Galvanic vestibular stimulation reveals disruption of ipsilesional brainstem pathways in hemiparetic stroke survivors

Angelo Bartsch-Jimenez [1,3]**,Hesam Azadjou**[2]**, and Francisco J Valero-Cuevas** [1*,2]

[1] *Division of Biokinesiology and Physical Therapy, University of Southern California, Los Angeles, California, USA*
[2] *Biomedical Engineering Department, University of Southern California, Los Angeles, California, USA*
[3] *Escuela de Kinesiología, Facultad de Medicina, Universidad de Valparaíso, Valparaíso, Chile*

Correspondence*:
F.J. Valero-Cuevas
valero@usc.edu

Background: The spatiotemporal structure of muscle coordination emerges from the interaction of descending cortical, spinal, vestibular, and brainstem pathways (Bartsch and Valero-Cuevas, 2025). Thus, disruptions in brainstem drive following stroke is thought to contribute to pathological synergies (Krakauer, 2005; Li et al., 2019).

Purpose: While the imbalance of cortico- and reticulo-spinal tracts is thought to contribute to pathological synergies, this study aims to exclude the contribution from vestibular motor output —as measured by IMC— following stroke.

Methods: As in Bartsch and Valero-Cuevas (2025), we used galvanic vestibular stimulation (GVS) to investigate the extent to which vestibular input to the brainstem may be disrupted in stroke survivors (n=14) with right hemiparesis, as compared with age-matched controls (n=14). We tested both arms under three tasks: rest, voluntary reaching movement with neutral and abducted shoulder positions while participants were subjected to three stimulus types: Sham, GVS, and No stimulation. sEMG was recorded from: Biceps (Bic) and Triceps Brachii (Tri), Anterior (ADelt), Middle (MDelt) and Posterior (PDelt) Heads of Deltoid, and Upper Trapezius (UTrap); and one nec kmuscle, Sternocleidomastoid (SCM), as a control for GVS. Pairwise magnitude-squared coherence from EMG was computed in the 8- 50Hz frequency range.

Results: We find that the effect of GVS to neck and arm muscles in the paretic side is similar to that in neurotypical individuals, both at rest and during voluntary reaching movements. However, and to our surprise, GVS is greatly suppressed in neck muscles on the non-paretic side —suggesting strong disruptions in ipsilesional brainstem pathways following stroke. Importantly, since stroke-related neural damage occurs at the upper motor neuron level, GVS is likely acting through a presynaptic mechanism on upper rather than intact lower motor neurons. While these disruptions may not affect arm movement, they may have important consequences to balance control in stroke.

Justification Statement: The emergence of pathological synergies is thought to result from a combination of damage to the corticospinal tract (CST) and increased drive from subcortical pathways, such as the

reticulospinal tract (RST) and vestibular motor output. While the role of the RST in pathological synergies has been relatively well-studied, the contribution of the vestibular output remains poorly understood. The vestibular motor output is known for its critical role in modulating motor neuron excitability, muscle tone, and postural control. Moreover, its upregulation after stroke has been linked to increased spasticity and maladaptive reflex responses. However, its specific contribution to pathological synergies during voluntary reaching movements has not been systematically addressed nor excluded. Current approaches to studying pathological synergies rely on methods —such as dimensionality reduction techniques for synergy extraction— that describe patterns of muscle co-activation without providing insights into their neural origin. Intermuscular coherence (IMC), on the other hand, offers a powerful tool for assessing shared neural drive to muscles, allowing researchers to identify synchronized neural drive across different frequency bands. Specifically, alpha-band (8–16 Hz) coherence is associated with subcortical drive (e.g., RST and vestibular motor output), while beta-band (16–30 Hz) coherence reflects cortical drive via the CST and vestibular output. This study aims to exclude the contribution of vestibular motor output to pathological synergies by measuring shared neural drive to muscles (as measured by intermuscular coherence) during voluntary arm movement. We hypothesize that intermuscular coherence increases during shoulder abduction, but it does not during vestibular stimulation. This selective increase would exclude a common vestibular mechanism underlying both changes.

1 KEYWORDS:

stroke, pathological synergies, vestibulospinal, galvanic vestibular stimulation