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An Etiological Paradigm Shift for Chronic Hemiplegic Shoulder Pain

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We appreciate the interest in the recently published manuscript, “Peripheral Nerve Stimulation Compared with Usual Care for Pain Relief of Hemiplegic Shoulder Pain.”¹ This study provides evidence that peripheral nerve stimulation (PNS) is an efficacious treatment for hemiplegic shoulder pain (HSP) and reduces pain to a greater extent and with longer durability than usual care. It has been correctly pointed out that there was no attempt by the study authors to provide a diagnosis for the occurrence of HSP based on the nociceptive and biomechanical models. This was not an oversight, rather is due to an alternative paradigm of thought regarding the etiology of chronic HSP considered to be distinct from the etiology of acute HSP. That is, HSP may initially be the result of one of the 31 or more of the direct causes of HSP, but maladaptive changes in the central nervous system are responsible for the pain persisting beyond the initial injury. It is apparent that identifiable pathologies on imaging studies have low correlation to the presence of HSP.^{2–4} Furthermore, there is a growing body of evidence to suggest that the nociceptive and biomechanical models should be reconsidered in favor of a central sensitization process in the etiology of chronic HSP.^{5–8}

The assertion that the study results are not valid due to heterogeneity of the cohorts deserves reexamination. The design of this study included randomized treatment allocation so that treatment status would not be confounded by either measured or unmeasured baseline characteristics.⁹ A review of baseline demographics (Table 1) indicates that the randomization procedure performed as expected to create well-balanced cohorts, even with the small sample sizes in this study.¹ There is no indication of lack of balance, and this is supported by the acknowledgement in the letter that glenohumeral subluxation occurs in similar proportions in both of the cohorts. Thus, the results should be considered valid and not due to chance.

Finally, the study authors embrace the need for exploration of the mechanism of action of PNS. At this point, there is no evidence to suggest the way in which PNS achieves pain relief or which subjects will receive benefit from treatment. When considering mechanism

of action, all mechanisms should be explored- including somatosensory function, anatomical pathology, biomechanics, genetic predisposition, psychological traits, among others.

Sincerely,

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Table 1
Demographics

	PNS n=13	Usual Care n=12
Female (%)	46.2	58.3
Age (years, median +/- IQ range)	54.0 (50.0 – 68.0)	55.5 (50.0 – 62.5)
Subluxation (%)	61.5	66.7
Race/Ethnicity (%)		
White	46.1	41.7
African American	53.9	50.0
Hispanic/Latino	0	8.3
Shoulder Pain > 18 mo. (&)	61.5	58.3
Time Since Stroke (years, median +/- IQ range)	2.6 (0.9 – 4.0)	2.6 (0.9 – 4.0)
Cortical Stroke (&)	66.7	54.5
Hemorrhagic Stroke (%)	38.5	8.3
Comorbidities at Baseline (&)		
Coronary Artery Disease	30.8	0
Congestive Heart Failure	0	0
Cardiac Arrhythmia	7.7	0
Diabetes Mellitus	38.5	41.7
Hypertension	76.9	100
Renal Dialysis	0	0
Pulmonary Disease	7.7	16.7
Peripheral Vascular Disease	0	8.3
Seizure Disorder	7.7	0
Osteoarthritis	23.1	0
Cancer	0	8.3
Analgesic Use at Baseline (%)		
Narcotic	7.7	8.3
Non-Narcotic	23.1	25.0
Aspirin	54.0	41.7
NSAID	NSAID	16.6
Anti-Epileptic	30.8	50.0

Demographics for PNS and Usual Care groups with chronic hemiplegic shoulder pain.

Abbreviations: PNS- peripheral nerve stimulation; IQ- interquartile; NSAID- Non-steroidal anti-inflammatory drug