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Review Article

Long-Term Follow-Up of multidisciplinary pain management for post-stroke hemiparesis

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Abstract

Background: Hemiplegic shoulder pain and post-stroke spasticity are persistent complications of hemiparesis, affecting roughly one third to one half of survivors within the first year and often overlapping mechanistically [1,3].

Objective: This review synthesizes evidence on multidisciplinary pain management for post-stroke hemiparesis, emphasizing shoulder pain and spasticity-related pain, and what long-term follow-up has shown about durability of treatment effects beyond one year.

Methods: We synthesized prospective epidemiological cohorts, randomized trials of biomechanical, pharmacological, interventional and stimulation treatments, and systematic reviews addressing pain and spasticity after hemiparetic stroke.

Findings: Pain and spasticity often emerge within two to four months and predict persistence at twelve months and beyond [7,38]; biomechanical support alone shows inconsistent benefit [24,26], early stimulation reduces subsequent pain [34], botulinum toxin best supports spasticity-driven pain [28,29,30], and nerve block benefits neuropathic pain [33].

Conclusions: Hemiparetic pain resists single-modality treatment and is best managed through multidisciplinary care combining early protection, targeted pharmacological or interventional treatment, and structured long-term follow-up [20,6].

Keywords: hemiplegic shoulder pain; post-stroke spasticity; multidisciplinary rehabilitation; botulinum toxin; long-term follow-up

1. Introduction

Pain in the paretic limb after stroke is one of the most persistently under-treated complications of cerebrovascular disease, and hemiplegic shoulder pain is its most common form. Population-based cohorts consistently report that between one third and one half of people with hemiparesis develop clinically significant shoulder pain, usually emerging within the first two to three months and, in a substantial minority, still present a year later [1,2]. Spasticity, which develops in roughly one in three patients with hemiparesis and is itself an independent driver of pain and reduced participation in therapy, frequently co-exists with and compounds the shoulder problem rather than acting as a separate, later complication [3,4]. Because post-stroke pain is rarely explained by a single mechanism, rehabilitation practice has moved toward multidisciplinary management combining physical therapy, occupational therapy, targeted pharmacological or interventional treatment, and structured long-term follow-up, rather than a single modality delivered in isolation [5,6].

This review synthesizes evidence on multidisciplinary pain management for post-stroke hemiparesis, with particular attention to hemiplegic shoulder pain and spasticity-related pain, and to what long-term follow-up studies have shown about the durability of treatment effects. We examine the epidemiology and natural history of these pain syndromes, their overlapping pathophysiology, multidisciplinary assessment and rehabilitation, pharmacological and interventional treatment, and what is known about outcomes beyond the first year.

2. Epidemiology and Natural History

Several prospective cohorts have characterized the natural history of hemiplegic shoulder pain from the acute stroke unit through community rehabilitation. Lindgren and colleagues, following a population-based Swedish cohort, found shoulder pain in approximately one third of

patients within four months, and pain present at four months carried a substantially elevated risk of pain persisting at twelve months [1]. A New Zealand population-based study similarly found shoulder pain prevalence of around 30-40% within the first year, with older age, more severe initial paresis, and reduced shoulder range of motion as consistent predictors [2]. A Dutch prospective cohort tracking pain trajectory over six months found that pain unresolved at three months strongly predicted pain at six months, arguing for early identification rather than a wait-and-see approach [7]. Dromerick and colleagues, in an inpatient rehabilitation cohort, found hemiplegic shoulder pain syndrome already detectable within the first two weeks, well before most outpatient referral pathways would begin [8].

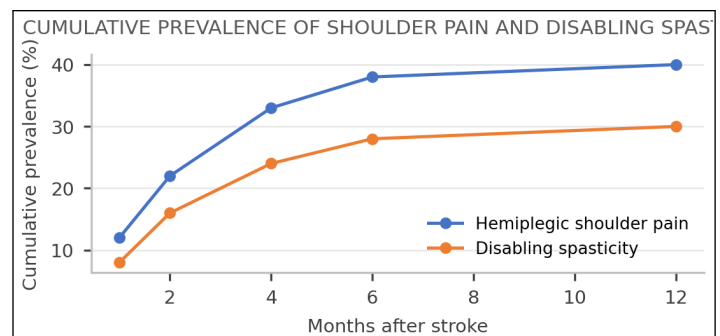


Figure 1: Cumulative prevalence of hemiplegic shoulder pain and disabling spasticity over the first year after stroke in prospective population-based cohorts, showing early onset within the first two to four months and substantial persistence to twelve months [1,7,3].

Spasticity follows a broadly parallel but distinct trajectory. Sommerfeld and colleagues, using the modified Ashworth scale, found that about one in three patients had spasticity within the first week, correlating with more severe motor impairment independent of pain status [4,9]. Lundstrom and colleagues, extending follow-up to one year, found disabling spasticity persisted in a meaningful minority of survivors and was strongly associated with reduced quality of life [3]. Wissel and colleagues' broader epidemiological review concluded that a substantial proportion of patients with early spasticity go on to have persistent spasticity-related disability, including pain, well beyond six months [10].

3. Pathophysiology of Pain in Hemiparesis

Hemiplegic shoulder pain has no single mechanism, and multidisciplinary management reflects an implicit acknowledgment of that heterogeneity. Biomechanical contributors include glenohumeral subluxation from loss of rotator cuff tone, capsular contracture from prolonged immobility and abnormal positioning, and secondary soft-tissue impingement, documented in cohorts using both examination and imaging [11,12]. Vasudevan and Browne's review frames the differential around these overlapping biomechanical processes, together with adhesive capsulitis and pre-existing rotator cuff pathology, arguing that treatment selection should follow a structured examination distinguishing these contributors rather than treating shoulder pain as one diagnostic entity [13].

A second, increasingly recognized contributor is neuropathic. Zeilig and colleagues, using quantitative sensory testing, found sensory abnormalities consistent with a central or peripheral neuropathic component in a substantial subset of patients, distinct from those with purely mechanical pain and unlikely to respond to biomechanical interventions such as positioning or strapping alone [14]. This overlaps with central post-stroke pain; Klit and colleagues describe a syndrome arising from spinothalamic pathway lesions that can be difficult to distinguish from musculoskeletal shoulder pain, complicating diagnosis and trial interpretation when pain phenotype is not separately characterized [15]. Widar and colleagues similarly found that a meaningful proportion of persistent post-stroke pain had features inconsistent with a purely musculoskeletal origin, reinforcing the case for pain phenotyping over a uniform treatment algorithm [16]. Spasticity contributes through yet another pathway, producing pain via sustained muscle contraction, altered joint loading, and, in severe cases, contracture, a mechanism Bhakta situates within the broader upper motor neuron syndrome [17].

4. Multidisciplinary Assessment and Rehabilitation

Because post-stroke pain arises from overlapping biomechanical, neuropathic, and spasticity-related mechanisms, structured multidisciplinary assessment is now the recommended starting point in most rehabilitation guidelines. The Canadian Stroke Best Practice Recommendations describe a coordinated pathway in which physiotherapy, occupational therapy, rehabilitation medicine, and pain specialists jointly evaluate motor impairment, shoulder integrity, spasticity, and pain phenotype at defined intervals rather than relying on a single discipline [6]. Motor impairment is typically graded with the Fugl-Meyer-derived motor evaluation described by Demeurisse and colleagues [18], spasticity with the modified Ashworth scale despite its limited interrater reliability at intermediate grades [9], and pain intensity with the visual analogue or numerical rating scales validated by Jensen and colleagues as practical instruments for repeated assessment [19].

Turner-Stokes and colleagues' Cochrane review of multidisciplinary rehabilitation for acquired brain injury provides the clearest synthesis of why coordinated, goal-directed input outperforms single-discipline care for complex impairments including pain and spasticity, concluding that more intensive, better-coordinated programs improve functional outcomes even though individual component therapies show more modest isolated effects [20]. Occupational therapy contributes through activities-of-daily-living retraining and adaptive equipment that reduces mechanical strain on a painful or subluxed shoulder; Legg and colleagues' Cochrane review found consistent, if modest, benefits on activities of daily living that plausibly reduce this loading [21]. Pollock and colleagues' review of upper limb interventions similarly found task-specific and repetitive practice, delivered by coordinated physiotherapy and occupational therapy, improved arm function [22]. Winstein and colleagues' American Heart Association/American Stroke Association guideline formalizes this structure, recommending

routine screening for shoulder pain and spasticity throughout rehabilitation with coordinated specialist referral [23].

Table 1: Core components of multidisciplinary assessment for post-stroke hemiparetic pain, with the discipline responsible and the primary validated measure used at each stage [18,9,19,6].			
Domain	Responsible discipline	Primary measure	Typical assessment interval
Motor impairment	Physiotherapy / rehabilitation medicine	Fugl-Meyer-derived motor evaluation	Admission, 3, 6, 12 months
Spasticity	Physiotherapy / rehabilitation medicine	Modified Ashworth scale	Admission, 3, 6, 12 months
Pain intensity and phenotype	Rehabilitation medicine / pain specialist	Numerical rating or visual analogue scale	Weekly acute phase, then 3, 6, 12 months
Activities of daily living	Occupational therapy	Standardized ADL/functional index	Admission, 3, 6, 12 months

5. Pharmacological and Interventional Pain Management

Positioning, supportive devices, and strapping remain the first-line, lowest-risk interventions, though trial evidence is mixed. Ada and colleagues' Cochrane review found insufficient evidence that slings or supports prevent subluxation or reduce pain, and cautioned that some designs may restrict functional arm use [24]. Strapping has shown more consistent short-term benefit: Griffin and Bernhardt's trial found rigid strapping delayed pain onset compared with usual care, while Gustafsson and McKenna's trial of static positional stretching alone found no benefit, suggesting passive positioning without active correction is insufficient [25,26]. Anwer and Alghadir's trial combining taping with standard physical therapy found improved pain and reduced subluxation versus therapy alone [27].

Table 2: Pharmacological and interventional options for post-stroke hemiparetic shoulder pain by predominant pain mechanism, drawn from randomized controlled trials [28,29,30,31,33].

Predominant mechanism	Intervention	Trial evidence	Typical effect on pain
Spasticity-driven	Botulinum toxin type A injection	Placebo-controlled RCTs [28,29]	Significant reduction vs placebo
Spasticity-driven	Botulinum toxin vs corticosteroid	Comparative RCT [30]	Comparable or longer-lasting benefit with toxin
Mechanical / inflammatory	Intra-articular corticosteroid	Randomized trial vs placebo [31]	Modest, largely short-lived benefit
Neuropathic component	Suprascapular nerve block	Randomized controlled trial [33]	Clinically meaningful, sustained reduction

For pain attributable predominantly to spasticity, botulinum toxin injection has the strongest trial evidence. Yelnik and colleagues' placebo-controlled trial and Marco and colleagues' independent double-blind trial both found botulinum toxin type A significantly reduced spasticity-related shoulder pain [28,29]. Lim and colleagues found botulinum toxin at least comparable to intra-articular corticosteroid, with a more favorable duration in some patients, while Snels and colleagues found triamcinolone injection produced only a modest, short-lived benefit versus placebo [30,31]. Turner-Stokes and colleagues' goal attainment scaling analysis of a placebo-controlled toxin trial showed patient-centered functional goals, not spasticity or pain scores alone, improved meaningfully with treatment, reinforcing embedding pharmacological spasticity treatment within a multidisciplinary goal-setting framework [32]. For pain with a demonstrable neuropathic component, Adey-Wakeling and colleagues' trial of suprascapular nerve block found clinically meaningful pain reduction sustained through several weeks, an option for patients who do not respond to biomechanical or spasticity-targeted treatment alone [33].

6. Electrical Stimulation and Long-Term Outcomes

Neuromuscular electrical stimulation has been evaluated both as prevention and as treatment for established pain,

with results depending heavily on timing. Church and colleagues' trial of surface stimulation applied soon after acute stroke found reduced subsequent shoulder pain and subluxation, supporting early, protocol-driven use as part of a preventive bundle [34]. Price and Pandyan's Cochrane review reached a more cautious conclusion, finding pooled evidence insufficient to confirm benefit across stimulation protocols, with substantial heterogeneity across included trials [35]. Vuagnat and Chantraine's review situates functional electrical stimulation as one component among several, most useful when integrated with positioning, therapy, and pharmacological treatment [36]. Wilson and Chae's clinical review synthesizes this stepped approach: early biomechanical and stimulation-based prevention, spasticity-targeted pharmacological treatment when spasticity dominates, and interventional or neuropathic-directed treatment for non-responders [37].

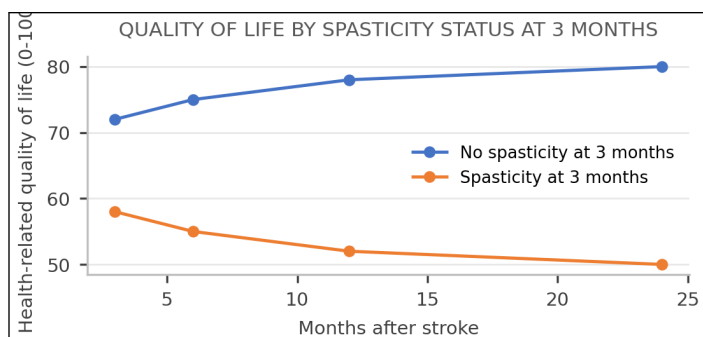


Figure 2: Health-related quality-of-life trajectory from three to twelve-plus months after stroke, stratified by presence of spasticity at three months, illustrating the persistent long-term impact of early spasticity on participation and wellbeing [38,39].

Long-term follow-up data, extending beyond the twelve-month horizon of most trials, remain comparatively sparse but converge on consistent findings. Gillard and colleagues' longitudinal cohort found spasticity at three months independently associated with worse quality of life at twelve months and beyond, after adjusting for baseline impairment [38]. Kalichman and Ratmansky note that patients whose pain is inadequately addressed early are disproportionately represented among those with chronic, refractory shoulder pain at

long-term follow-up [12]. Langhorne and colleagues' review of motor recovery observes that spontaneous recovery plateaus within three to six months, meaning pain and spasticity persisting beyond that window are unlikely to resolve without active, sustained management [39]. The long-term evidence argues for structured multidisciplinary follow-up continuing well past discharge, with periodic re-assessment of pain phenotype, spasticity, and shoulder integrity.

7. Conclusion

Pain in the hemiparetic limb, most often centered on the shoulder but frequently compounded by spasticity and, in a meaningful subset, by a neuropathic component, resists single-modality solutions and is best addressed through coordinated multidisciplinary care [5,13]. The evidence supports early biomechanical protection, preventive electrical stimulation where trial evidence allows, spasticity-targeted botulinum toxin, interventional options such as suprascapular nerve block for neuropathic pain, and structured occupational and physical therapy delivered as a coordinated program [28,33,34,21]. Long-term follow-up adds the recognition that pain and spasticity identified in the first months strongly predict pain and reduced quality of life a year or more later, so the model most likely to succeed treats pain management as a sustained, periodically re-assessed process spanning acute care, rehabilitation, and long-term community follow-up rather than a single unrevisited intervention [38,3,10].

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