer), the temperatures they experienced were not necessarily reflective of outdoor seasonal temperatures. Even if there was an effect of temperature on spasticity, the effect may have dissipated by the time patients were tested in the temperature controlled testing environment. There is also some research data to suggest that a cold breeze or air-conditioned environments can increase spasticity^[20,21]. As such, even indoor temperatures, if sufficiently cold enough, could potentially worsen spasticity. Future studies should perform spasticity measurements in temperature controlled rooms (cold and hot) to mimic seasonal outdoor temperature variations more accurately.

There are several factors that may explain the lack of change in BoNTA dosage between summer and winter months. It is a complex process for physicians to determine the most appropriate BoNTA dosage for an individual. Once a stable dose has been achieved (usually after 2-3 injection cycles) physicians typically do not vary doses from season to season. One explanation for this is that there are multiple means of managing spasticity, including oral anti-spasticity medications, as well as daily stretching exercises, which can both be used to manage fluctuations in spasticity levels without altering the BoNTA dosage (Graham, 2013). We did not record the dose of oral anti-spasticity medications in summer vs. winter. In addition, an increase in spasticity – in particular with respect to the lower extremities – may allow for improved function in certain cases where the increased tone can assist with ambulation or transfers. In these cases the goal may not be to reduce spasticity because it is serving a functional purpose.

The use of the MAS as a measure of spasticity could be considered a limitation of this study. Despite being a commonly used clinical measure of spasticity, there remains a high degree of subjectivity to the scale^[3]. Although lengthier and more complex to administer, the Tardieu Scale is considered a more objective measure of spasticity^[3] and could be used in future studies to increase the reliability and validity of results. There are a variety of environmental factors thought to increase levels of spasticity; however, this study focused specifically on the effect of seasonal variations in temperature on spasticity levels and related BoNTA dosage. There are a number of variables that were not accounted for in this study that may affect spasticity, such as fatigue, stress, illness, and injury, among many others^[4]. We did not include other data such as physiotherapy and occupational therapy interventions in this chart review that may have potentially affected the outcome measures. Future prospective studies should consider a research design that would allow for more rigorous control of confounding variables.

CONCLUSIONS

Seasonal temperature variations were found to have no effect on either spasticity level, as measured by the MAS, or BoNTA dosage. Future studies could consider using a temperature controlled room to simulate outdoor temperature, examine the use of additional spasticity management methods in conjunction with BoNTA dosage, and use a more objective spasticity measure.

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CONFLICT OF INTERESTS

The Author have no conflicts of interest to declare.

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- Peer reviewer:** Fang Li, MD, PhD, Department of Rehabilitation Medicine, Huashan Hospital, Fudan University, Shanghai, 200040, China.