

Cervicogenic Dizziness: **A Vestibular Clinician's Deep Review of Mechanism, Diagnosis and Management**

Vestibular Medicine for Vestibular Physicians

Systemic and Multisensory Balance Disorders — Module 4.6

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and senior vestibular physiotherapists working at the deep end of systemic and multisensory balance practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

- Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.
- Clinical Insight:** Clinically relevant observations for direct application in assessment and management.
- Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.
- Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Cervicogenic dizziness (CGD) is a clinical syndrome in which non-vertiginous dizziness, unsteadiness or spatial disorientation is generated by disordered afferent input from the cervical spine, occurring in temporal and mechanical association with neck pain or dysfunction [1,4,5]. The term — used interchangeably with proprioceptive cervical dizziness — denotes the proprioceptive subtype that is the focus of this review, as distinct from vascular or bony compressive mechanisms that are far rarer [5,13]. First framed by Ryan and Cope and refined by Wrisley and colleagues, the entity has remained both clinically familiar and scientifically contested because there is no confirmatory test and the diagnosis rests on exclusion plus a supportive cervical examination [1,4,10].

Dizziness and vertigo affect up to a fifth of adults each year, and neck pain is one of the most prevalent musculoskeletal complaints worldwide, so the two symptoms coincide frequently by chance alone [12,39]. This base-rate overlap is the central epidemiological problem of CGD: co-occurrence of neck pain and dizziness does not establish causation, and neither neck pain nor demonstrable cervical dysfunction is sufficient to implicate the neck as the driver of dizziness [7,9]. Precise prevalence figures are therefore unreliable, with estimates varying widely according to the diagnostic threshold applied and the population studied [10,12]. The condition is reported more often in people with chronic mechanical neck pain, in older adults with degenerative cervical change, and after whiplash and concussion, where altered cervical afferentation is biologically plausible and frequently measurable [5,35].

Rather than asking whether a patient simply has or does not have CGD, contemporary authors advocate weighing the **degree** to which the cervical spine contributes to a presentation that is often multifactorial — a nil, minor, major, or compensatory role — because mixed presentations are the rule [7,12]. This framing is particularly useful to the vestibular physician, for whom CGD is rarely the whole story and far more often one contributor among vestibular, visual and psychological factors [9,39].

□ **Key Point:** Cervicogenic dizziness is non-vertiginous dizziness arising from disordered cervical proprioception, diagnosed by exclusion plus positive cervical findings and a response to cervical treatment. Because neck pain and dizziness are both common, co-occurrence alone never confirms causation [1,4,7].

Sensory Integration for Head-on-Trunk Position

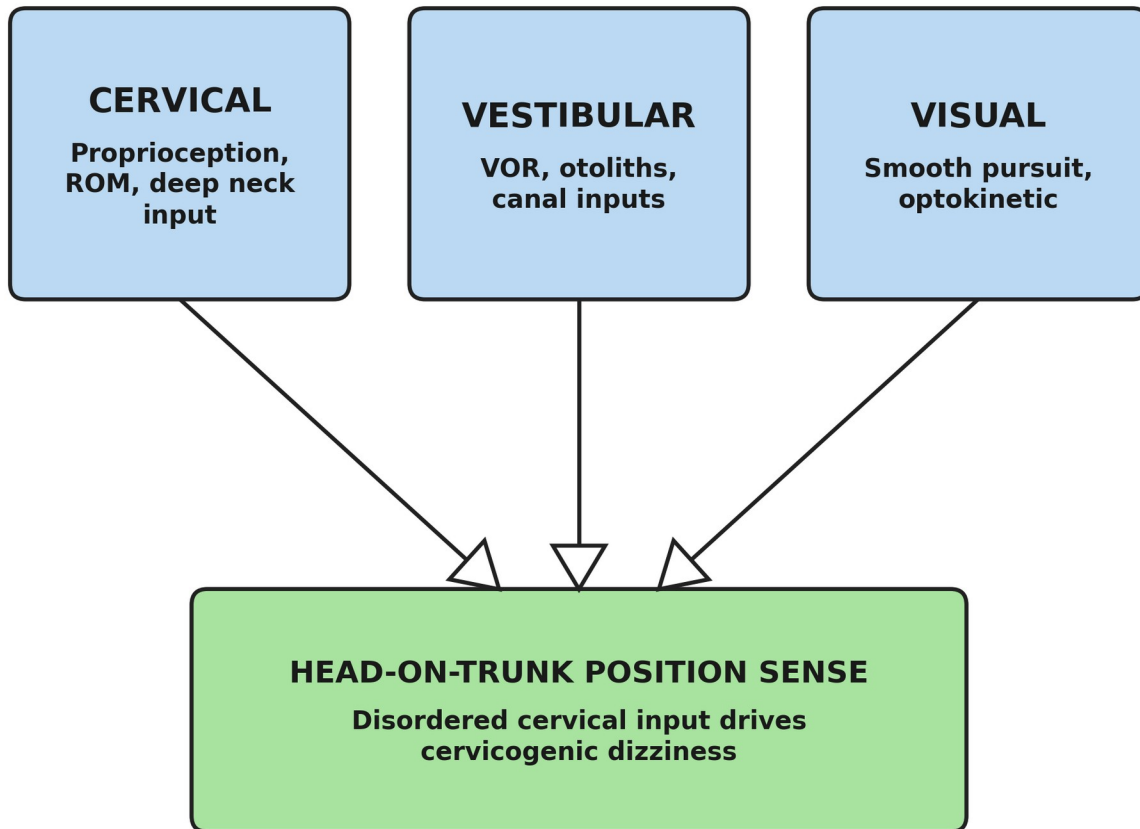


Figure 1. Sensory integration for head-on-trunk position — cervical, vestibular and visual signals converge to determine orientation; cervicogenic dizziness arises from disordered cervical input

Source: Adapted from Treleaven [3] and Peng et al. [6].

Table 1. Populations and contexts in which a cervicogenic contribution to dizziness is more likely.

Context	Why a cervical contribution is plausible	Key references
Chronic mechanical neck pain	Pain-related inhibition and altered spindle signalling impair cervical proprioception	[5,6]
Whiplash-associated disorder	Trauma to upper cervical structures produces measurable sensorimotor deficits	[15,16,17]
Older adults with degenerative change	Receptor density and integration decline; falls risk rises	[26,35]
Post-concussion presentations	Concomitant cervical injury commonly accompanies the brain injury	[44]
Forward-head posture / suboccipital dysfunction	Sustained suboccipital load alters proprioceptive output	[11]

Classically, several mechanisms have been proposed by which the neck can produce dizziness: the proprioceptive mechanism — the dominant, best-supported form addressed in this review — together with rare vascular compression of the vertebral artery and, historically, irritation of the cervical sympathetic plexus, the last now largely discredited [9,13]. Separating the common proprioceptive form

from the rare vascular form is the single most important conceptual step, because their investigation and management differ entirely [13,40].

II. Pathophysiology — Cervical Afferents and Sensory Reweighting

Maintaining stable orientation of the head on the trunk, and of the eyes on a target during head and neck movement, depends on the integration of three afferent streams: vestibular, visual and cervical proprioceptive [3,6,7]. The deep upper cervical muscles — notably rectus capitis posterior major and minor, obliquus capitis, longus capitis and longus colli — contain among the highest densities of muscle spindles of any muscle in the body, and the facet joint capsules and ligaments add further mechanoreceptive input [6,11]. These signals project to the vestibular nuclei, the cerebellum, the superior colliculus and the central cervical nucleus, where they are combined with labyrinthine and retinal information to drive the cervico-ocular, cervicocollic and cervicospinal reflexes [3,6].

Cervical Proprioceptive Pathways and Reflexes

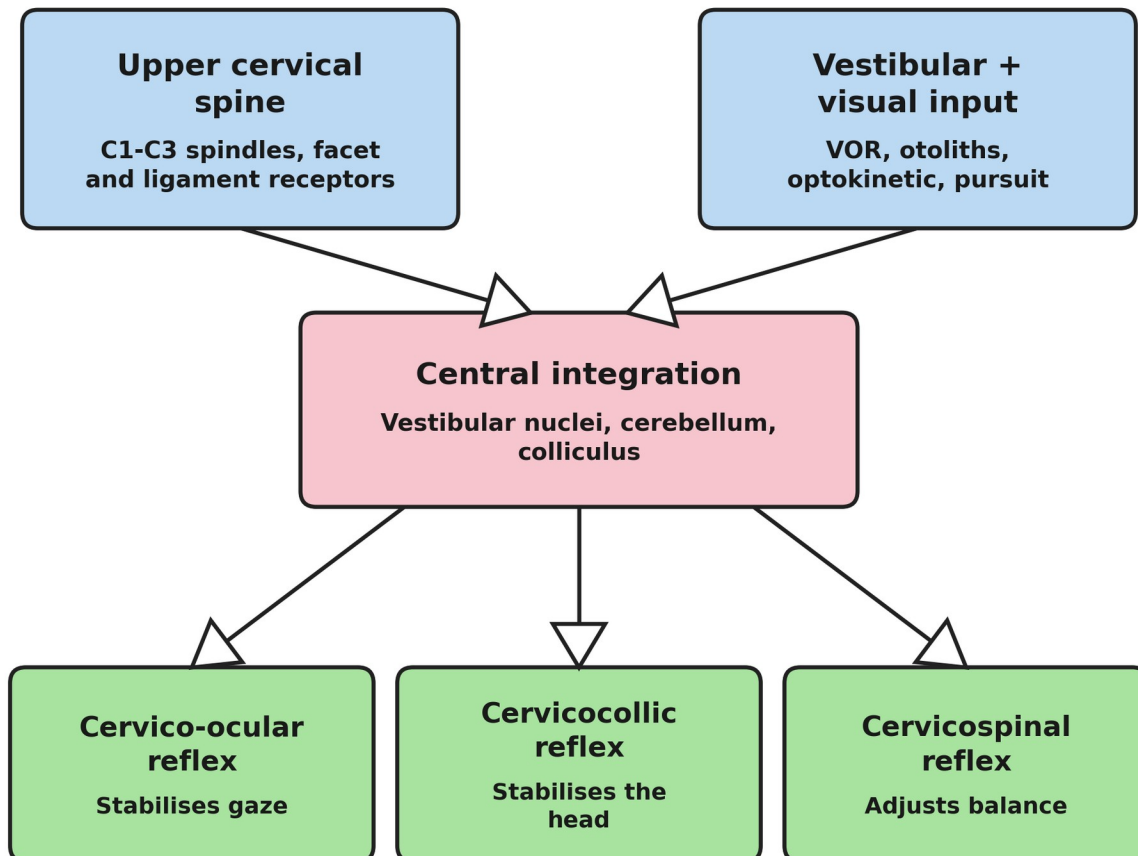


Figure 2. Cervical proprioceptive pathways and reflexes — upper cervical receptors feed central integration with vestibular and visual input to generate stabilising reflexes

Source: Adapted from Peng et al. [6] and Treleaven [3].

When cervical afferent input is distorted — by nociception, by structural or functional change in the cervical muscles and joints, or by degenerative receptor loss — the central nervous system receives a signal that conflicts with intact vestibular and visual information [5,6,10]. Experimental neck muscle pain induced by hypertonic saline reproduces measurable changes in postural sway and head–eye coordination, supporting a causal link between altered cervical signalling and sensorimotor disturbance

[6]. The resulting sensory mismatch is interpreted by the brain as illusory motion or instability; in isolation the effect is small, but it is amplified when cervical dysfunction is superimposed on reduced vestibular function, high visual demand, or fatigue [3,9].

Sensory reweighting and central maladaptation

Beyond the peripheral mismatch, contemporary models emphasise maladaptive central processing. The nervous system normally reweights its reliance among the three channels according to their reliability; in persistent neck disorders this reweighting can become maladaptive, locking in excessive dependence on visual or vestibular input and sustaining the error signal even after the original cervical insult settles [12]. This central component helps explain why some patients remain symptomatic despite apparently resolved neck pathology and why purely peripheral treatment sometimes disappoints [7,12]. It also positions CGD within the same sensorimotor-integration framework used for other multisensory disorders familiar to the vestibular physician [8,12].

Pathophysiology of Cervicogenic Dizziness

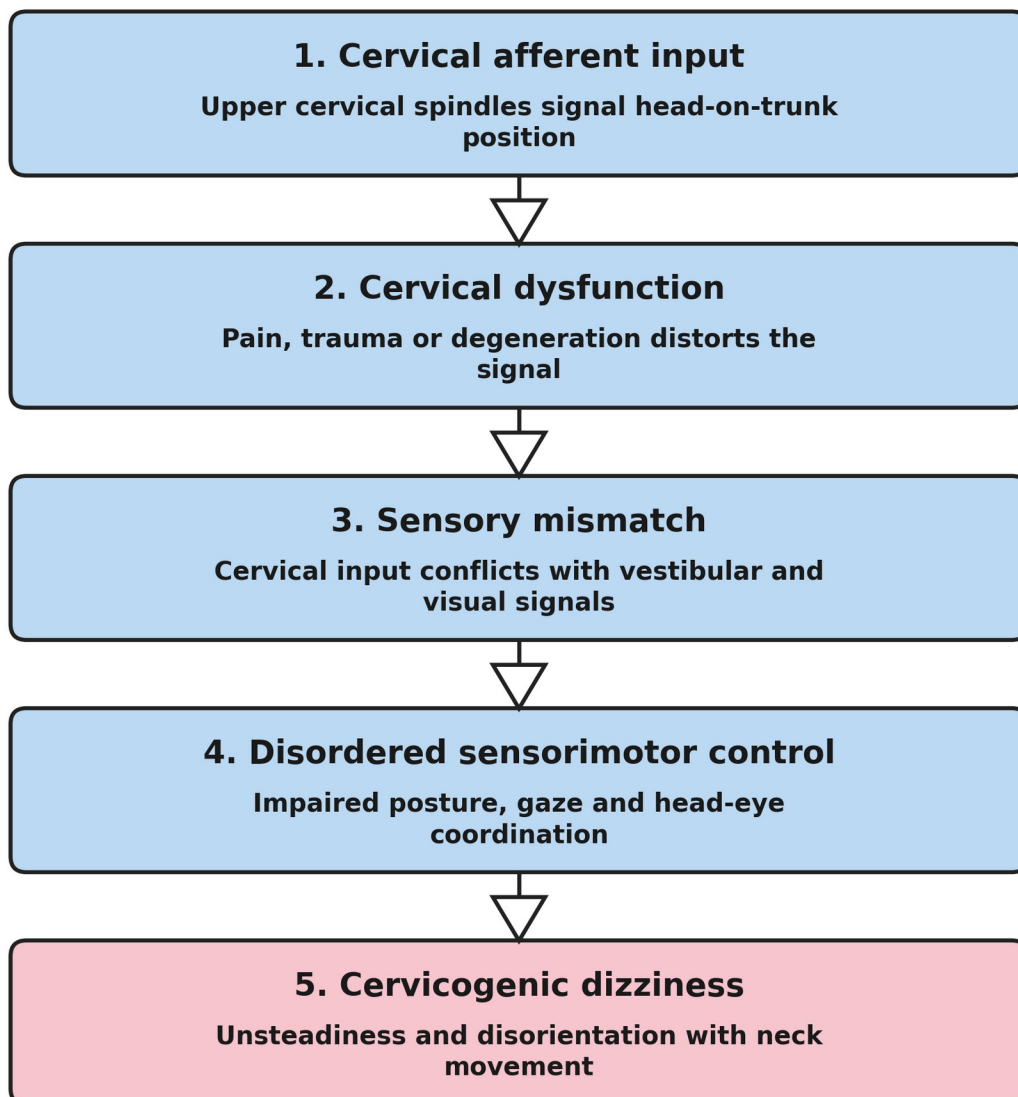


Figure 3. Pathophysiology of cervicogenic dizziness — a cascade from cervical afferent input through dysfunction, sensory mismatch and disordered control to dizziness
Source: Adapted from Li et al. [5] and De Hertogh et al. [12].

□ **Clinical Insight:** The upper cervical segments (C1–C3) carry the proprioceptive load most relevant to dizziness. Examination and treatment that target the suboccipital and upper cervical region are therefore more likely to influence symptoms than mid- or lower-cervical interventions alone [6,11].

The cervical receptors most relevant to dizziness are the muscle spindles of the deep, short suboccipital muscles, which are densely innervated and operate over the small ranges typical of fine head-on-trunk control [6,11]. Their afferents converge in the central cervical nucleus and vestibular nuclei with otolith and canal signals, providing the substrate for the tight coupling of neck position with eye and postural responses [3,6]. The density of these spindles, far exceeding that of limb muscles, is thought to explain why upper cervical dysfunction exerts a disproportionate influence on orientation [6,11].

The cervico-ocular reflex is normally weak in healthy adults but is plastic: its gain rises when vestibular function is reduced, so the neck assumes a larger role in gaze stabilisation [3,8]. This adaptive up-weighting is helpful after vestibular loss, but it becomes a liability when cervical input is itself disordered, where it can produce or amplify movement-related visual disturbance and unsteadiness [3,8].

III. Clinical Features — The Neck–Dizziness Symptom Complex

Patients with CGD typically describe unsteadiness, light-headedness, a sensation of floating or swimming, or spatial disorientation rather than true rotatory vertigo [1,4,9]. The absence of a spinning illusion is an important discriminating feature, although it is not absolute, and some patients report brief disequilibrium provoked by head-on-trunk movement [9,13]. Symptoms are characteristically related to neck position and movement, are worse with sustained postures such as prolonged computer work, and tend to ease with rest and with resolution of the neck problem [1,15].

The defining clinical association is the temporal and mechanical coupling of dizziness with neck pain or stiffness: the dizziness emerges with, or shortly after, the cervical complaint and fluctuates with it [4,14]. Episodes usually last minutes to hours rather than seconds, distinguishing them from the very brief spells of benign paroxysmal positional vertigo [4,9]. Commonly associated features include cervicogenic headache, restricted and painful neck movement, visual complaints such as difficulty focusing during head movement, and a subjective sense of poor balance that is often more prominent than objective findings [3,15,38].

Where the presentation follows trauma — most often whiplash — dizziness and unsteadiness are reported by a substantial proportion of patients and are associated with greater neck pain, larger cervical joint position error, and poorer balance and eye-movement control than in those without dizziness [15,17]. These traumatic presentations tend to be more disabling and more persistent, and they carry a higher burden of comorbid anxiety and hypervigilance that can amplify symptom report [16,35].

□ **Clinical Pearl:** If a patient describes movement- and posture-related unsteadiness that tracks with their neck pain — worse after a long day at the screen, better when the neck settles — and denies a true spinning illusion, a cervical contribution is plausible and the neck warrants formal examination [1,9].

Case-level and observational reports reinforce the pattern seen in larger series: patients in whom discrete episodes of neck pain and dizziness rise and fall together, and in whom targeted treatment of the neck relieves both, provide the clearest clinical signal of a cervicogenic contribution [42]. Conversely, where dizziness persists unchanged despite resolution of neck pain, a primary cervical cause becomes far less likely and other contributors should be sought [35,42].

IV. Diagnosis — Criteria, Bedside Tests and the Role of Exclusion

CGD remains a diagnosis of exclusion supported by positive cervical findings; there is no single confirmatory laboratory or imaging test [4,10,45]. A pragmatic, stepwise approach is therefore essential:

first exclude peripheral and central vestibular disease and vascular causes, then demonstrate cervical musculoskeletal and sensorimotor dysfunction, and finally look for a response to cervically directed treatment [4,5]. Reiley and colleagues set out exactly such a structured diagnostic sequence, and Li and colleagues have proposed explicit working criteria drawn from the available evidence [4,5].

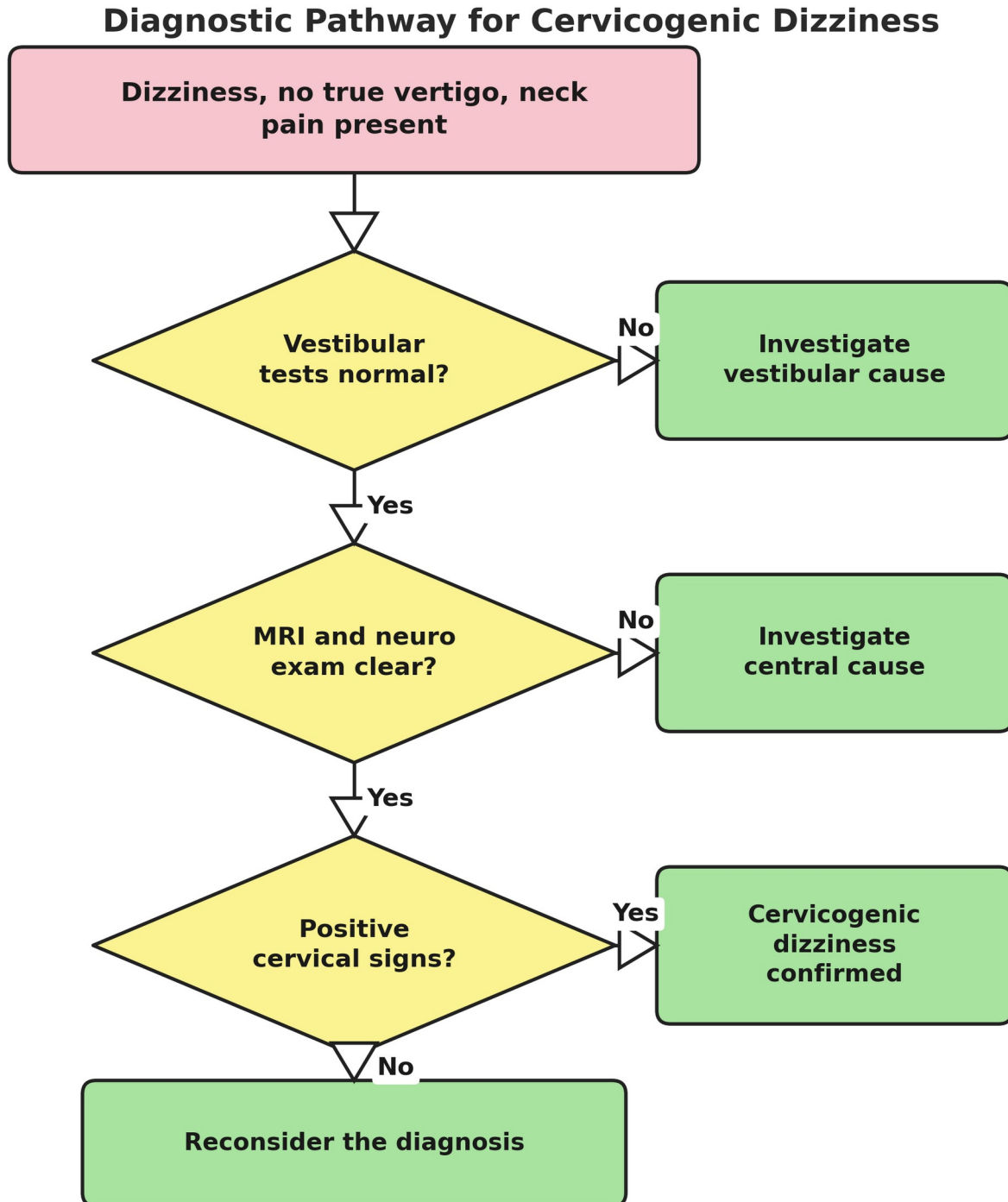


Figure 4. Diagnostic pathway for cervicogenic dizziness — rule out vestibular causes, rule out central and vascular causes, then confirm positive cervical signs and response

Source: Adapted from Reiley et al. [4] and Li et al. [5].

Table 2. Working diagnostic criteria for proprioceptive cervicogenic dizziness (synthesised).

Domain	Supportive feature
Symptom quality	Non-vertiginous dizziness, unsteadiness or disorientation, not true spinning [1,9]

Neck association	Dizziness temporally and mechanically linked to neck pain, stiffness or movement [4,14]
Cervical signs	Restricted/painful cervical motion and impaired sensorimotor control on testing [5,18]
Exclusion	Vestibular, central and vascular causes reasonably excluded [4,10]
Response	Improvement in dizziness with treatment directed at the cervical spine [2,5]

No bedside test is pathognomonic, but several measures of cervical afferent function support the diagnosis when interpreted as a cluster rather than in isolation [18,24]. The smooth pursuit neck torsion (SPNT) test — comparing smooth-pursuit gain with the head neutral versus the trunk rotated under a fixed head — is the best-studied, showing high sensitivity and specificity for cervical dizziness in whiplash cohorts and remaining abnormal where central and peripheral vestibular controls are normal [21,19,20]. Cervical joint position error (JPE), the head-repositioning accuracy measured after active rotation, is increased in neck-pain and whiplash populations, although it correlates only weakly with balance and SPNT abnormality, underlining that a single test is insufficient [18,22,23]. Newer bedside adjuncts such as the Romberg neck-torsion test extend the same principle of unmasking cervically driven instability [41].

The subjective burden of dizziness should be quantified, both to support the diagnosis and to track change. The Dizziness Handicap Inventory remains the standard self-report instrument and is widely used in CGD trials [36]. The Bárány Society's diagnostic criteria for competing entities — particularly vestibular migraine — should be applied explicitly during the exclusion process so that treatable mimics are not overlooked [37].

Cervical Sensorimotor Assessment Battery

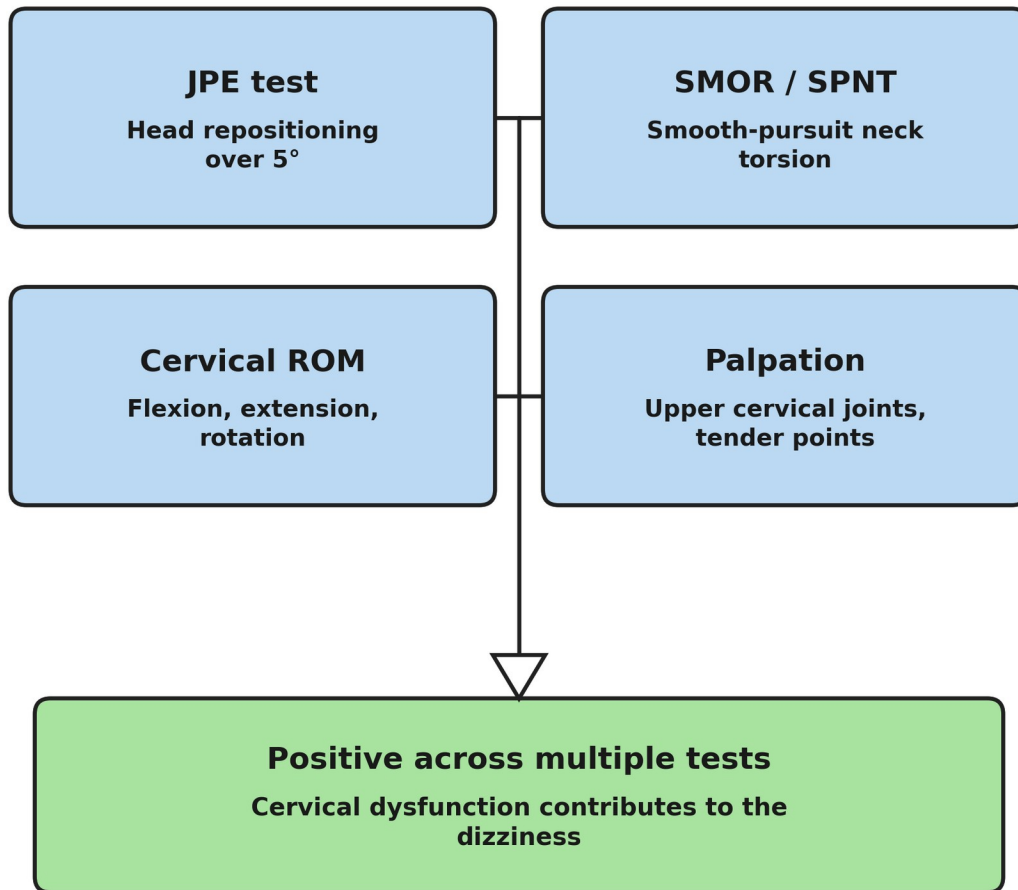


Figure 5. Cervical sensorimotor assessment battery — joint position error, smooth-pursuit neck torsion, range-of-motion and segmental palpation interpreted as a cluster

Source: Adapted from de Zoete et al. [24] and Treleaven et al. [18].

□ **Important:** No cervical test confirms CGD on its own. Before attaching the label, obtain vHIT or caloric testing and consider imaging if there are red flags — sudden vertigo with spontaneous nystagmus, progressive symptoms, new hearing loss, or focal neurology — because these point away from a cervical cause [4,10,39].

Interpreting these tests requires awareness of their limitations. The SPNT is the most specific single measure, but it is influenced by nociceptive as well as proprioceptive factors, and reported gain thresholds vary between laboratories [19,21]. Joint position error has only modest reliability and limited specificity in isolation, and normative values differ with age and method [22,24]. None of the tests has been validated against an accepted reference standard for cervicogenic dizziness, which is precisely why a converging cluster of findings, rather than any single positive result, should drive interpretation [24].

V. Cervical Sensorimotor Assessment Battery

Because no test stands alone, the vestibular physician should assemble a battery that samples the different facets of cervical sensorimotor control [24]. Factor-analytic work shows that the commonly used tests measure largely independent skills — postural balance, head steadiness, movement accuracy and perceived verticality — so a combination is required to capture the breadth of dysfunction [24]. The

examination begins with a thorough cervical musculoskeletal assessment: active and passive range of motion, segmental palpation of the upper cervical joints for tenderness and restriction, and reproduction of the patient's symptoms by cervical provocation [5,18].

Targeted sensorimotor tests then follow. Cervical JPE is assessed with a laser mounted on a headband: the patient relocates to a neutral or target head position with eyes closed, and an error beyond approximately 4.5 degrees (commonly cited as more than about 5 degrees) is regarded as abnormal [18,22]. The SPNT test evaluates neck-afferent influence on eye-movement control and is the most specific single test of cervical afferentation available [19,21]. Standing balance is examined under varied sensory conditions and during head movement, and oculomotor control (smooth pursuit, gaze stability and head–eye coordination) is screened, since deficits here are characteristic of cervical afferent disturbance [3,17]. Assessment of the deep cervical flexors with the craniocervical flexion test adds information about the motor side of control [25].

Table 3. Core cervical sensorimotor tests and their interpretation.

Test	What it probes	Supportive (positive) finding
Cervical joint position error	Head-repositioning accuracy / proprioception	Relocation error beyond ~4.5–5° [18,22]
Smooth-pursuit neck torsion	Neck-afferent influence on eye movement	Reduced pursuit gain in torsion vs neutral [19,21]
Romberg neck torsion	Cervically provoked postural instability	Increased sway in neck-torsion position [41]
Balance with head movement	Multisensory postural control	Increased sway / steps with neck motion [17,24]
Craniocervical flexion test	Deep cervical flexor control	Poor activation / endurance of deep flexors [25]
Cervical ROM and palpation	Musculoskeletal dysfunction	Restricted, painful motion; symptomatic upper-cervical joints [5]

□ **Clinical Insight:** Interpret the battery as a pattern: isolated abnormalities are common and non-specific, but a cluster of positive cervical sensorimotor findings in a patient whose dizziness tracks with the neck materially strengthens the case for a cervicogenic contribution [18,24].

The examination should also gauge how heavily the patient relies on each sensory channel, since those who have become visually or vestibularly dependent may need that dependence addressed directly in rehabilitation [3,24]. Documenting deep cervical flexor activation and endurance completes the picture, linking the sensory findings to the motor-control deficits that treatment will target [25].

VI. Differential Diagnosis

The differential is broad because so many disorders combine dizziness with neck symptoms [4,39]. The most important first step is to separate cervicogenic from peripheral and central vestibular disease. In cervicogenic presentations the head-impulse test is normal and slow neck movement reproduces symptoms, whereas acute peripheral vestibulopathy produces a spinning illusion with an abnormal head-impulse test and spontaneous nystagmus [4,20]. Comparison studies confirm that the SPNT distinguishes whiplash-related cervical dysfunction from true vestibular pathology such as that associated with vestibular schwannoma [20].

Vestibular migraine is the single most common mimic and is frequently comorbid; it should be assessed against the Bárány Society criteria and considered whenever episodic dizziness occurs with migrainous features [37]. Cervicogenic headache shares the trigemino-cervical convergence territory and can co-exist, but its trigeminal pain pattern and provocation help separate it [38]. Benign paroxysmal positional vertigo, orthostatic and other non-vestibular causes of dizziness, and post-concussion presentations all enter the differential, the last being particularly important because cervical and brain injury commonly coincide [39,44].

□ **Important:** Although vascular causes are rare, they are dangerous. Dizziness provoked specifically by sustained head rotation, or accompanied by other posterior-circulation symptoms, should prompt consideration of rotational vertebral artery (bow hunter's) syndrome and cervical arterial pathology, and appropriate imaging before any manual therapy [40].

Table 4. Differentiating cervicogenic dizziness from key mimics.

Condition	Discriminating features	References
Peripheral vestibulopathy	Spinning vertigo, abnormal head-impulse test, spontaneous nystagmus	[4,20]
Vestibular migraine	Episodic, migrainous features, meets Bárány criteria	[37]
BPPV	Seconds-long positional vertigo with characteristic nystagmus on Dix–Hallpike	[4]
Cervicogenic headache	Unilateral pain with trigeminal referral and cervical provocation	[38]
Post-concussion dizziness	History of head injury; mixed cervical and central features	[44]
Rotational vertebral artery / vascular	Dizziness on sustained rotation, posterior-circulation signs	[40]
Non-vestibular (orthostatic, anxiety)	Light-headedness with standing or situational anxiety triggers	[39]

VII. Management — Manual Therapy, Sensorimotor Rehabilitation and Adjuncts

Once a cervical contribution is established and dangerous causes excluded, management is directed at the cervical spine and at restoring sensorimotor control [2,5]. Manual therapy is the most consistently supported intervention. A systematic review and meta-analysis found moderate-quality evidence that manual therapy reduces dizziness, cervical and balance symptoms, with a stronger effect when manual therapy is combined with exercise [30]. Randomised trials of sustained natural apophyseal glides and passive joint mobilisation show reductions in dizziness frequency and handicap and improvements in cervical range of motion that are maintained at twelve months, without adverse effects [28,29].

Combined Manual + Vestibular Treatment

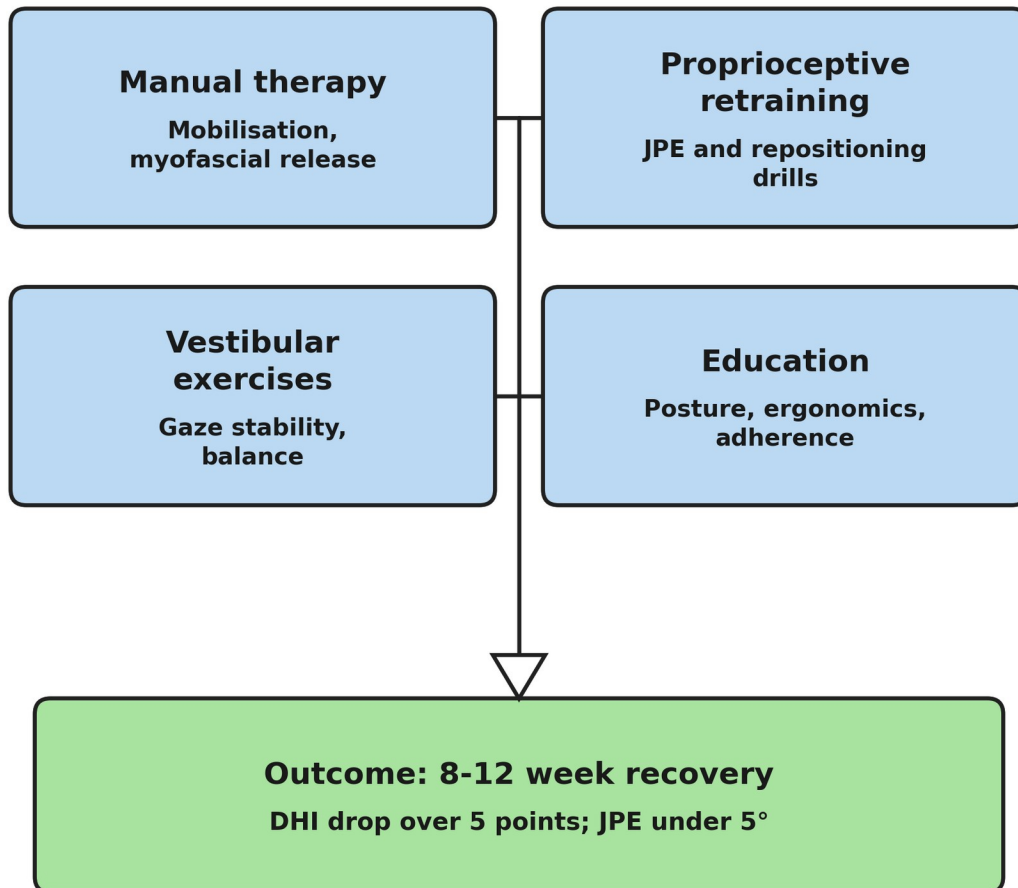


Figure 6. Combined manual and vestibular treatment — manual therapy, proprioceptive retraining, vestibular exercises and education combine to drive recovery

Source: Adapted from Reid et al. [28] and Sremakaew et al. [31].

Sensorimotor rehabilitation complements manual therapy. Adding joint-position-sense, oculomotor and balance training to local neck treatment best addresses deficits in cervical proprioception and balance and better sustains improvements in pain and disability than local treatment alone [31]. Programmes that include gaze-stability and head-eye coordination work, proprioceptive retraining of head-repositioning accuracy, and graded balance tasks target the specific impairments identified on the assessment battery [3,31]. In whiplash-related and elderly presentations, vestibular and balance rehabilitation reduces self-perceived dizziness handicap and improves postural control, and does so without aggravating neck pain [32,33,34].

Patient education and self-management are integral: explaining the benign, treatable nature of the problem, addressing posture and ergonomics, and prescribing a home programme support adherence and durable recovery [5,27]. Physiotherapy-led programmes that incorporate a behavioural component perform particularly well for the dizziness dimension [34]. Where manual care is delivered, single-group and feasibility data also describe symptom improvement, though the strongest evidence remains for physiotherapy-delivered manual therapy combined with exercise [43,30]. There is no established pharmacological treatment for CGD itself; medication is reserved for comorbid pain, migraine or anxiety identified during assessment [13,39].

Table 5. Management options and the strength of supporting evidence.

Intervention	Typical content	Evidence
Manual therapy	SNAGs, mobilisation, soft-tissue/myofascial techniques	Moderate; meta-analysis and RCTs [28,29,30]
Sensorimotor retraining	JPE/oculomotor drills, gaze stability, balance	Supportive RCT evidence [31,34]
Vestibular/balance rehab	Graded balance and habituation tasks	Beneficial in WAD and elderly [32,33]
Education & self-management	Reassurance, posture/ergonomics, home program	Recommended adjunct [5,27]
Pharmacology	For comorbid pain/migraine/anxiety only	No direct CGD indication [13,39]

Practical delivery matters. In the trial evidence, manual therapy was given over a small number of sessions — typically two to six over six weeks — with a self-treatment component to consolidate gains, and the benefits persisted at twelve months [28,29]. Before any manual technique is applied, the clinician must screen for vascular and other contraindications, since end-range rotation and sustained positions can be hazardous where vertebral artery pathology is present [40]. Techniques are then matched to the dominant impairment, combining cervical mobilisation with the sensorimotor and balance components described above [28,31].

VIII. Refractory Disease, Comorbidity and Emerging Therapies

A minority of patients fail to respond to first-line manual therapy and sensorimotor rehabilitation. The first response to apparent treatment failure is to question the diagnosis: re-screen for a missed or co-existing vestibular, central or vascular cause, since CGD is so often part of a mixed picture and the cervical role may have been over-weighted [7,12]. A stepped-care structure — confirm contribution, treat, review at six to eight weeks, then either maintain or reassess — keeps this re-evaluation explicit [30].

Stepped-Care Management Algorithm

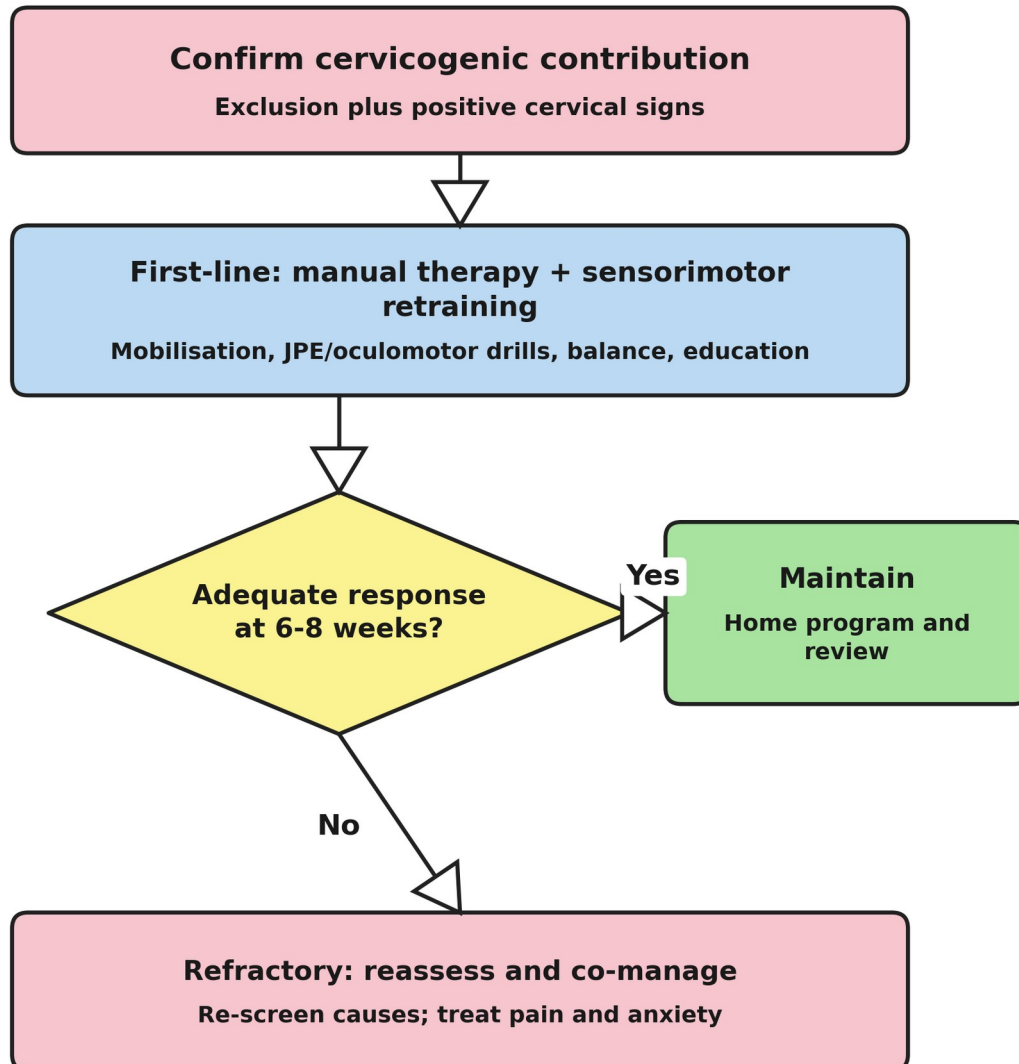


Figure 7. Stepped-care management algorithm — confirm the cervical contribution, deliver first-line therapy, review response, and escalate or maintain accordingly

Source: Adapted from De Vestel et al. [30] and Li et al. [5].

Comorbidity is the usual reason for persistence. Maladaptive central sensory reweighting, comorbid vestibular migraine, anxiety and hypervigilance, and chronic pain mechanisms can all sustain symptoms despite adequate cervical treatment, and each may need to be addressed in its own right [12,16]. Because much of the persistence of CGD appears to be centrally mediated, future therapeutic gains are likely to come from interventions that target central maladaptation alongside the periphery, and from better-defined diagnostic criteria that allow refractory cases to be characterised and compared [12]. At present the emerging-therapy evidence base is thin, and the priority is rigorous re-assessment and multidisciplinary co-management rather than novel modalities [7,30].

□ **Clinical Pearl:** Treatment failure in presumed CGD should trigger a diagnostic rethink, not simply more of the same. The most common reason for non-response is an unrecognised vestibular or central contributor [7,12].

IX. Prognosis, Outcome Measures and Special Populations

For most patients with a genuine cervicogenic contribution, the prognosis is favourable: manual therapy and sensorimotor rehabilitation produce meaningful, durable reductions in dizziness and handicap, with benefits maintained up to a year after a short course of treatment [28,29]. Outcomes should be tracked with validated measures. The Dizziness Handicap Inventory is the principal patient-reported outcome, supplemented by dizziness frequency and intensity ratings, cervical range of motion, head-repositioning accuracy and balance measures used as objective endpoints in the trial literature [36,29,34].

Prognosis is more guarded in traumatic and chronic presentations. Whiplash-related CGD is associated with greater baseline sensorimotor disturbance and a higher comorbidity burden, and a proportion of patients remain symptomatic despite treatment, although behaviourally informed exercise programmes improve the dizziness dimension [16,17,34]. Older adults are an important special population: neck pain in the elderly is associated with greater sensorimotor disturbance than in younger people, and the resulting instability has direct implications for falls prevention, making assessment and management of cervical contribution part of broader balance and falls strategies [26,35].

Table 6. Factors associated with a poorer prognosis in cervicogenic dizziness.

Factor	Why it worsens outcome	References
Traumatic (whiplash) onset	Greater sensorimotor deficit and disability at baseline	[16,17]
Comorbid anxiety / hypervigilance	Amplifies symptom report and sustains avoidance	[16,34]
Long symptom duration / central reweighting	Maladaptive central processing persists	[12]
Older age with degenerative change	Reduced integration; falls risk	[26,35]
Unrecognised mixed pathology	Cervical treatment alone insufficient	[7,12]

Maintenance underpins durability. Because cervical sensorimotor control can decondition, a brief ongoing home programme of head-repositioning, gaze-stability and balance work helps preserve gains, and patients should be counselled that occasional symptom recurrence during flare-ups of neck pain is expected and manageable rather than a sign of treatment failure [5,29].

X. Controversies, Guidelines and Future Directions

CGD remains one of the more contested diagnoses in vestibular medicine, and the vestibular physician should hold it with appropriate caution [10,12]. The central controversy is the absence of a gold-standard test: because the diagnosis depends on exclusion, a supportive cluster of cervical findings, and a treatment response, it is vulnerable to over-diagnosis whenever neck pain and dizziness merely coincide [1,10]. Sceptical and supportive authors nonetheless converge on a pragmatic position — that a physiological basis exists, that cervical afferent input demonstrably contributes to balance and gaze control, and that the diagnosis should be applied carefully and tested by response to treatment [10,12].

There are no formal society diagnostic criteria for CGD comparable with the Bárány Society definitions for vestibular migraine or PPPD, and this gap is a recognised priority [5,12]. Proposed working criteria and structured diagnostic pathways have improved consistency, but they await prospective validation [4,5]. The most useful conceptual advance for routine practice is to grade the cervical contribution rather than treat CGD as a binary entity, which aligns assessment with the multifactorial reality of the dizzy patient [7].

Future directions include developing and validating consensus diagnostic criteria, identifying more specific cervical afferent tests, clarifying the relative contribution of peripheral versus central mechanisms, and designing trials that stratify by the cause and degree of cervical involvement [12,30]. A consistent, agreed test battery would also allow the heterogeneous evidence base to be pooled more meaningfully

[24,30]. Until then, the safest practice combines disciplined exclusion, a cluster-based cervical assessment, conservative cervical and sensorimotor treatment, and honest communication about diagnostic uncertainty [4,5,12].

□ **Key Point:** Cervicogenic dizziness is best understood not as a yes/no diagnosis but as a graded cervical contribution to a usually multifactorial problem — diagnosed by careful exclusion and a cluster of cervical findings, and confirmed by response to cervically directed treatment [5,7,12].

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