

Effects of Autonomic Modulation on Myofascial Pain and Heart Rate Variability in Post-stroke Individuals

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Abstract

Stroke is a leading cause of chronic disability, frequently accompanied by Myofascial Pain Syndrome (MPS) and Autonomic Nervous System (ANS) dysregulation. Autonomic modulation, including slow diaphragmatic breathing, Heart Rate Variability (HRV) biofeedback and mindfulness-based relaxation, offers a non-pharmacological approach targeting both musculoskeletal and autonomic impairments simultaneously. A randomized controlled trial was conducted on 40 chronic post-stroke individuals (≥ 6 months post-stroke), randomly allocated into an Autonomic Modulation Group (AMG, $n = 20$) and a Control Group (CG, $n = 20$) receiving conventional physiotherapy. Both groups underwent 4 weeks of intervention (35 minutes per session, 5 days per week). Primary outcomes included pain intensity [Visual Analogue Scale [VAS]], pressure pain threshold (PPT via algometry) and HRV indices (root mean square of successive differences [RMSSD], high-frequency power [HF] and LF/HF ratio), measured at baseline and after 4 weeks. The AMG demonstrated significantly greater improvements in VAS (mean change: 3.3 ± 0.8 vs. 1.2 ± 0.6 ; $P < 0.001$), PPT (0.7 ± 0.3 vs. 0.2 ± 0.2 kg/cm²; $P < 0.001$), RMSSD (11.4 ± 4.0 vs. 1.5 ± 2.5 ms; $P < 0.001$), HF power (130 ± 40 vs. 15 ± 30 ms²; $P < 0.001$) and a greater reduction in LF/HF ratio (1.2 ± 0.4 vs. 0.3 ± 0.3 ; $P < 0.001$) compared to the CG. Autonomic modulation significantly reduces myofascial pain and restores autonomic balance in post-stroke individuals, supporting its integration as an adjunct in neurorehabilitation.

Keywords: Autonomic Modulation, Heart Rate Variability, Stroke Rehabilitation, Neurorehabilitation, Physiotherapy, Health Risk.

Introduction

Stroke remains a leading cause of long-term disability worldwide, producing persistent motor, sensory and musculoskeletal sequelae that substantially compromise functional recovery (1, 2). Among these complications, myofascial pain syndrome (MPS) is highly prevalent in the post-stroke population, predominantly affecting the cervical, shoulder and upper back musculature. The pathophysiology is multifactorial, involving aberrant motor recruitment, spasticity, postural asymmetry and compensatory overactivation of unaffected musculature (3). Myofascial trigger points arising from these mechanisms produce localized and referred pain, restrict range of motion and further impede rehabilitation outcomes (3, 4). The burden of this complication is considerable: a multicentre prospective study of 443 stroke survivors reported an overall post-stroke pain prevalence of 29.6%, rising to 42.7% in the subacute phase and explicitly characterised this pain as largely undertreated (5). Cross-

sectional evaluation using standardized palpation criteria has confirmed a very high burden of myofascial trigger points in the post-stroke shoulder girdle, with latent trigger points identified in up to 92% of cases at the infraspinatus and active trigger points in half of cases, trigger-point density being moderately correlated with pain severity (6). Interventional evidence remains limited to small, short-duration studies; for example, trigger-point lidocaine blockade produced pain relief at one and three weeks in a cohort of only 21 patients, with benefit not sustained by four months, illustrating both the plausibility of a myofascial contribution and the paucity of adequately powered, longer-term trials (7). Reviews of post-stroke pain have further noted that its true prevalence remains difficult to establish because of inconsistent diagnostic criteria across studies and have called explicitly for future prospective research using standardized case definitions (8), while broader appraisals of

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post-stroke pain management confirm that it continues to be underdiagnosed and undertreated relative to the evidence that treatment improves functional outcomes (9). Collectively, this literature indicates that although the musculoskeletal burden of post-stroke myofascial pain is well documented descriptively, randomized interventional evidence, particularly evidence targeting the autonomic contribution to this pain, remains scarce.

Beyond the musculoskeletal domain, autonomic nervous system (ANS) dysregulation is a well-documented consequence of stroke. This manifests as reduced heart rate variability (HRV), sympathetic predominance and blunted parasympathetic modulation (10-12). Autonomic imbalance is associated with impaired cardiovascular recovery, persistent fatigue, emotional dysregulation and elevated stroke-related mortality (11). Critically, ANS dysfunction contributes directly to pain amplification: elevated sympathetic activity sensitizes peripheral nociceptors, impairs microvascular perfusion and perpetuates the ischemic milieu of myofascial tissues (13). This bidirectional relationship between autonomic dysfunction and nociception is further substantiated by evidence that sympathetically driven peripheral sensitization involves upregulation of alpha-1 adrenoreceptors on nociceptors and sympathetic sprouting within sensory ganglia, mechanisms explicitly implicated in myofascial and widespread pain conditions (14). Autonomic modulation techniques including slow diaphragmatic breathing, HRV-biofeedback and mindfulness-based relaxation represent a theoretically sound, non-invasive approach that may concurrently address both musculoskeletal and autonomic deficits. Breathing at the resonant frequency (approximately 6 breaths/min) activates baroreflex mechanisms, augments vagal tone and attenuates central sympathetic drive (15-17). HRV-biofeedback training enhances cardiovagal control and self-regulation and reduces chronic pain symptoms across diverse populations (18,19). Mindfulness-based relaxation modulates emotional and cognitive components of pain perception through central autonomic network reorganization (20,21).

Despite this mechanistic rationale, several gaps persist in the current evidence base. First, existing studies of post-stroke pain interventions have

predominantly targeted peripheral or biomechanical contributors, with comparatively few randomized trials evaluating interventions that act directly on autonomic regulation. Second, diagnostic criteria for myofascial trigger points have historically varied across studies, limiting comparability and pooled interpretation of findings. Third, follow-up periods in prior autonomic-intervention studies have generally been short and objective, quantifiable biomarkers of autonomic change, as opposed to self-reported outcomes alone, have been inconsistently reported alongside pain outcomes. Fourth, to the best of our knowledge, no randomized trial to date has concurrently quantified both myofascial pain outcomes and multi-domain HRV indices within a chronic post-stroke population receiving a structured, multi-component autonomic modulation protocol.

Despite these promising mechanisms, the specific impact of structured autonomic modulation on myofascial pain outcomes in post-stroke individuals, a population uniquely vulnerable to compounded neuromuscular and autonomic injury, remains insufficiently studied. HRV provides a validated, objective biomarker of sympathovagal balance, enabling direct quantification of autonomic intervention effects. The overarching aim of the present study was to evaluate the effects of a structured, three-component autonomic modulation protocol, comprising slow diaphragmatic breathing, HRV-biofeedback and mindfulness-based relaxation, on myofascial pain and cardiac autonomic regulation in chronic post-stroke individuals. The specific objectives were:

- (a) to compare within-group changes in pain intensity (VAS), pressure pain threshold and HRV indices (RMSSD, HF power, LF/HF ratio) from baseline to four weeks in an Autonomic Modulation Group and a Control Group receiving conventional physiotherapy; and
- (b) to compare the magnitude of these changes between groups.

The novelty of this study lies in its concurrent assessment of musculoskeletal (pain and pressure pain threshold) and multi-parameter autonomic (time- and frequency-domain HRV) outcomes within a single structured protocol in the chronic post-stroke population, directly linking autonomic recalibration to myofascial pain relief rather than

examining either domain in isolation, thereby generating evidence to support the integration of mind-body physiotherapeutic strategies into post-stroke neurorehabilitation.

Methodology

Study Design

A randomized controlled trial (RCT) with a pre-test and post-test design was conducted and reported in accordance with the CONSORT 2010 statement (22), as shown in Figure 1. The study was approved by the Institutional Scientific Review Board (Reference No.: 034/05/2025/ISRB/PGSR/SCPT). All participants provided written informed consent prior to enrolment. The study was conducted in accordance with the Declaration of Helsinki (2013 revision).

Participants

A total of 40 post-stroke individuals were recruited from Saveetha Medical College and Hospital, Chennai (Saveetha Nagar, Thandalam campus: approximate GPS coordinates 13.0270°N, 80.0165°E). Inclusion criteria were: adults aged 40-70 years; post-stroke duration ≥ 6 months (chronic phase); confirmed presence of active myofascial trigger points in the trapezius or cervical musculature assessed by standardized manual palpation; and ability to comprehend and follow instructions. Participants were excluded if they had cardiac arrhythmias or implanted cardiac devices, uncontrolled hypertension (systolic >180 mmHg), significant cognitive impairment (Mini-Mental State Examination score <24), or any prior participation in autonomic training or biofeedback-based rehabilitation.

Participants were randomized using a simple random allocation method (computer-generated random numbers) into two groups of 20 participants each: the Autonomic Modulation Group (AMG) and the Control Group (CG). Recruitment, randomization and follow-up are summarised in the CONSORT flow diagram, as shown in Figure 1; no participants were excluded after eligibility screening and no attrition occurred during the 4-week intervention period.

Sample Size

A sample of 40 participants (20 per group) was recruited based on clinical feasibility and in alignment with comparable pilot RCTs in neurorehabilitation (18). Future adequately

powered replication studies using formal a priori power calculations are recommended.

Interventions

Both groups received 35-minute sessions, 5 days per week, for 4 consecutive weeks.

Autonomic Modulation Group (AMG): Participants received a structured three-component protocol:

- (a) Slow Diaphragmatic Breathing (15 min) - guided breathing at 6 breaths/min targeting baroreflex resonance frequency, with an exhalation and inhalation ratio of 2:1, emphasizing diaphragmatic expansion and minimizing accessory cervical muscle recruitment;
- (b) HRV-Biofeedback Training (10 min) - real-time cardiac coherence monitoring using a validated finger-sensor device, with participants coached to synchronize respiratory cycles with displayed HRV waveforms at resonant frequency (5-7 breaths/min); and
- (c) Mindfulness-Based Relaxation (10 min) - structured guided audio script incorporating progressive body scanning, sequential cervical and shoulder muscle relaxation and non-evaluative breath awareness.

Control Group (CG): Participants received a structured conventional physiotherapy protocol delivered under the direct one-on-one supervision of a licensed physiotherapist throughout every session, comprising: range of motion (ROM) exercises for the cervical and shoulder girdle (10 min, performed as 3 sets of 10 repetitions per movement plane at a low-to-moderate, pain-free intensity); static and dynamic stretching of the upper trapezius, levator scapulae and pectoralis major/minor (10 min, each static stretch held for 20-30 seconds for 3 repetitions per muscle group); and progressive resistance training using theraband for shoulder girdle stabilizers (15 min, 3 sets of 10-12 repetitions per exercise, beginning with a low-resistance band and progressing to the next resistance level approximately every 1-2 weeks according to individual tolerance, consistent with standard progressive-overload principles). Therapist supervision, exercise dosage and progression criteria were standardized across all Control Group sessions using a written treatment protocol checklist to minimize inter-session variability.

Outcome Measures

All outcomes were assessed at baseline and at 4 weeks by a blinded assessor.

Pain Intensity: Visual Analogue Scale (VAS; 0-10 cm), a reliable and validated tool for pain assessment in neurological populations.

Pressure Pain Threshold (PPT): Measured using a calibrated handheld pressure algometer (units: kg/cm²) at the most sensitive myofascial trigger point in the upper trapezius, as per standardized protocols.

Heart Rate Variability (HRV): HRV was monitored using a wrist-worn photoplethysmography-based device (Apple Watch). Recordings were obtained over a continuous 5-minute epoch with participants seated quietly in a temperature-controlled room (approximately 22-24°C), minimizing ambient noise and conversation, after a minimum 10-minute rest period; participants were instructed to breathe spontaneously (unpaced) and to remain still to minimize motion artefact. Raw beat-to-beat (RR) interval data were exported from the Apple Health application and processed using validated third-party HRV analysis software, which applied automated artefact-correction (ectopic beat removal and interpolation) prior to time-domain and frequency-domain analysis; recordings with more than 5% corrected beats were excluded and repeated. Time-domain HRV (RMSSD) was calculated as the root mean square of successive differences between normal-to-normal intervals, a validated measure reflecting parasympathetic (vagal) activity, while frequency-domain indices (HF power, LF/HF ratio) were derived by fast Fourier transform spectral analysis of the RR-interval series. We note that the Apple

Watch/Apple Health platform natively reports only the time-domain SDNN metric; the frequency-domain HRV indices reported here (HF power, LF/HF ratio) required third-party spectral analysis software applied to exported RR-interval data rather than being retrieved directly from the native Apple Health application and this is acknowledged as a methodological constraint in the Limitations (23, 24).

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Normality was assessed using the Shapiro-Wilk test. Within-group pre-to-post differences were evaluated using paired-samples t-tests for normally distributed data. Between-group differences in change scores were analyzed using independent-samples t-tests. Statistical significance was set at $P < 0.05$ (two-tailed). Data are presented as mean $\pm$ standard deviation (SD). Figures were produced using R software, version 4.3.0.

Results

Participant Flow and Baseline Characteristics

Of the 56 post-stroke individuals assessed for eligibility, 16 were excluded and 40 met the eligibility criteria and were randomized {20 to the Autonomic Modulation Group (AMG) and 20 to the Control Group (CG)}. No participants were lost to follow-up or discontinued the intervention and all 40 participants were included in the final analysis. Participant flow through enrolment, allocation, follow-up and analysis is summarised in Figure 1, presented in accordance with the CONSORT 2010 statement (22).

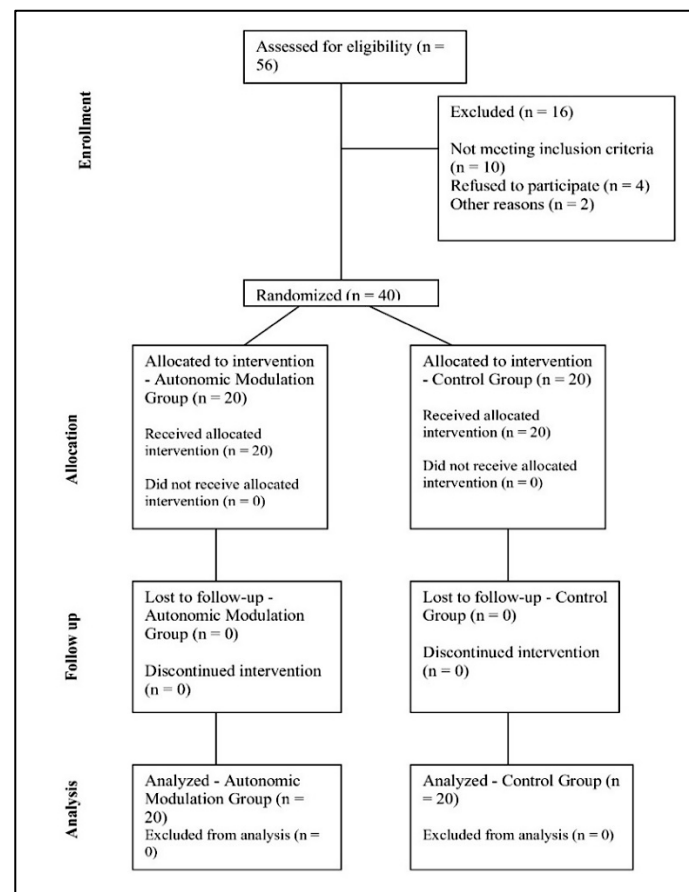


Figure 1: CONSORT Flow Diagram of Participant Enrolment, Allocation, Follow-up and Analysis

Note: n = Sample Size at Each Stage of Enrolment, Allocation, Follow-up and Analysis, AMG = Autonomic Modulation Group, CG = Control Group.

Within-group effects: Autonomic Modulation Group

The within-group analysis for the Autonomic Modulation Group (AMG) demonstrated a coherent and multidimensional therapeutic response, encompassing both pain reduction and autonomic restoration. As detailed in Table 1, all outcome measures improved significantly following the 4-week intervention.

Pain intensity showed a marked decline, with VAS scores reducing from 6.8 ± 1.1 to 3.5 ± 0.9 ($t = 11.42$, $P < 0.001$), reflecting a substantial and clinically meaningful attenuation of perceived pain. This was paralleled by a significant increase in PPT from 2.4 ± 0.5 to 3.1 ± 0.6 kg/cm² ($t = 5.31$, $P < 0.001$), indicating reduced peripheral nociceptive sensitivity and improved myofascial tolerance.

Crucially, these musculoskeletal improvements were accompanied by robust autonomic recalibration. RMSSD increased from 18.4 ± 4.5 to 29.8 ± 5.2 ms ($t = 9.14$, $P < 0.001$) and HF power

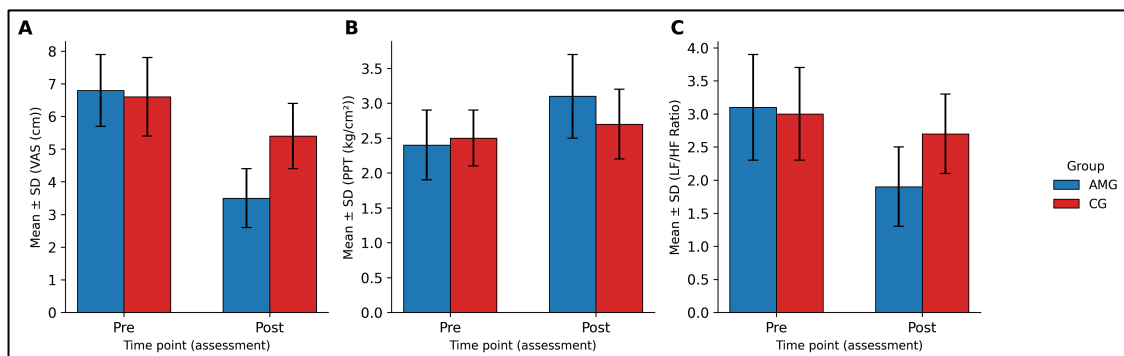
rose from 210 ± 58 to 340 ± 70 ms² ($t = 7.92$, $P < 0.001$), both reflecting enhanced parasympathetic (vagal) modulation. Simultaneously, the LF/HF ratio decreased from 3.1 ± 0.8 to 1.9 ± 0.6 ($t = 7.23$, $P < 0.001$), indicating a system-level shift away from sympathetic dominance toward autonomic balance. In relative terms, this corresponds to a 48.5% reduction in VAS pain score, a 29.2% increase in PPT, a 61.9% increase in RMSSD, a 61.9% increase in HF power and a 38.7% reduction in LF/HF ratio within the AMG over the 4-week intervention period, indicating that the magnitude of autonomic change closely paralleled the magnitude of pain-related change.

The convergence of pain reduction and vagal enhancement suggests that the intervention did not merely alleviate symptoms but modulated the underlying neurophysiological drivers of pain. This integrated response is visually reinforced in Figure 2, where the simultaneous improvement in VAS, PPT and LF/HF ratio illustrates the coordinated recovery across both musculoskeletal and autonomic domains.

Table 1: Within-group Comparison of Pre- and Post-intervention Outcomes in the Autonomic Modulation Group (AMG) - Paired t-test

Outcome Measure	Mean $\pm$ SD (Pre)	Mean $\pm$ SD (Post)	Paired t-value	P-value
VAS (0-10 cm)	6.8 $\pm$ 1.1	3.5 $\pm$ 0.9	11.42	P < 0.001
Pressure Pain Threshold (kg/cm ²)	2.4 $\pm$ 0.5	3.1 $\pm$ 0.6	5.31	P < 0.001
RMSSD (ms)	18.4 $\pm$ 4.5	29.8 $\pm$ 5.2	9.14	P < 0.001
HF Power (ms ²)	210 $\pm$ 58	340 $\pm$ 70	7.92	P < 0.001
LF/HF Ratio	3.1 $\pm$ 0.8	1.9 $\pm$ 0.6	7.23	P < 0.001

Note: AMG: Autonomic Modulation Group, VAS = Visual Analogue Scale, PPT = Pressure Pain Threshold, RMSSD = Root Mean Square of Successive Differences, HF = High-Frequency Power, LF/HF = Low-Frequency to High-Frequency Ratio, SD = Standard Deviation.

**Figure 2:** Pre- and Post-intervention Comparison of Pain and Autonomic Outcomes in the Autonomic Modulation Group (AMG, blue) and Control Group (CG, red), mean $\pm$ SD. (A) Visual Analogue Scale (VAS) pain score, (B) Pressure Pain Threshold (PPT), (C) LF/HF ratio

Within-group Effects: Control Group

In contrast, the Control Group (CG) demonstrated a partial and predominantly peripheral response, as summarized in Table 2. While statistically significant improvements were observed in pain-related outcomes, the magnitude and scope of change were notably limited.

VAS scores decreased from 6.6 ± 1.2 to 5.4 ± 1.0 ($t = 4.28$, $P = 0.001$) and PPT increased modestly from 2.5 ± 0.4 to 2.7 ± 0.5 kg/cm² ($t = 2.21$, $P = 0.039$). These findings indicate that conventional physiotherapy can produce localized mechanical and symptomatic relief.

However, autonomic indices showed minimal responsiveness. RMSSD increased only marginally (19.1 ± 4.1 to 20.6 ± 4.2 ms, $P = 0.050$), HF power changes were non-significant (215 ± 62 to $230 \pm$

65 ms², $P = 0.092$) and the LF/HF ratio reduction was borderline (3.0 ± 0.7 to 2.7 ± 0.6 , $P = 0.053$). Collectively, these findings indicate that conventional physiotherapy did not meaningfully engage central autonomic regulatory mechanisms. Notably, the CG achieved a comparable relative VAS reduction to its own pre-treatment pain level (18.2%) alongside near-flat autonomic indices (RMSSD +7.9%, HF power +7.0%, LF/HF -10.0%), reinforcing that peripherally directed conventional physiotherapy dissociates pain relief from autonomic recalibration, unlike the coupled response observed in the AMG.

This divergence is clearly illustrated in Figure 3, where the limited upward shift in RMSSD and HF power in the CG contrasts sharply with the pronounced autonomic gains observed in the AMG.

Table 2: Within-group Comparison of Pre- and Post-intervention Outcomes in the Control Group (CG) - Paired t-test

Outcome Measure	Mean $\pm$ SD (Pre)	Mean $\pm$ SD (Post)	Paired t-value	P-value
VAS (0-10 cm)	6.6 $\pm$ 1.2	5.4 $\pm$ 1.0	4.28	P < 0.001
Pressure Pain Threshold (kg/cm ²)	2.5 $\pm$ 0.4	2.7 $\pm$ 0.5	2.21	P = 0.039
RMSSD (ms)	19.1 $\pm$ 4.1	20.6 $\pm$ 4.2	2.08	P = 0.050
HF Power (ms ²)	215 $\pm$ 62	230 $\pm$ 65	1.77	P = 0.092
LF/HF Ratio	3.0 $\pm$ 0.7	2.7 $\pm$ 0.6	2.05	P = 0.053

Note: CG = Control Group, VAS = Visual Analogue Scale, PPT = Pressure Pain Threshold, RMSSD = Root Mean Square of Successive Differences, HF = High-Frequency Power, LF/HF = Low-Frequency to High-Frequency Ratio, SD = Standard Deviation.

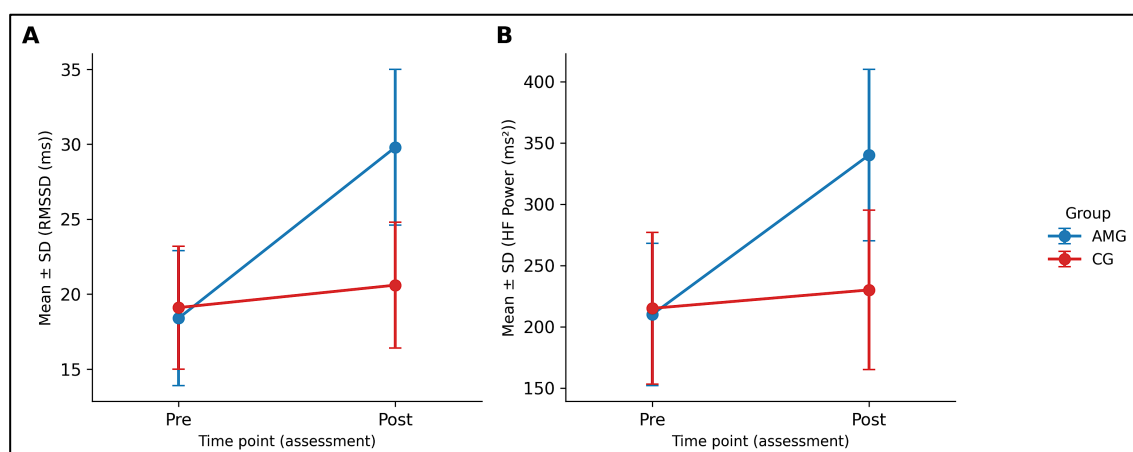


Figure 3: Pre- and Post-intervention Changes in Heart Rate Variability Indices in the Autonomic Modulation Group (AMG, blue) and Control Group (CG, red), mean $\pm$ SD. (A) RMSSD, (B) HF power.

Note: AMG = Autonomic Modulation Group, CG = Control Group, RMSSD = Root Mean Square of Successive Differences, HF = High-Frequency Power, SD = Standard Deviation.

Between-group Comparison: Magnitude and Specificity of Effects

The superiority of autonomic modulation becomes unequivocal when examining between-group differences in change scores, as presented in Table 3. Across all variables, the AMG demonstrated significantly greater improvements, with large effect magnitudes and consistent statistical significance ($P < 0.001$ for all comparisons).

The reduction in pain intensity was nearly threefold greater in the AMG (3.3 ± 0.8) compared to the CG (1.2 ± 0.6) ($t = 9.46$, $P < 0.001$), while PPT gains were more than three times higher (0.7 ± 0.3 vs. 0.2 ± 0.2 kg/cm², $t = 6.53$, $P < 0.001$). These findings indicate a substantially enhanced capacity for both subjective pain relief and objective nociceptive modulation.

The most pronounced differences were observed in autonomic parameters. RMSSD increased by 11.4 ± 4.0 ms in the AMG versus 1.5 ± 2.5 ms in the

CG ($t = 9.55$, $P < 0.001$) and HF power improved by 130 ± 40 ms² compared to 15 ± 30 ms² ($t = 10.47$, $P < 0.001$). Furthermore, the reduction in LF/HF ratio was significantly greater in the AMG (1.2 ± 0.4) than in the CG (0.3 ± 0.3) ($t = 8.58$, $P < 0.001$), confirming a more profound restoration of sympathovagal equilibrium. Expressed as between-group effect ratios, the AMG change score exceeded the CG change score by approximately 2.8-fold for VAS, 3.5-fold for PPT, 7.6-fold for RMSSD, 8.7-fold for HF power and 4.0-fold for LF/HF ratio, indicating that the relative advantage of autonomic modulation was most pronounced for the autonomic (HRV) outcomes rather than the pain outcomes alone.

These between-group differences are visually consolidated in Figure 4, where the distribution of mean change scores demonstrates a consistent and substantial advantage of the AMG across all domains, reinforcing the specific and targeted efficacy of autonomic modulation.

Table 3: Between-group Comparison of Mean Change Scores - Independent Samples t-test

Outcome Measure	AMG Change (Mean $\pm$ SD)	CG Change (Mean $\pm$ SD)	Independent t-value	P-value
VAS (0-10 cm)	3.3 ± 0.8	1.2 ± 0.6	9.46	$P < 0.001$
Pressure Pain Threshold (kg/cm ²)	0.7 ± 0.3	0.2 ± 0.2	6.53	$P < 0.001$
RMSSD (ms)	11.4 ± 4.0	1.5 ± 2.5	9.55	$P < 0.001$
HF Power (ms ²)	130 ± 40	15 ± 30	10.47	$P < 0.001$
LF/HF Ratio	1.2 ± 0.4	0.3 ± 0.3	8.58	$P < 0.001$

Note: AMG = Autonomic Modulation Group, CG = Control Group, VAS = Visual Analogue Scale, PPT = Pressure Pain Threshold, RMSSD = Root Mean Square of Successive Differences, HF = High-Frequency Power, LF/HF = Low-Frequency to High-Frequency Ratio, SD = Standard Deviation.

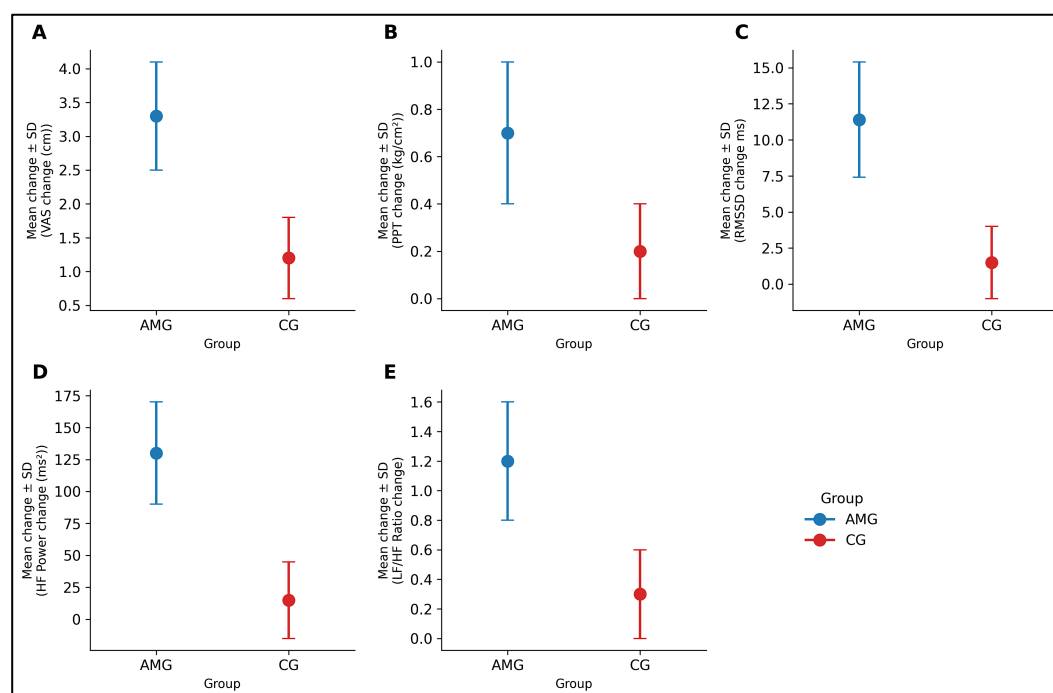


Figure 4: Between-group Comparison of Mean Change Scores (Post Minus Pre) in the Autonomic Modulation Group (AMG, blue) and Control Group (CG, red), mean $\pm$ SD. (A) VAS, (B) PPT, (C) RMSSD, (D) HF power, (E) LF/HF ratio

Note: AMG = Autonomic Modulation Group, CG = Control Group, VAS = Visual Analogue Scale, PPT = Pressure Pain Threshold, RMSSD = Root Mean Square of Successive Differences, HF = High-Frequency Power, LF/HF = Low-Frequency to High-Frequency Ratio, SD = Standard Deviation.

Integrated Outcome Pattern

Taken together, the results reveal a clear mechanistic distinction between interventions. Conventional physiotherapy produced modest, primarily peripheral improvements, whereas autonomic modulation induced simultaneous and interlinked changes in pain perception, nociceptive sensitivity and autonomic regulation. The alignment of increased vagal activity (RMSSD, HF) with reduced pain (VAS) and increased PPT suggests that pain reduction in the AMG is closely coupled with autonomic rebalancing. This pattern strongly supports the interpretation that targeting autonomic dysfunction yields broader and more integrated therapeutic benefits in post-stroke myofascial pain.

Discussion

The present study investigated the effects of a structured autonomic modulation protocol on myofascial pain and HRV in chronic post-stroke individuals. The principal finding is that 4 weeks of slow diaphragmatic breathing, HRV-biofeedback and mindfulness-based relaxation produced significantly greater improvements in pain

intensity, PPT and all HRV parameters compared to conventional physiotherapy.

Post-stroke myofascial pain arises from interacting mechanisms including aberrant motor recruitment, spasticity, compensatory muscle overactivity and sympathetically driven peripheral sensitization (1-3). The presence of trigger points in cervical and shoulder muscles creates a self-perpetuating cycle of ischemia, nociceptive sensitization and pain chronification (25, 26). The substantial VAS reductions and PPT improvements observed in the AMG are consistent with evidence that parasympathetic activation suppresses peripheral nociceptor excitability, improves myofascial microcirculation and reduces central sensitization (27, 28). Beyond this peripheral action, vagal afferent signalling is understood to inhibit spinal nociceptive transmission directly and to engage descending pain-modulatory circuits, including the periaqueductal grey and rostral ventromedial medulla, providing a plausible central mechanism for the analgesic effect observed (14, 29). Breathing at the resonant frequency of 6 breaths/min activates baroreflex pathways, augments vagal efferent output and

attenuates sympathetic hyperactivity, thereby reducing peripheral inflammatory and nociceptive drive (15, 30). Slow breathing-induced increases in vagal tone are additionally proposed to enhance regional microvascular perfusion through attenuation of sympathetically mediated vasoconstriction and improved baroreflex-mediated haemodynamic regulation; direct human evidence remains mixed, with paced slow breathing shown to increase microvascular flow in cranial vascular beds but to leave muscle sympathetic nerve activity and forearm vascular conductance unchanged in some controlled physiological studies (31-33), indicating that any peripheral perfusion benefit specific to myofascial tissue warrants direct confirmation. These physiological effects are further reinforced by the central analgesic actions of mindfulness, which modulates prefrontal and anterior cingulate cortical activity, reducing affective and evaluative dimensions of pain perception (34, 35). More broadly, sustained engagement of vagal afferent-brainstem-forebrain circuits through HRV-biofeedback and mindfulness practice has been proposed to drive neuroplastic reorganization of prefrontal-limbic pain-regulatory networks, offering a mechanistic bridge between short-term autonomic recalibration and longer-term modulation of pain perception (36).

Comparison with Previous Research

The direction of our findings is broadly consistent with, though not identical to, prior autonomic-intervention trials in stroke and chronic pain populations. In a randomized sham-controlled pilot study of acute ischaemic stroke patients, nine sessions of HRV-biofeedback produced a significant increase in SDNN relative to sham stimulation, supporting the feasibility and cardiac-autonomic efficacy of biofeedback-based autonomic training in the stroke population (37), a finding that agrees in direction with the RMSSD and HF power gains observed in our AMG. A quasi-experimental (non-randomized) study similarly reported that HRV-biofeedback improved cognitive, motor, psychological and autonomic outcomes in post-stroke rehabilitation over a 6-month follow-up, although its non-randomized design and longer intervention period limit direct comparison with the present 4-week RCT (38). In contrast, a randomized trial of mindfulness-based cognitive therapy in stroke survivors found no

significant improvement in depression or anxiety relative to standard care over 6 weeks (39), illustrating that not all mind-body interventions yield uniform benefit in this population and suggesting that outcome domain, dose and baseline symptom severity may moderate treatment response. At the level of chronic pain populations more broadly, systematic reviews and meta-analyses of mindfulness-based interventions and of HRV-biofeedback report, respectively, a small-to-moderate reduction in pain intensity across pooled trials (40) and moderate-to-large effects that are more consistent for anxiety and physiological regulation than for pain specifically (41). Against this backdrop, the large between-group effect sizes observed in the present trial, as presented in Table 3, are at the higher end of previously reported effects. This may reflect the multi-component nature of our protocol, which combined breathing, biofeedback and mindfulness rather than a single modality, the chronic and homogeneous post-stroke myofascial pain population studied, or the comparatively small sample size. Therefore, these effect sizes should be interpreted with appropriate caution pending replication in larger, multicentric cohorts.

The HRV improvements within the AMG - elevated RMSSD, increased HF power and reduced LF/HF ratio - directly corroborate the autonomic mechanisms underlying the intervention. RMSSD and HF power are established indices of cardiac vagal modulation and their significant increase confirms enhanced parasympathetic tone following the protocol. The reduction in LF/HF ratio indicates a shift away from sympathetic dominance, which is particularly relevant given that post-stroke autonomic imbalance is associated with poor functional prognosis and elevated all-cause mortality (10, 42, 43). These findings align with randomized evidence demonstrating that HRV-biofeedback augments cardiovagal control through respiratory-cardiac oscillation entrainment and baroreceptor resetting (18, 44-46).

In contrast, the CG showed limited and, for some HRV indices, statistically non-significant improvements. Conventional physiotherapy, targeting primarily biomechanical deficits through ROM exercises, stretching and strengthening, does not act directly on central or peripheral autonomic pathways (47, 48). This mechanistic divergence

explains the substantially larger between-group effect sizes across all outcome domains. As detailed in Tables 1-3, the CG did achieve statistically significant within-group pain relief (VAS and PPT), indicating that biomechanically targeted therapy retains genuine, clinically relevant value; however, the parallel HRV indices in the CG moved only marginally and, for HF power and LF/HF ratio, did not reach conventional statistical significance, whereas every AMG outcome, pain-related and autonomic alike, improved with high statistical confidence. This pattern, most clearly visualised in the between-group change-score comparison presented in Table 3 and Figure 4, supports the interpretation that the additional benefit of autonomic modulation is concentrated in the autonomic domain rather than being a generic, non-specific effect of receiving supervised therapy. The neurovisceral integration model provides a theoretical framework for interpreting the pain-HRV relationship observed here. Higher HRV indexes greater prefrontal inhibition of limbic structures, facilitating superior pain regulation, emotional control and behavioral flexibility (15). Consistent with this model, prior systematic evidence has established inverse relationships between HRV and pain severity, with higher vagal tone predicting better pain coping and functional outcomes (49).

Practical Implications and Recommendations

These findings carry several practical implications for post-stroke neurorehabilitation. First, the three intervention components used here (slow diaphragmatic breathing, HRV-biofeedback and mindfulness-based relaxation) are low-cost, low-risk and require only a portable finger-sensor biofeedback device, making the protocol feasible for adoption in outpatient physiotherapy and community rehabilitation settings without specialized equipment. Second, delivery in short, structured sessions (35 minutes, 5 days per week) over 4 weeks proved sufficient to produce measurable autonomic and musculoskeletal change, suggesting that autonomic modulation could realistically be layered onto existing physiotherapy schedules as an adjunct rather than a replacement for conventional care, consistent with implementation models in which HRV-biofeedback is delivered as a structured weekly add-on until skill acquisition is achieved (50).

Third, clinicians managing post-stroke myofascial pain that responds incompletely to conventional physiotherapy alone may consider incorporating autonomic modulation techniques, particularly where objective HRV monitoring is available to titrate and track treatment response. We recommend that future implementation studies formally evaluate cost-effectiveness, therapist training requirements and patient adherence over longer treatment durations before broader clinical adoption is advocated.

Limitations

Several limitations merit acknowledgment. The sample size ($n = 40$) limits statistical power and generalizability. The absence of long-term follow-up precludes conclusions about durability of effects. Blinding of participants to group allocation was not feasible. The study population was restricted to chronic post-stroke individuals (≥ 6 months post-stroke); consequently, these findings are applicable only to the chronic phase and cannot be generalised to patients in the acute or subacute phase of stroke, in whom autonomic status, pain mechanisms and rehabilitation potential may differ substantially, without further dedicated evidence. In addition, frequency-domain HRV indices (HF power, LF/HF ratio) were derived from a consumer wrist-worn photoplethysmography device via third-party spectral analysis of exported RR-interval data rather than gold-standard ECG-based acquisition; ultra-short recording epochs and photoplethysmography-derived RR series are more susceptible to motion and detection artefact than laboratory ECG, particularly for frequency-domain measures and this should be considered when interpreting the absolute magnitude of HRV change reported here (23, 24). Future multicentric RCTs with larger samples, active control conditions, long-term follow-up and additional biomarkers (e.g., inflammatory cytokines, baroreflex sensitivity) are warranted to confirm and extend these findings.

Conclusion

A structured 4-week autonomic modulation protocol incorporating slow diaphragmatic breathing, HRV-biofeedback and mindfulness-based relaxation resulted in significant improvements in both myofascial pain and cardiac autonomic regulation among individuals with chronic post-stroke pain. Compared with

conventional physiotherapy, the autonomic modulation group demonstrated greater reductions in pain intensity, greater improvements in pressure pain threshold and more pronounced changes in RMSSD, HF power and LF/HF ratio, indicating concurrent improvement across musculoskeletal and autonomic domains. These findings suggest that autonomic modulation may provide benefits beyond symptom-focused pain management by addressing autonomic dysregulation that may contribute to persistent post-stroke pain.

The principal contribution of this trial is the concurrent evaluation of pain, pressure pain sensitivity and multiple HRV parameters within a single structured intervention in a chronic post-stroke population. This integrated approach provides preliminary clinical evidence supporting the incorporation of autonomic modulation strategies as an adjunct to conventional neurorehabilitation, particularly for individuals whose myofascial pain remains insufficiently responsive to conventional physiotherapy. However, the findings should be interpreted cautiously because of the modest sample size, single-centre design, short intervention and follow-up period, absence of participant blinding and reliance on a consumer wrist-worn device with third-party processing for frequency-domain HRV assessment. These factors may limit the precision, durability and generalisability of the observed effects.

Future research should therefore evaluate this approach in larger, adequately powered multicentre randomized controlled trials with longer-term follow-up, active comparator conditions, standardized laboratory-grade ECG-based HRV assessment and objective measures of autonomic and physiological function. Studies should also examine treatment adherence, dose-response relationships, cost-effectiveness and whether baseline autonomic dysfunction or pain characteristics can identify individuals most likely to benefit. Such research will help determine the sustainability, clinical applicability and scalability of autonomic modulation within comprehensive post-stroke neurorehabilitation.

Abbreviations

AMG: Autonomic Modulation Group, CG: Control Group, HF: High-Frequency Power, HRV: Heart Rate Variability, MPS: Myofascial Pain Syndrome,

PPT: Pressure Pain Threshold, RCT: Randomized Controlled Trial, RMSSD: Root Mean Square of Successive Differences, SD: Standard Deviation, VAS: Visual Analogue Scale.

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Author Contributions

Simran Sharma D: Conceptualization, Methodology Development, Participant Recruitment, Data Collection, Data Curation, Preparation of the Original Manuscript Draft, Aravind S: Study Design, Methodology Development, Statistical Analysis, Project Administration, Manuscript Drafting, Prathap Suganthirababu: Methodological Guidance, Formal Data Analysis, Interpretation of Findings, Critical Review of the Manuscript, Aravindh B: Participant Assessment, Data Collection, Data Management, Manuscript Review, Jeevarathinam Thirumalai: Study Supervision, Conceptual Guidance, Formal Analysis, Manuscript Review and Editing, Overall Project Oversight. All authors read and approved the final manuscript.

Conflict of Interest

The authors declare no Conflict of Interest

Data Availability

The data that support the findings of this study are not publicly available due to confidentiality and privacy restrictions.

Declaration of Artificial Intelligence (AI) Assistance

The authors acknowledge the use of QuillBot, an AI-assisted language tool, solely for language refinement and paraphrasing of certain parts of the manuscript, as English is not the authors' native language. Its use was limited to improving language clarity, readability, grammar and overall expression. All AI-assisted content was critically reviewed, edited and verified by the authors to ensure accuracy, originality and compliance with academic standards. The authors take full responsibility for the integrity and validity of the manuscript. No generative AI tools were used to create, modify, or generate any figures in this manuscript. All figures were generated using R software, version 4.3.0, based on the study data and statistical analyses.

Ethics Approval

This study was approved by the Institutional Scientific Review Board of Saveetha College of Physiotherapy, Chennai, India (Reference No.: 034/05/2025/ISRB/PGSR/SCPT). Written informed consent was obtained from all participants prior to enrolment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision).

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