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Central Post-Stroke Pain: Evidence-Based Rehabilitation Strategies for Neuropathic Pain Management

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ABSTRACT

Introduction: Central Post-Stroke Pain (CPSP) is a common and challenging condition that affects 8-10% of stroke survivors, significantly impacting their quality of life and functional recovery. Despite its prevalence, effective management remains complex and multifaceted.

Contents: This review explores rehabilitation strategies for CPSP, emphasizing the role of both pharmacological and non-pharmacological interventions. Exercise therapy, including strength training, aerobic conditioning, and flexibility exercises, plays a pivotal role in alleviating pain, enhancing neural plasticity, and improving functional outcomes. Additionally, physical modalities such as transcranial magnetic stimulation (rTMS) and acupuncture, along with psychological approaches like Cognitive Behavioral Therapy (CBT) and Acceptance and Commitment Therapy (ACT), address the emotional and cognitive components of CPSP.

Conclusion: A multidisciplinary, patient-centered approach integrating exercise, physical modalities, and psychosocial support is essential for optimizing CPSP management. Further research is needed to refine and standardize rehabilitation protocols to achieve long-term pain relief and improved recovery outcomes for stroke survivors.

Keywords: Exercise therapy; pain; transcranial magnetic stimulation

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Introduction

Stroke, a critical health emergency, remains a significant global burden. According to the World Health Organization (WHO), stroke is the second leading cause of death worldwide and the primary cause of long-term disability. Each year, over 13 million new stroke cases are reported, posing a serious challenge in providing effective and holistic care for stroke survivors (WHO, 2018). Amidst the evolving landscape of post-stroke care, one aspect that is often overlooked yet has a profound impact is central post-stroke pain (CPSP).

Epidemiological data reveal that CPSP, a chronic pain condition that emerges following a stroke, affects approximately 8-10% of the stroke population [1]. This high prevalence underscores the importance of understanding and effectively managing post-stroke pain, given its detrimental effects on quality of life, physical function, and psychological well-being.

Given these striking epidemiological findings, this review aims to explore in-depth the rehabilitation strategies for managing CPSP. To begin, it is essential to recognize that CPSP arises as a result of damage to the central nervous system, primarily caused by stroke. Stroke itself can result from various factors, including ruptured or blocked blood vessels, leading to insufficient blood supply to the brain. Advances in understanding the pathophysiology of stroke have provided further insights into the prevalence and risk factors associated with CPSP.

In the context of rehabilitation management, clinicians must prioritize accurate evaluation and diagnosis. Comprehensive clinical and neurological examinations are crucial to assess the extent of central nervous system damage and its impact on post-stroke pain. Additionally, advanced diagnostic tools, such as neuroimaging, offer deeper insights into the structural and functional changes in the brains of affected individuals. Rehabilitation approaches for CPSP extend beyond pharmacological management. Non-pharmacological therapies, including physiotherapy, occupational therapy, and cognitive therapy, play a pivotal role in restoring motor function, mitigating psychological impacts, and enhancing overall quality of life. Recent research also highlights the critical role of neuroplasticity in supporting rehabilitation and the brain's adaptation to injury.

Nevertheless, pharmacological treatment remains an integral component of CPSP management. Analgesics, antidepressants, and anticonvulsants are commonly used to address neuropathic pain and associated psychological symptoms. However, the choice of medication and dosage must be tailored to individual patient characteristics, with careful monitoring for potential side effects. In this context, a holistic approach that incorporates psychosocial support is equally indispensable. Family support, psychological counseling, and participation in support groups can provide the understanding, emotional reinforcement, and motivation necessary for a more effective recovery journey.

This review emphasizes the importance of a multidisciplinary and patient-centered approach to managing CPSP, integrating both pharmacological and non-pharmacological strategies to optimize outcomes

for stroke survivors.

Methods

This manuscript is a narrative review that summarizes current evidence regarding rehabilitation strategies for Central Post-Stroke Pain (CPSP). A literature search was conducted using PubMed, Scopus, ScienceDirect, and Google Scholar databases for articles published between 2009 and 2025. The search terms included "Central Post-Stroke Pain", "Stroke Rehabilitation", "Exercise Therapy", and "Neuropathic Pain". Original studies, randomized controlled trials, systematic reviews, meta-analyses, and clinical practice guidelines focusing on rehabilitation interventions for CPSP were included. Studies unrelated to rehabilitation management or addressing peripheral neuropathic pain were excluded. The selected literature was narratively synthesized according to rehabilitation modalities and their clinical applications.

Pain Physiology

Pain is a complex physiological process that serves as a protective mechanism, alerting the body to potential harm and enabling appropriate responses. The mechanism of pain involves three primary steps: (1) the detection of harmful stimuli by sensory receptors (transduction), (2) the transmission of electrical signals through neural pathways, and (3) the modulation of pain intensity at various levels of the nervous system. These processes collectively result in the perception of pain following the initiation and cessation of harmful stimuli.[2]

Nociceptive transduction is the process through which harmful stimuli are converted into electrical signals in neurons, starting with the transformation of physical stimuli into chemical signals. These signals are then turned into electrical impulses in sensory neurons, with further conversion into chemical messengers at the synaptic level. This process, essential for the body’s perception of touch and pain, occurs when the somatosensory system opens ion channels in response to physical stimuli, allowing electrochemical signals to be transmitted to higher neural centers. Sensory coding plays a key role in translating receptor signals into recognizable sensations by encoding four primary attributes of a stimulus: modality, location, intensity, and duration.[3] This allows the body to interpret sensory inputs and perceive the external environment. Neurons, which consist of soma, axons, and dendrites, form networks that transmit signals through synapses, either exciting or inhibiting neural activity depending on the chemical messengers involved. When neurons receive signals, they generate action potentials that travel along axons and activate synapses, transmitting messages to other neurons and facilitating communication between different regions of the brain and spinal cord.[4]

The somatosensory system detects a wide range of mechanical stimuli, from light touch to bladder distension, with mechanosensitive nerve fibers being crucial in detecting these stimuli. Nociceptors convert

harmful stimuli into electrical signals, triggering inflammatory responses and sensitization, which heighten swelling and pain in the affected area. Transmission of nociceptive signals occurs when primary nociceptors send signals to second-order neurons in the dorsal horn of the spinal cord, carried by Aδ and C fibers.[5] These fibers synapse with second-order neurons in laminae II and V, where nociceptive-specific (NS) neurons in lamina I respond to C fiber inputs and wide dynamic range (WDR) neurons in lamina V process a variety of stimuli. Pain modulation, which controls the transmission of pain signals, occurs at the brain, brainstem, and spinal cord levels, involving inhibitory control from higher centers and natural opioid and cannabinoid systems, among others. Finally, pain perception is shaped by the activation of a distributed cortical network known as the pain neuromatrix, with key regions such as the somatosensory cortex, insula, anterior cingulate cortex, prefrontal cortex, and amygdala working together to form the overall experience of pain.[2]

Sensitization Theory

The sensitization theory of pain explains that high-intensity, noxious stimuli, even if brief and without tissue damage, can activate nociceptors and induce pain. Small-diameter nerve fibers with high thresholds activate nociceptors, causing transient pain. In contrast, larger-diameter fibers with lower thresholds transmit sensations such as pressure and touch. These fibers communicate with other neurons in the spinal cord.[5]

Tissue injury and the release of inflammatory mediators increase nociceptor sensitivity, leading to repeated activation. This sensitization involves inflammatory mediators, which heighten nociceptor responsiveness and result in primary hyperalgesia—an increased response to both painful and non-painful stimuli. [5]

Persistent pain signals can enhance the sensitivity of spinal cord neurons, a phenomenon known as wind-up. This leads to secondary hyperalgesia and the spread of pain to surrounding areas. Inhibitory interneurons and descending inhibitory pathways help mitigate this sensitization. Analgesics and pain management strategies also play a role in reducing sensitization.[5]

Inflammatory mediators and pro-inflammatory cytokines, such as interleukin (IL)-1β and IL-6, exacerbate pain and inflammation. Effective treatment can reduce the levels of these cytokines. Genetic factors also influence individual responses to postoperative pain. [5]

Central Pain Syndrome Post Stroke

Central Pain Syndrome (CPS), initially introduced by Edinger in 1891, was notably elaborated by Déjerine and Roussy in their seminal 1906 work "Le syndrome thalamique". [6]They described severe, persistent, paroxysmal pain occurring on the hemiplegic side, unresponsive to analgesics, linked primarily to lesions in

the optic thalamus and adjacent posterior internal capsule. Subsequent studies, notably by Head and Holmes (1911) and Riddoch (1938), expanded understanding, revealing that central pain could result from lesions beyond the thalamus, thus leading to the contemporary preference for the term Central Post-Stroke Pain (CPSP). [7]

CPSP, categorized under central neuropathic pain disorders, arises directly from lesions or dysfunctions within the central nervous system (CNS).[8] Diagnosing CPSP remains challenging due to overlapping symptoms with other pain types post-stroke, including headaches, musculoskeletal pain, hemiplegic shoulder pain, and spasticity-related pain.[9] Current definitions emphasize the presence of neuropathic pain characteristics, sensory disturbances, confirmed CNS lesions, and the exclusion of other potential pain etiologies[10]

Epidemiologically, CPSP affects approximately 1% to 12% of stroke survivors, with higher prevalence (up to 18%) among those experiencing sensory deficits.[11] CPSP symptoms typically manifest as spontaneous dysesthesias or stimulus-triggered pain described as burning, freezing, stabbing, or squeezing sensations. Allodynia and hyperalgesia are also frequently reported, significantly impacting quality of life.[1]

Clinically, CPSP can result from lesions at various CNS locations, including the thalamus, lenticulocapsular region, cerebral cortex, pons, and medulla.[12] Thalamic lesions remain most commonly associated with CPSP, specifically involving the ventral posterolateral nucleus.[13] However, cortical, lenticulocapsular, pontine, and medullary strokes also contribute uniquely to CPSP presentations, influenced by lesion location and associated sensory pathway disruptions.[12]

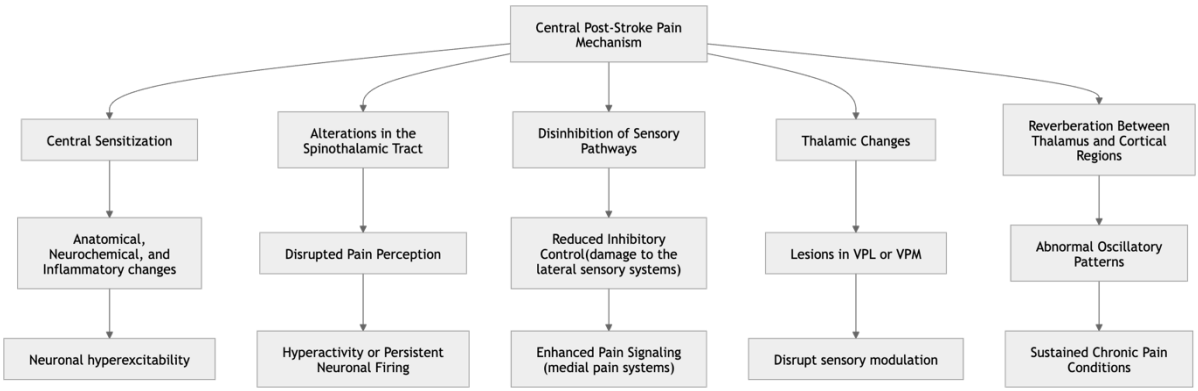

Figure 1 Mechanism of Central Post-Stroke Pain

The pathophysiological mechanisms underlying CPSP include central sensitization, alterations in spinothalamic tract function, disinhibition of sensory pathways, and specific thalamic changes.[1] Central sensitization involves anatomical, neurochemical, and inflammatory changes after CNS lesions, resulting in neuronal hyperexcitability. Alterations in the spinothalamic tract disrupt normal pain and temperature perception, potentially causing abnormal hyperactivity or persistent neuronal firing. Disinhibition theories

suggest reduced inhibitory control within sensory pathways due to damage to the lateral sensory systems, leading to enhanced pain signaling by medial pain systems.[14] Moreover, thalamic changes are significant, where lesions in nuclei like the ventral posterior lateral (VPL) or ventral posterior medial (VPM) can disrupt sensory modulation, leading to dysesthesia and central pain. Functional imaging studies show reduced regional cerebral blood flow in thalamic areas during spontaneous pain episodes and increased neuronal activity indicative of hyperexcitability. Additionally, abnormal oscillatory patterns between the thalamus and cortical regions, known as reverberation, have been implicated in sustaining chronic central pain conditions. [15]

Understanding these complex mechanisms is crucial for improving CPSP diagnosis, prognosis, and management strategies, thereby enhancing post-stroke patient outcomes and quality of life.

Management and Psychosocial Considerations of Central Post-Stroke Pain (CPSP)

Management of Central Post-Stroke Pain (CPSP) remains challenging due to its variable responsiveness to treatment and the complexity of associated symptoms, especially among elderly populations where medication side effects often limit therapeutic dosing. [16] Current clinical practices primarily involve trial-and-error pharmacological approaches, frequently employing combinations of medications, though few rigorous randomized controlled trials (RCTs) are available. Notably, a placebo-controlled trial investigating prophylactic amitriptyline (75 mg/day) following acute thalamic stroke demonstrated no significant preventive effects. [16]

Pharmacologically, antidepressants, particularly tricyclic antidepressants like amitriptyline, are considered first-line treatments for CPSP, effectively reducing pain severity. However, side effects such as sedation and dry mouth remain common. Serotonin-norepinephrine reuptake inhibitors (SNRIs) may offer a safer alternative, particularly for patients with cardiovascular risks, whereas selective serotonin reuptake inhibitors (SSRIs) generally appear less effective for neuropathic pain. [16] Anticonvulsants, including gabapentin and pregabalin, effectively reduce neuropathic pain by decreasing neuronal hyperactivity. Pregabalin, specifically, has shown substantial clinical benefit and acceptable tolerability. Lamotrigine offers moderate effectiveness but has inconsistent efficacy in central neuropathic conditions, whereas carbamazepine demonstrated minimal benefit for CPSP.[16] Opioids, although effective for neuropathic pain, are generally second-line therapies due to their side-effect profiles, withdrawal rates, and limited long-term effectiveness in CPSP populations.

Psychosocial factors, notably self-efficacy, psychological flexibility, and coping strategies, significantly impact CPSP management outcomes. Chronic post-stroke pain patients frequently demonstrate low self-efficacy, limited psychological flexibility, and maladaptive coping behaviors, negatively affecting

quality of life. Promising evidence suggests enhancing psychological flexibility and self-efficacy through motivational interviewing and structured psychological therapies can substantially improve chronic pain management outcomes in stroke survivors. However, further studies with larger sample sizes are necessary to comprehensively validate these psychosocial interventions and integrate them effectively into CPSP management protocols.[17]

Exercise Interventions in the Medical Rehabilitation of Central Post-Stroke Pain (CPSP)

Central Post-Stroke Pain (CPSP) presents significant rehabilitation challenges due to its persistent nature and impact on functional recovery. Exercise therapy, widely recognized as a cost-effective and accessible intervention, plays a crucial role in managing CPSP, enhancing functional capacity, and improving the overall quality of life of stroke survivors [18]

Exercise interventions for CPSP primarily involve strength training, aerobic conditioning, flexibility exercises, and innovative approaches like mirror therapy. Regular structured exercise has been shown to reduce pain intensity, enhance neural plasticity, modulate inflammatory processes, and ultimately improve neurological and functional outcomes post-stroke.[19]

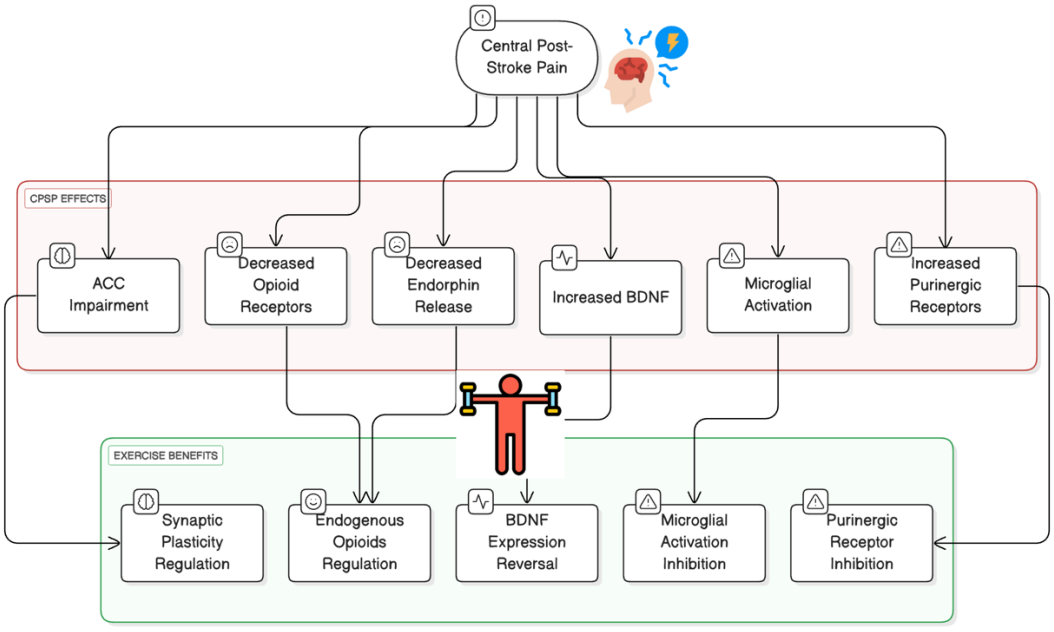

Figure 2. Exercise Mechanism to reduce Central pain post stroke

Strength training significantly alleviates CPSP by reducing muscle weakness, increasing pain thresholds, and minimizing pain sensitivity. Recommended protocols include resistance exercises targeting major muscle groups at intensities ranging from 30% to 80% of one repetition maximum (1-RM), performed two to three times per week.[20] A randomized clinical trial using structured band-resistance and range-of-motion

training demonstrated notable reductions in pain severity, highlighting the therapeutic value of progressive strength exercises.[21]

Aerobic training, an integral component of post-stroke rehabilitation, effectively manages CPSP through cardiovascular health enhancement and analgesic mechanisms, including modulation of central sensitization and inflammatory cytokines.[22] Standard aerobic exercise guidelines recommend moderate-intensity sessions (40%–59% of heart rate reserve), performed 20–40 minutes per session, three to five times weekly, progressively increasing intensity and duration according to individual patient tolerance.[23]

Flexibility exercises, particularly stretching and therapeutic yoga, address musculoskeletal complications frequently seen post-stroke, such as muscle spasms, joint contractures, and limited range of motion. Studies by Schmid et al. (2014) indicate that an eight-week structured yoga intervention significantly decreases CPSP severity and improves patient-reported quality of life.[24] Flexibility exercises should ideally be integrated into daily routines to maintain muscle length, joint mobility, and functional independence.[25]

Mirror therapy, another innovative exercise intervention, utilizes visual feedback mechanisms to correct maladaptive somatosensory cortex reorganization associated with CPSP. This therapy improves sensory-motor congruence, reducing the perception of pain through cortical recalibration and restoration of sensory integration. [26]

Neurobiologically, exercise interventions stimulate neural adaptations critical for pain modulation. Exercise-induced enhancements in brain-derived neurotrophic factor (BDNF), endogenous opioid systems, inhibition of microglial activation, and regulation of neuroinflammatory markers have been identified as potential analgesic mechanisms. These adaptations collectively foster neural plasticity, reduce central sensitization, and improve functional recovery.[22]

Complementing exercise, neuromodulation techniques have emerged as promising adjunctive therapies for CPSP. High-frequency repetitive transcranial magnetic stimulation (rTMS), particularly when applied over the primary motor cortex (M1), is currently the most extensively studied non-invasive brain stimulation technique. Randomized controlled trials have demonstrated significant reductions in pain intensity immediately after treatment, with analgesic effects persisting for approximately two to four weeks. The proposed mechanism involves modulation of cortical excitability, restoration of thalamocortical connectivity, and activation of descending inhibitory pain pathways. Although the short-term efficacy of rTMS is well established, maintenance protocols and optimal stimulation parameters remain under investigation. [27] Transcranial direct current stimulation (tDCS) is another non-invasive neuromodulation technique that delivers low-intensity electrical currents to modulate cortical activity. Several clinical studies have demonstrated

reductions in pain intensity and improvements in sensory discrimination following stimulation of the primary motor cortex. Compared with rTMS, tDCS is less expensive, portable, and easier to implement in clinical settings, although its analgesic effects appear to be smaller and evidence remains limited.[28] Deep Brain Stimulation (DBS) represents an invasive neurosurgical option reserved for patients with refractory CPSP who fail conventional pharmacological and rehabilitation treatments. DBS commonly targets the periventricular/periaqueductal gray matter or sensory thalamic nuclei to modulate abnormal pain processing. Although several observational studies have reported clinically meaningful pain relief, current evidence is insufficient to recommend DBS as routine treatment because of limited randomized controlled trials and potential surgical risks. [29] Acupuncture and electroacupuncture, derived from traditional Chinese medicine, have also demonstrated comparable analgesic effectiveness to pharmacological approaches, providing valuable adjunctive treatment strategies.

Psychosocial interventions, including Cognitive Behavioral Therapy (CBT) and Acceptance and Commitment Therapy (ACT), are pivotal components of comprehensive CPSP rehabilitation. CBT helps patients reduce pain perception and improve psychological adjustment through structured skill-building techniques such as cognitive restructuring, relaxation, and activity pacing.[30] ACT further enhances coping mechanisms by promoting psychological flexibility, mindfulness, and cognitive defusion, facilitating better acceptance and management of chronic pain experiences, thus improving overall quality of life.[31]

Implementing an individualized, multidisciplinary rehabilitation strategy that integrates structured exercise, targeted physical modalities, and tailored psychosocial interventions is essential to optimize pain management, enhance functional independence, and significantly improve the long-term outcomes of CPSP patients. Continued rigorous research is warranted to refine these integrated rehabilitation approaches and establish standardized, evidence-based protocols for CPSP management.

Conclusion

Rehabilitation for Central Post-Stroke Pain (CPSP) requires a comprehensive, multidisciplinary approach due to its complex nature and significant impact on quality of life. Structured physical exercise remains a key component of CPSP management, as it improves pain, mobility, muscle strength, and functional recovery when tailored to individual patient needs. Current evidence suggests that progressive resistance and aerobic exercise performed for approximately 8–12 weeks provide the most consistent clinical benefits.

Psychosocial interventions, including Cognitive Behavioral Therapy (CBT) and Acceptance and Commitment Therapy (ACT), together with patient education, are essential for improving coping strategies, treatment adherence, and self-management. Ongoing monitoring and collaboration among healthcare providers, patients, and families are also crucial to optimize rehabilitation outcomes.

Among adjunctive rehabilitation modalities, repetitive transcranial magnetic stimulation (rTMS) has the strongest evidence for short-term pain relief, whereas transcranial direct current stimulation (tDCS) may be considered an alternative non-invasive option. Deep Brain Stimulation (DBS) should be reserved for carefully selected patients with refractory CPSP because of its invasive nature and limited clinical evidence. Future high-quality studies are needed to establish standardized rehabilitation protocols regarding treatment intensity, duration, and long-term effectiveness.

Conflict of Interest

The author declares that there is no conflict of interest related to the content of this manuscript.

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