The Temporal Relationship between Stress and Spasticity, a Comparison between Subjects with and without Intact Thyroid Function

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Spasticity is defined as a velocity dependent increase in skeletal muscle resistance to passive stretch. While spasticity severity is highly variable, it often leads to significant losses of functional movement which deteriorate quality of life, impede activities of daily living, and restrict life participation. Recent studies have suggested that stress may be a factor in spasticity severity. Multiple studies into stress modulation of neuropathic pain have revealed a ten-day delay between the occurrence of significant stressors and latent flares in pain intensity. Current evidence indicates this is due to delayed effects of both stress-induced thyroxine (T4) on central sensitization of pain pathways & increased peripheral nerve excitability. Spasticity involves activation of both IA muscle spindle input and alpha motor neuron output, the expression of both may be modulated by this same stress hormone. It is plausible that severity of spasticity may also show demonstrable decreases ten days after significant stressors. Anticipating and controlling fluctuations in spasticity is important not only in ameliorating the frustrations caused by spasticity, but is also necessary to maximize the benefits of physical therapy.

Purpose

This study’s purpose is to begin exploring the temporal relationship between stress & spasticity, to see if some patients experience latent episodic increases in spasticity due to stress.

Case Description

Every day for 12 weeks, two subjects completed stress and spasticity inventories. Subject 1 is an 84 year-old-male with history of L CVA in 2014. Subject 2 is a 65 year-old-female with history of L CVA in 2015. While our exclusion criteria precluded subject 2 due to thyroidectomy, this candidate was allowed to participate in order to determine inverse support to our hypothesis. Stress was measured with a visual analog scale stress (VAS), with low stress to left and elevated stress to right. VASS has shown high reliability to measure stress with r = +0.91 when concurrently compared to Daily Stress Inventory. Spasticity was measured via visual analog functional scale (VAFS), with low spasticity to left and elevated spasticity related function ten days after peak spasticity. VASS has shown high reliability to measure spasticity with r = +0.91 when concurrently compared to Daily Stress Inventory. Spasticity was measured via visual analog functional scale (VAFS), with low spasticity to left and elevated spasticity related function ten days after peak spasticity. VASS has shown high reliability to measure spasticity with r = +0.91 when concurrently compared to Daily Stress Inventory.

Discussion

Patient 1 supported the 10-day lag hypothesis with 70% correlation. However, if you include the days surrounding Day 10 (days 9 and 11) for error, he supports the hypothesis with 90% correlation. The fact that our study only included analysis at Day 10 was a significant limitation. Another limitation was not being able to account for overlap in a serial lag analysis.

Conclusion

Based on results, evidence supports an increase in CVA-related spasticity ten days after high stress episodes. This relationship was not observed in our female subject s/p thyroidectomy, lending inverse support to the hypothesis that latent ten-day effects may be due to the HPT axis.