Vestibular Modulation of Pain: A Review of Neuroanatomical Pathways and Mechanisms

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Highlights:

- Vestibular pathways interact with pain circuits at multiple CNS levels
- Thalamic vestibular projections may integrate with nociceptive signals
- Vestibular pathways may modulate pain via PAG–RVM and predictive coding

Abstract

Background and Aim: Chronic pain remains a major clinical challenge, with many patients experiencing insufficient relief from conventional therapies. Emerging evidence suggests functional overlap between vestibular networks and central pain-processing systems. This narrative review aimed to evaluate the potential of vestibular stimulation to modulate nociceptive processing and influence pain perception. Relevant literature was identified through PubMed, Scopus, and Web of Science searches, and studies addressing vestibular–pain interactions were narratively reviewed.

Recent Findings: Clinical observations indicate that cold caloric vestibular stimulation may transiently alleviate various forms of central and neuropathic pain, including central post-stroke pain, phantom limb pain, spinal cord injury–related pain, and complex regional pain syndrome. Small randomized and quasi-experimental trials provide partial support for these findings, although reported effects are generally short-lived and heterogeneous across studies. Laboratory-based psychophysical experiments further suggest that vestibular input can alter pain thresholds and sensory integration processes. Functional neuroimaging studies implicate vestibulo–thalamo–insular and vestibulo–limbic circuits, highlighting shared neural pathways involved in both vestibular and nociceptive processing. Collectively, the evidence suggests that vestibular stimulation may influence both the sensory-discriminative and affective-motivational dimensions of pain.

Conclusion: Vestibular stimulation may represent a promising non-invasive approach for modulating pain perception through central multisensory integration mechanisms. Current evidence suggests potential analgesic effects; however, findings remain heterogeneous and the durability of these effects is not yet well established. Further large, well-designed randomized controlled trials are needed to determine clinical efficacy, optimal stimulation parameters, and long-term outcomes before routine clinical implementation can be considered.

Keywords: Vestibular stimulation; caloric vestibular stimulation; galvanic vestibular stimulation; pain modulation; pain perception

Introduction

Chronic pain is a major global health problem affecting hundreds of millions of individuals worldwide and represents one of the leading causes of long-term disability. Beyond its direct effects on physical functioning, chronic pain is associated with psychological distress, impaired social participation, reduced quality of life, and substantial socioeconomic costs resulting from healthcare utilization and productivity loss. Despite advances in pharmacological, behavioral, and interventional pain management strategies, many patients continue to experience inadequate symptom control or treatment-related adverse effects. Consequently, there is increasing interest in identifying novel, non-invasive approaches capable of modulating pain processing through central nervous system mechanisms [1, 2]. The vestibular system, located within the inner ear, comprises three semicircular canals and two otolith organs (the utricle and saccule), which detect angular and linear acceleration of the head and contribute to balance, gaze stabilization, spatial orientation, and postural control [3]. Vestibular information is transmitted from hair cells to the vestibular nuclei through the eighth cranial nerve and subsequently distributed through extensive multisynaptic projections to the cerebellum, thalamus, cortex, brainstem, and spinal cord [4-6]. These widespread connections position the vestibular system as an important hub for multisensory integration, enabling interactions with visual, somatosensory, proprioceptive, autonomic, and cognitive networks.

Increasing evidence suggests that vestibular processing extends beyond its traditional role in balance and spatial orientation. In recent years, the vestibular system has been implicated in a variety of higher-order functions, including body representation, emotional regulation, interoception, spatial cognition, and perception of self-motion. Such findings have prompted growing interest in the potential contribution of vestibular networks to pain processing. Pain is now recognized as a multidimensional experience influenced not only by peripheral nociceptive input but also by cognitive, emotional, attentional, and multisensory factors [7]. Accordingly, neural systems involved in sensory integration may play an important role in shaping pain perception [8]. Several anatomical and functional observations support the existence of vestibular–pain interactions. Vestibular projections reach numerous regions involved in nociceptive processing, including the thalamus, insular cortex, anterior cingulate cortex, somatosensory cortices, amygdala, and other limbic structures [9, 10]. These regions contribute to sensory-discriminative, affective, and cognitive dimensions of pain perception. Neuroimaging studies have demonstrated that Caloric Vestibular Stimulation (CVS) can alter functional connectivity within thalamo-insular networks in individuals with central pain syndromes[11-14]. Clinical reports have further suggested that vestibular stimulation may reduce symptoms such as tactile allodynia, central post-stroke pain, and phantom limb pain in selected patient populations [15]. Experimental investigations in healthy participants provide additional evidence for vestibular modulation of nociceptive processing. Galvanic Vestibular Stimulation (GVS) has been associated with reductions in nociceptive evoked potentials and increases in thermal pain thresholds, suggesting that vestibular activation may influence central pain processing mechanisms [16-18]. Similarly, a virtual-reality study showed that visual stimuli carrying vestibular information can modulate pain perception, suggesting that vestibular influences on pain may involve broader multisensory integration processes rather than a single dedicated pathway [19].

Several neurobiological mechanisms have been proposed to explain these observations. Ascending vestibular projections may influence nociceptive processing through interactions with thalamic, insular, cingulate, and limbic networks involved

in sensory integration and salience detection. Vestibular signals may also alter attentional allocation, body representation, predictive coding processes, and interoceptive awareness, all of which are known to influence pain perception [20, 21]. In addition to these ascending mechanisms, growing evidence suggests that vestibular inputs may interact with descending pain modulatory systems. Anatomical and experimental studies indicate potential connections between vestibular nuclei and brainstem structures involved in endogenous pain control, including the Periaqueductal Gray (PAG), parabrachial complex, locus coeruleus, and Rostral Ventromedial Medulla (RVM). Through these networks, vestibular activation may influence descending inhibitory or facilitatory pathways that regulate spinal nociceptive transmission, although the precise mechanisms remain incompletely understood [21]. Evidence from animal studies further supports a functional relationship between vestibular and nociceptive systems. Vestibular lesions have been shown to alter baseline nociceptive thresholds, modify responses of wide dynamic range neurons, and influence behavioral responses to painful stimuli [20, 22]. These findings suggest that vestibular input may contribute to tonic regulation of nociceptive processing and maintenance of pain homeostasis. Despite increasing interest in this field, important gaps remain in the current literature. Existing studies vary substantially with respect to stimulation modality, stimulation parameters, study populations, outcome measures, and methodological quality. Furthermore, the relative contribution of ascending multisensory pathways, vestibulo–limbic interactions, and descending pain modulatory systems remains unclear. The clinical significance, durability of treatment effects, and optimal parameters for vestibular stimulation have also not been established. As a result, current evidence remains fragmented and difficult to integrate into a coherent mechanistic framework. Accordingly, the present review aims to synthesize current evidence regarding the neuroanatomical pathways and mechanisms linking vestibular and pain systems. Particular emphasis is placed on vestibulo–thalamo–cortical networks, vestibulo–limbic interactions, multisensory integration processes, and potential engagement of descending pain modulatory pathways. By examining both experimental and clinical evidence, this review sought to identify areas of consensus and uncertainty, clarify the mechanisms through which vestibular stimulation may influence pain perception, and highlight priorities for future research and clinical translation.

Method

This narrative review synthesized evidence examining the effects of vestibular stimulation and vestibular function on pain perception, nociceptive processing, and pain modulation mechanisms. A structured literature search was conducted in PubMed, Scopus, and Web of Science for studies published between 2000 and March 2025. Additional relevant studies were identified through manual screening of reference lists from eligible articles.

The search strategy combined vestibular- and pain-related terms using Boolean operators. Search terms included "vestibular stimulation" OR "vestibular system" OR "galvanic vestibular stimulation" OR "GVS" OR "noisy galvanic vestibular stimulation" OR "nGVS" OR "caloric vestibular stimulation" OR "CVS" OR "rotational vestibular stimulation" AND "pain" OR "nociception" OR "nociceptive processing" OR "pain perception" OR "analgesia" OR "chronic pain". Relevant MeSH terms were used in PubMed when applicable. Studies were eligible for inclusion if they investigated vestibular stimulation or vestibular function in relation to pain perception, nociceptive processing, or pain modulation. Both human and animal studies were considered. Eligible study designs included experimental studies, exploratory clinical trials, observational studies, and case reports. Animal studies were included when they provided mechanistic, neurophysiological, or behavioral evidence relevant to vestibular–nociceptive interactions. Conference abstracts, editorials, letters, non-English publications, articles without accessible full text, and narrative reviews were excluded. The search identified 48 records.

After removal of 9 duplicate records, 39 records underwent title and abstract screening. Eleven records were excluded because they were not relevant to the review topic, were not published in English, or lacked accessible full-text versions. 28 full-text articles were assessed for eligibility. Following full-text review, 12 articles were excluded because they did not meet the predefined inclusion criteria. Ultimately, 16 studies were included in the final synthesis. The study selection process is illustrated in Figure 1. Given the substantial heterogeneity in study populations, vestibular stimulation modalities, outcome measures, and methodological approaches, quantitative meta-analysis was not feasible. Findings were therefore synthesized narratively and organized according to intervention type, including CVS, GVS, nGVS, rotational vestibular stimulation, and visual–vestibular illusion paradigms. Clinical and experimental evidence were reviewed separately where appropriate. Neuroimaging and neurophysiological findings were additionally summarized to highlight potential mechanisms underlying vestibular–nociceptive interactions. The preparation and reporting of this narrative review were informed by the Scale for the Assessment of Narrative Review Articles (SANRA) recommendations to enhance methodological transparency and reporting quality.

Discussion

The available evidence collectively suggests that vestibular stimulation may influence pain perception and nociceptive processing across both clinical and experimental settings. Findings from case reports, small case series, animal studies, and controlled laboratory experiments indicate that modulation of vestibular inputs can be associated with measurable changes in pain-related outcomes. Among the various stimulation modalities, the most consistent evidence has been reported for cold CVS in central neuropathic pain conditions, including Central Post-Stroke Pain (CPSP), Spinal Cord Injury (SCI)-related pain, phantom limb pain, and Complex Regional Pain Syndrome (CRPS), where transient reductions in spontaneous pain intensity, allodynia, or pain unpleasantness have been described [12, 23]. Complementary evidence from experimental studies in healthy participants suggests that GVS and visual–vestibular manipulations may alter pain thresholds and modulate nociceptive cortical responses, further supporting a potential interaction between vestibular and pain-processing systems [17, 19]. The mechanisms underlying these effects remain incompletely understood. Current evidence points toward the involvement of distributed neural networks linking vestibular, sensory, affective, and autonomic systems. In particular, vestibulo–thalamo–cortical, vestibulo–limbic, and brainstem pathways have emerged as plausible substrates through which vestibular signals may influence pain perception [24]. Neuroimaging, neurophysiological, and anatomical studies provide converging support for these interactions; however, the available evidence remains largely indirect. Consequently, proposed mechanisms should be interpreted as biologically plausible hypotheses rather than established causal pathways. Further experimental studies are required to determine whether these neural interactions directly mediate the analgesic effects observed following vestibular stimulation [12, 19, 25].

Vestibular stimulation has also been discussed within the broader context of non-invasive neuromodulation. Conceptually, it shares certain features with other neuromodulatory approaches, including repetitive Transcranial Magnetic Stimulation (rTMS), transcranial Direct Current Stimulation (tDCS), Spinal Cord Stimulation (SCS), and Deep Brain Stimulation (DBS), all of which aim to modify central nervous system activity to influence pain perception [21, 26]. However, direct comparative studies are currently unavailable, precluding meaningful conclusions regarding relative efficacy, mechanisms of action, cost-effectiveness, or clinical utility. Any comparison between vestibular stimulation and these established techniques should therefore be regarded as tentative and hypothesis-generating rather than evidence-based.

Several important limitations of the current evidence base should also be considered. First, many of the available studies involve small sample sizes, limiting statistical power and increasing susceptibility to both type I and type II errors. Second, a substantial proportion of the literature consists of case reports, case series, and exploratory investigations, which are inherently vulnerable to selection bias and publication bias. Third, follow-up periods are typically short, making it difficult to determine whether reported analgesic effects are transient or clinically durable. Additional challenges arise from considerable heterogeneity across studies in terms of patient populations, stimulation modalities, stimulation parameters, outcome measures, and methodological quality. This variability complicates direct comparison between studies and limits the ability to draw firm conclusions regarding optimal treatment protocols or patient selection criteria.

Taken together, the current literature provides encouraging preliminary evidence that vestibular stimulation may influence pain perception through multisensory and central neuromodulatory mechanisms. Nevertheless, the evidence remains heterogeneous, and many mechanistic and clinical questions remain unresolved. Future research should prioritize adequately powered randomized controlled trials, standardized stimulation protocols, longer follow-up periods, and mechanistic investigations capable of distinguishing causal effects from associative findings. Such efforts will be essential for determining the clinical relevance, therapeutic durability, and translational potential of vestibular-based approaches to pain modulation. Table 1 summarizes the major vestibular stimulation modalities, stimulation parameters, proposed mechanisms, and reported analgesic effects. Table 2 provides a structured overview of the principal neuroanatomical, neuroimaging, experimental, and clinical evidence supporting the role of the vestibular system in pain modulation and nociceptive processing.

Condition-specific patterns

In this section we summarize observations linking vestibular perturbations to analgesic responses across discrete clinical syndromes. By "condition-specific patterns" we mean: 1) the clinical phenotypes in which analgesia has been reported (for example, CPSP, spinal cord injury-related pain, phantom limb pain, and CRPS; 2) characteristic response features such as onset, duration and laterality (i.e. whether effects are ipsi- or contralateral to stimulation); and 3) mechanistic signatures detected using neuroimaging or neurophysiology (for example, altered thalamo-insular connectivity). Defining these dimensions up front helps delineate which patient groups may be most likely to benefit from vestibular neuromodulation and which mechanistic measures are most informative. In CPSP, relief after CVS often coincided with normalization of thalamus–insula connectivity, suggesting a network-level reset [15]. In CRPS, the analgesic effects may be mediated through modulation of abnormal body schema and multisensory integration [12]. Phantom limb pain cases showed both analgesia and alteration of phantom limb position or movement, pointing to vestibular influence on body ownership representation [11]. Experimental pain studies revealed that GVS polarity and visual–vestibular congruence can produce lateralized modulation of thresholds, implying hemispheric or directional tuning in vestibulo–nociceptive circuits.

Neurophysiological mechanisms

The vestibular system comprises the semicircular canals and otolith organs (utricle and saccule), whose hair cells transduce head motion and gravitational forces. Afferent signals travel via the vestibular nerve to the vestibular nuclei in the medulla and pons. An overview of these pathways is presented in Figure 2. These nuclei have widespread projections: 1)ascending projections to the thalamus, particularly the Ventro-Posterior Lateral (VPL), Ventro-Posterior Inferior (VPI), and intralaminar nuclei, which also receive nociceptive input from the spinothalamic tract. 2) projections to the cerebellum,

especially the nodulus and uvula, which contribute to sensorimotor integration and could indirectly influence pain perception via cerebello-thalamo-cortical loop 3) connections to brainstem reticular formation and PAG, key nodes in descending pain inhibition [9]. and 4) projections to limbic and paralimbic cortices, particularly the posterior insula, parietal operculum, and anterior cingulate, via the thalamus [11].

Brainstem integrating with nociceptive systems

The vestibular nuclei and the spinal trigeminal nucleus share reciprocal connections. Vestibular input can modulate Wide Dynamic Range (WDR) neurons in the dorsal horn through multisynaptic brainstem relays. Experimental lesion studies show that disrupting vestibular nuclei alters nociceptive reflex thresholds, supporting a modulatory role [15].

Thalamic convergence and gating

The VPL and VPI thalamic nuclei act as convergence points for vestibular and nociceptive afferents. In functional Magnetic Resonance Imaging (MRI), CVS alters activity in these nuclei, coinciding with analgesic reports in CPSP [15]. The intralaminar nuclei (centromedian–parafascicular complex) are also implicated in pain perception and attentional salience; vestibular modulation here could alter pain salience rather than primary intensity.

Insular and salience network involvement

The posterior insula encodes multimodal interoceptive inputs, including vestibular and nociceptive signals [27]. Vestibular activation may influence pain by altering integration within the insula–anterior cingulate–prefrontal network, shifting attention and emotional appraisal of pain. Sudden vestibular perturbations activate the salience network (anterior insula and dorsal anterior cingulate), which can down-regulate ongoing nociceptive processing.

Descending pain control

Vestibular nuclei project to the PAG and RVM, both central to descending inhibitory control. Electrical stimulation of vestibular nuclei in animal models reduces dorsal horn neuron excitability and increases tail-flick latency (an index of nociceptive threshold) [15]. CVS and GVS in humans may recruit similar pathways, releasing endogenous opioids, serotonin, and norepinephrine into the spinal cord dorsal horn [9].

Neurochemical mediators

Vestibular stimulation can alter firing in serotonergic neurons in the dorsal raphe and noradrenergic neurons in the locus coeruleus, both of which project to pain-modulatory circuits in the spinal cord and brainstem. These monoaminergic effects could explain transient analgesia and modulation of pain-related evoked potentials after GVS [17].

Predictive coding and body schema recalibration

From a computational neuroscience perspective, pain is influenced by predictions about bodily state. Vestibular perturbations introduce sensory prediction errors in body orientation and movement. This may force the brain to update its body schema, which can indirectly alter the weighting of nociceptive input (predictive coding framework). Such recalibration is evident in phantom limb patients, where CVS changes perceived limb position and reduces pain [11, 20].

Clinical Implications

As detailed in Table 1, cold caloric vestibular stimulation demonstrates the most consistent and reproducible acute analgesic effects, particularly in central neuropathic pain conditions. Vestibular stimulation—particularly cold CVS—holds promise as a low-cost, non-invasive adjunct for neuropathic pain conditions such as CPSP, CRPS, and phantom limb pain. Its rapid, bedside applicability and absence of surgical risk make CVS especially attractive in clinical settings. GVS and nGVS may offer home-based treatment potential, pending standardized safety and sham protocols. Vestibular approaches may be

especially beneficial when combined with rehabilitation methods targeting body representation, such as mirror therapy, graded motor imagery, or cognitive behavioral therapy, due to their capacity to recalibrate distorted body schema [12, 19].

Conclusion

In summary, preliminary evidence suggests that vestibular stimulation, including Caloric Vestibular Stimulation (CVS), Galvanic Vestibular Stimulation (GVS), and visual–vestibular illusion paradigms, may influence pain perception in both clinical and experimental settings. Although several studies have reported acute analgesic effects, the current evidence base is limited by small sample sizes, methodological heterogeneity, and predominantly short-term outcome assessments. While converging clinical, behavioral, and mechanistic findings indicate a potential interaction between vestibular and pain-processing systems, the underlying mechanisms and clinical significance of these effects remain incompletely understood. Consequently, vestibular stimulation should currently be regarded as a promising but still investigational approach to pain modulation rather than an established therapeutic intervention. Further well-designed studies are needed to clarify its efficacy, durability of effects, and potential role in pain management, particularly in patients with refractory neuropathic pain conditions.

Future directions

Current evidence suggests that acute analgesic responses to vestibular perturbation may be more likely in central neuropathic pain syndromes, particularly CPSP and in disorders involving altered body representation, such as phantom limb pain and CRPS. These conditions are often associated with central sensitization and dysfunction within thalamo-cortical or thalamo-insular networks. Although these observations remain preliminary, future studies may benefit from exploring whether pain phenotype (e.g. central neuropathic, peripheral neuropathic or nociceptive pain) , disturbances in body representation (phantom sensations, proprioceptive mismatch or neglect-like symptoms), neurophysiological or neuroimaging markers including altered thalamo-insular connectivity or abnormal nociceptive evoked potentials and prior responsiveness to neuromodulation influence treatment outcomes. Incorporating mechanistic outcome measures, such as functional connectivity analyses, evoked potentials, and quantitative sensory testing, may further clarify the biological basis of treatment response and support the development of more targeted patient-selection strategies.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest

The author declares no conflicts of interest related to this work.

Human and Animal Rights

All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards, including the Declaration of Helsinki and its later amendments, as well as national and institutional research com

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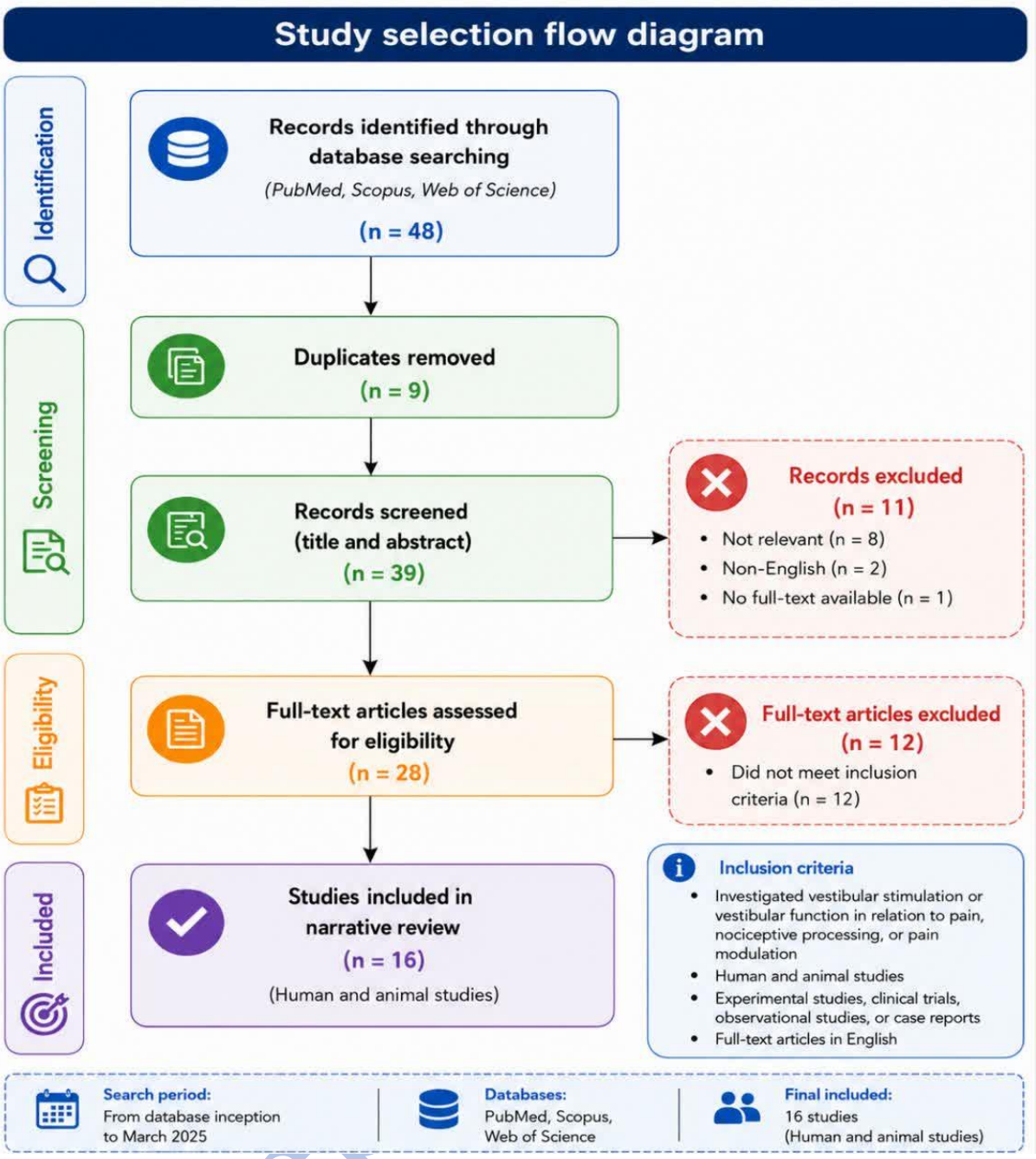

Figure 1. Flow diagram of the study selection process.

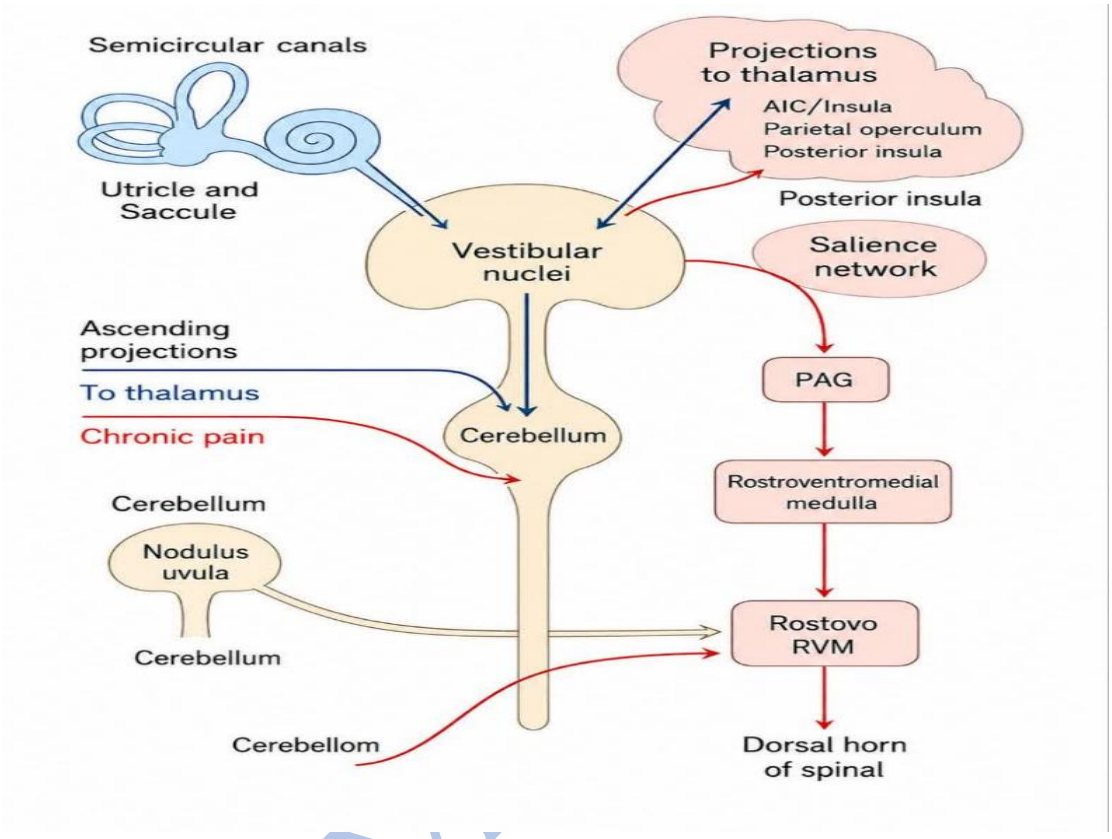

Figure 2. Neurophysiological pathways linking the vestibular system to pain modulation. Schematic representation of vestibula r inputs from the semicircular canals and otolith organs projecting to vestibular nuclei, with ascending pathways to the thalamus, cerebellum, and cortical regions, and descending pathways to the PAG–RVM–spinal cord system. Blue lines indicate acute pain mechanisms; red lines indicate chronic pain mechanisms. The figure was created by the author.

Table 1. Vestibular stimulation modalities, parameters and their reported effects on pain processing

Representative studies	Publication Year	Modality	Typical parameters	Proposed mechanisms*	Reported pain effects
Spitoni et al. [15]; McGeoch and Ramachandran [25] ; LeChapelain et al. [24]; Ngo et al. [12]	2016, 2008,2001,2024	Cold caloric vestibular stimulation	Air −4°C or water 30°C; 40–60 s; unilateral irrigation; supine with head elevated ~30°	Activation of vestibular afferents; modulation of thalamocortical and multisensory processing	Transient reduction of pain reported in central post-stroke pain, phantom limb pain, CRPS, and SCI-related neuropathic pain; effects generally last minutes to <1 h
McGeoch et al. [23]; McGeoch and Ramachandran [25] ; LeChapelain et al. [24]	2008,2008,2001	Warm caloric vestibular stimulation (CVS)	Air/water 40–44°C; 40–60 s; unilateral irrigation	Activation of vestibular afferents with opposite endolymphatic flow compared with cold CVS; possible multisensory reweighting	Analgesic effects reported in some studies, but evidence is limited and less consistent than for cold CVS
Hagiwara et al. [17]	2020	Galvanic vestibular stimulation (GVS)	Binaural bipolar stimulation; typically 1–2 mA; 10–90 s stimulation blocks	Modulation of vestibular nerve activity and vestibulo-thalamo-cortical networks; possible engagement of descending pain modulatory systems	Alterations in pain thresholds and reductions in nociceptive cortical responses reported in experimental studies
Daniel et al. [19]	2020	Visual–vestibular stimulation / vestibular illusions	Optic-flow, vection, or virtual-reality paradigms; typically 1–5 min	Multisensory integration, predictive coding, and body-schema recalibration	Short-lasting changes in thermal pain thresholds reported; evidence remains preliminary

Abbreviations: CVS, caloric vestibular stimulation; GVS, galvanic vestibular stimulation; CRPS, complex regional pain syndrome; SCI, spinal cord injury.

* Proposed mechanisms reflect current hypotheses derived from experimental and neuroimaging studies and should not be interpreted as definitively established causal pathways.

Table 2. Key evidence supporting vestibular modulation of pain

Study	Publication Year	Study type	Population	Vestibular intervention	Main findings	Proposed mechanism
Balaban [9]	2004	Neuroanatomical study	Animal models	Vestibular pathway tracing	Vestibular nuclei project to parabrachial and autonomic regulatory centers	Anatomical substrate linking vestibular, autonomic, andpain-related networks
Lopez et al. [11]	2012	ALE meta-analysis	Human neuroimaging studies	Vestibular stimulation	Identified a core vestibular cortical network involving the posterior insula, parietal operculum, and temporo-parietal junction	Cortical overlap between vestibular and pain-processing regions
Le Chapelain et al. [24]	2001	Clinical observational study	Paraplegic patients	Cold CVS	Induced phantom body sensations and altered body representation	Recalibration of body schema through multisensory integration
McGeoch et al. [23]	2008	Clinical proof-of-concept study	Central post-stroke pain patients	Cold CVS	Significant transient reduction in pain intensity following vestibular stimulation	Thalamic sensory reweighting and multisensory integration
McGeoch and Ramachandran [25]	2008	Case report	Spinal cord injury pain	Cold CVS	Reduction of chronic neuropathic pain symptoms	Vestibulo-thalamic modulation of pain processing
Ferre et al. [16]	2015	Experimental neurophysiology study	Healthy adults	CVS	Reduced nociceptive evoked potentials following vestibular stimulation	Cortical inhibition of nociceptive processing
Spitoni et al. [15]	2016	Single-case study with fMRI	Severe central post-stroke pain	Cold CVS	Immediate reduction in pain and somatoparaphrenia accompanied by changes in functional connectivity	Thalamic-insular-cingulate modulation and restoration of body representation
Hagiwara et al. [17]	2020	Experimental neurophysiology study	Healthy adults	GVS	Attenuated laser-evoked cortical responses and increased pain thresholds	Modulation of cortical nociceptive processing and activation of descending inhibitory pathways
Habig et al. [20]	2023	Functional neuroimaging study	Healthy adults	Vestibular and nociceptive stimulation	Overlapping thalamic activation patterns during vestibular and painful stimulation	Shared thalamic processing of vestibular and nociceptive information
Bouisset et al. [21]	2025	Integrative review	Human and clinical studies	CVS and GVS	Evidence supports vestibular influences on pain perception, embodiment, and multisensory integration	Salience-network modulation and vestibular-pain network interactions

Abbreviations: ALE, activation likelihood estimation; CVS, caloric vestibular stimulation; fMRI, functional magnetic resonance imaging; GVS, galvanic vestibular stimulation