Peripheral Neuropathy and Sensory Ataxia:

A Vestibular Physician's Deep Review of Proprioceptive Loss, Gait Disturbance, and Systemic Investigation

Vestibular Medicine for Vestibular Physicians

Systemic & Multisensory Balance Disorders — Module 4.5

Australian Dizziness Clinics | www.AustralianDizzinessClinics.com
Version 1.0 | June 2026

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 vestibular physiotherapists working at the deep end of systemic and multisensory vestibular 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.

Table of Contents

I. Introduction and Epidemiology

Peripheral neuropathy is among the most prevalent neurological conditions in the developed world, affecting an estimated 2.4% of the general population and rising to 8% in individuals over 55 years of age [1,2]. When the neuropathy selectively or predominantly involves large myelinated sensory fibres — those responsible for proprioception, vibration sense, and two-point discrimination — the clinical consequence is a characteristic syndrome of gait ataxia, Romberg's sign, and sensory disturbance that vestibular physicians encounter regularly and must evaluate systematically [3,4].

Sensory ataxia, defined as loss of motor coordination arising from impaired proprioceptive afferent input rather than cerebellar or vestibular dysfunction, represents a diagnostically important subgroup within the broader 'dizziness and imbalance' phenotype [5]. Unlike vestibular ataxia, which improves with visual fixation and is characterised by directional nystagmus, sensory ataxia is dominated by proprioceptive dependence: the patient compensates adequately in well-lit environments but decompensates dramatically in the dark or when visual input is occluded [6]. This Romberg-positive pattern anchors the clinical diagnosis and drives the subsequent investigation strategy.

The aetiological landscape of large-fibre peripheral neuropathy causing sensory ataxia is broad. Diabetic peripheral neuropathy (DPN) is by far the most prevalent systemic cause, affecting 50% of individuals with diabetes over their lifetime and contributing substantially to the 20–50% prevalence of peripheral neuropathy in diabetic cohorts [7,8]. Nutritional deficiencies, particularly vitamin B12 and folate, account for a significant reversible fraction — clinically important because early identification and replacement can prevent permanent neuronal loss [9]. Immune-mediated neuropathies, including chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) and Sjögren-associated sensory ganglionopathy, represent a treatable but frequently underdiagnosed category [10,11].

□ **Key Point:** Peripheral neuropathy affects ~2.4% of the general population and rises to 8% in those over 55. Among dizziness clinic populations, it is a principal contributor to multisensory balance failure and should be actively screened for in every patient with Romberg-positive imbalance [1,2,3].

Paraneoplastic sensory neuronopathies — characterised by inflammatory destruction of the dorsal root ganglia driven by onconeural antibodies — are rarer but carry critical implications, since the neuropathy may precede the cancer diagnosis by months to years [12,13]. Hereditary neuropathies, chiefly Charcot-Marie-Tooth disease and Friedreich's ataxia, present in younger patients with a slowly progressive course and strong family histories [14].

In specialised dizziness clinic populations, the contribution of peripheral neuropathy to imbalance is systematically underestimated. Studies of patients attending vestibular and balance clinics report that large-fibre sensory neuropathy is a primary or significant contributing cause in 15–25% of presentations, and co-exists with vestibular dysfunction (as in CANVAS — Cerebellar Ataxia with Neuropathy and bilateral Vestibular Areflexia Syndrome) in a meaningful fraction [15,16]. The clinical and functional consequences are substantial: patients with combined somatosensory and vestibular loss have threefold higher fall rates than those with a single deficit [17].

Table 1. Prevalence of Peripheral Neuropathy — Key Population Data.

Population group	Prevalence (%)	Primary reference
General population (all ages)	2.4%	England et al. [1]
Adults aged over 55	8%	England et al. [1]
Diabetes mellitus (lifetime risk)	50%	Tesfaye et al. [7]
Type 2 diabetes (cross-sectional)	20–50%	Boulton et al. [8]
Dizziness / balance clinic attendees	15–25%	Agrawal et al. [3]
CANVAS syndrome (combined SN + VN + cerebellar)	Rare; underdiagnosed	Cortese et al. [15]
Idiopathic chronic sensory neuropathy	Up to 25% of neuropathies	Dyck et al. [4]

II. Pathophysiology — Large-Fibre Neuropathy, Proprioceptive Loss, and the Role of the Dorsal Columns

The proprioceptive afferent pathway begins at specialised mechanoreceptors — Ia muscle spindle afferents and Golgi tendon organs in muscle, Meissner's and Pacinian corpuscles in skin — whose signals are carried centrally by large, heavily myelinated Aα and Aβ axons with conduction velocities in the range of 40–70 metres per second [18,19]. These fibres enter the spinal cord via the dorsal root and ascend ipsilaterally in the dorsal columns — the gracile fasciculus (lower limbs) and cuneate fasciculus (upper limbs) — to synapse at the nucleus gracilis and nucleus cuneatus in the caudal medulla [19,20].

Second-order neurons then decussate in the medulla and ascend contralaterally in the medial lemniscus to the ventral posterolateral thalamus (VPLc nucleus), from which third-order neurons project to the primary somatosensory cortex (postcentral gyrus, Brodmann areas 3a and 2) [20,21]. This pathway is anatomically and clinically distinct from the spinothalamic tract, which carries pain and temperature via small-diameter unmyelinated C fibres and crosses the midline at the level of entry. This distinction underlies the characteristic neurological pattern of dorsal column disease: preserved pain and temperature sensation with profound loss of vibration, joint position sense, and two-point discrimination [19].

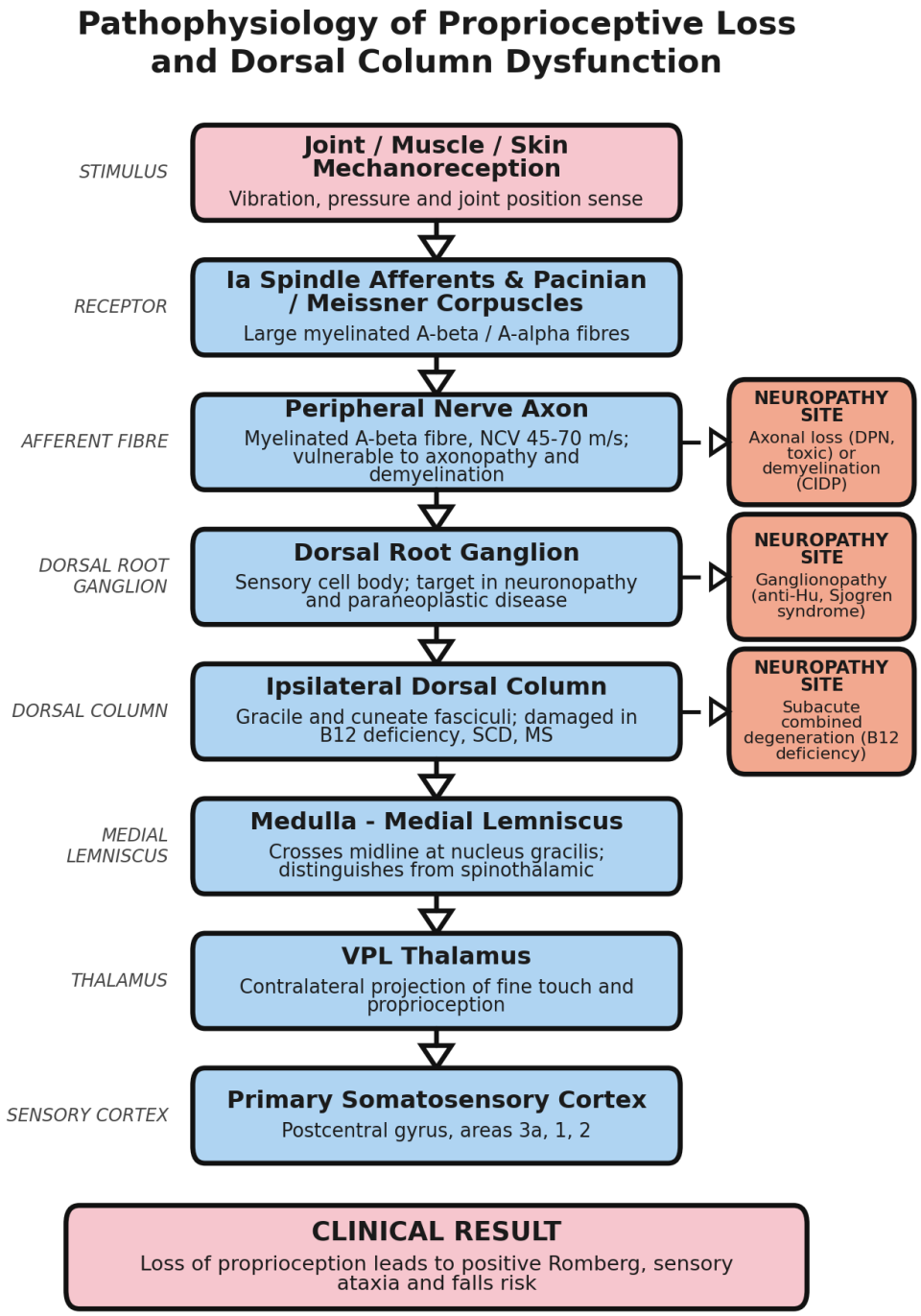

Figure 1. Pathophysiology of Proprioceptive Loss and Dorsal Column Dysfunction — anatomical pathway from peripheral mechanoreceptors to somatosensory cortex, with sites of vulnerability in different neuropathy types.
Source: Adapted from Midroni and Bilbao [18] and Schaumburg and Berger [19].

Large-fibre vs. small-fibre neuropathy

The distinction between large-fibre and small-fibre neuropathy is clinically important because these syndromes have different presentations, different investigation requirements, and different aetiological profiles [22,23]. Large-fibre neuropathy — the substrate of sensory ataxia — manifests with absent ankle reflexes, impaired vibration sense at the malleoli and great toes, positive Romberg test, abnormal nerve conduction studies (prolonged or absent sensory nerve action potentials), and characteristic gait ataxia [3,22]. Small-fibre neuropathy, by contrast, presents with burning, allodynia, and autonomic features with preserved reflexes and normal NCS, requiring skin punch biopsy for diagnosis [23].

□ **Clinical Insight:** A positive Romberg test indicates reliance on visual compensation for postural stability and implies dorsal column or large-fibre proprioceptive dysfunction. Its sensitivity for detecting large-fibre neuropathy exceeds that of bedside vibration testing at the malleolus, particularly in early disease [3,6].

Pathological mechanisms in peripheral neuropathy

Peripheral neuropathies affecting large fibres fall into three principal pathological categories, each with distinct electrophysiological signatures on nerve conduction studies [24,25]. Axonal neuropathies — the most common pattern in diabetic, toxic, and nutritional neuropathies — involve primary axon degeneration (Wallerian-type or dying-back), with reduced SNAP amplitude but relatively preserved conduction velocity. Demyelinating neuropathies — seen in CIDP, hereditary motor and sensory neuropathy type 1 (CMT1A), and post-infectious immune neuropathies — produce slowed conduction velocity, conduction block, and temporal dispersion, with relative preservation of axon count until late disease [24,25,26].

Neuronopathies (ganglionopathies) are a distinct third category in which the primary insult targets the dorsal root ganglion cell body rather than its peripheral axon [12,13]. Causes include paraneoplastic anti-Hu antibody-mediated inflammation, Sjögren's syndrome, and idiopathic sporadic ganglionopathy. Because the cell body is destroyed, Wallerian degeneration proceeds both distally and proximally (anterograde and retrograde), producing a pattern of sensory loss that may be non-length-dependent, asymmetric, and associated with a pronounced proprioceptive deficit out of proportion to the distal sensory loss [12,13]. Nerve conduction studies show diffuse sensory nerve action potential (SNAP) loss without a clear distal-to-proximal gradient — a key distinguishing feature from the typical length-dependent pattern of metabolic neuropathies [25].

Dorsal column disease

Isolated dorsal column dysfunction without peripheral nerve involvement produces sensory ataxia by a central rather than peripheral mechanism. Subacute combined degeneration (SCD) of the spinal cord, caused by vitamin B12 deficiency, produces a characteristic bilateral posterior and lateral column demyelination. The posterior columns carry proprioception and vibration; their dysfunction produces Romberg positivity, pseudoathetosis, and a sensory ataxic gait indistinguishable from peripheral large-fibre neuropathy at bedside — but distinguishable by the intact or brisk reflexes (unless concurrent peripheral neuropathy is present), the absence of abnormal NCS, and the MRI finding of dorsal column T2 signal hyperintensity [9,27]. Cervical spondylotic myelopathy can also compress the posterior columns, as can multiple sclerosis demyelinating plaques in the posterior cord [27,28].

□ **Important:** When a patient presents with Romberg-positive sensory ataxia and brisk deep tendon reflexes, dorsal column disease (subacute combined degeneration, cervical myelopathy, or MS) must be excluded before attributing the syndrome to peripheral neuropathy alone. MRI spine is mandatory in this clinical context [27,28].

III. Clinical Features — Gait Ataxia, Romberg's Sign, and Sensory Symptoms

The clinical presentation of large-fibre sensory neuropathy causing sensory ataxia is remarkably consistent across aetiologies, reflecting the shared anatomical substrate [3,5]. The cardinal features are: (1) progressive gait unsteadiness that worsens in reduced lighting and on uneven surfaces; (2) positive Romberg's sign — loss of balance with eyes closed while standing with feet together; (3) impaired proprioception at the ankles and, in more advanced disease, at the knees and fingers; (4) impaired vibration sense with a tuning fork at 128 Hz; and (5) absent or markedly reduced ankle deep tendon reflexes [3,5,22].

The gait pattern in sensory ataxia is wide-based, with high steppage and stamping quality as the patient attempts to substitute visual and auditory cues for absent proprioceptive feedback [5,6]. The patient characteristically watches their feet while walking, unlike cerebellar ataxia where visual compensation is

less effective. Tandem gait is severely impaired early, even when the Romberg test is only weakly positive, and is a more sensitive screening tool in early-to-moderate neuropathy [6,29].

Romberg's test and its variants

Romberg's test, described in 1846, remains one of the most sensitive bedside instruments for identifying dorsal column and large-fibre proprioceptive loss [30]. In its standard form, the patient stands with feet together and arms by their sides, first with eyes open and then with eyes closed: sway that is substantially greater with eyes closed, or frank loss of balance requiring stepping, constitutes a positive result [6,30]. Modified Romberg variants — tandem Romberg, foam Romberg (standing on foam with eyes closed), and sensory organisation testing (SOT) on a computerised dynamic posturography platform — increase sensitivity and allow quantitative assessment of somatosensory, visual, and vestibular contributions to balance [31,32].

The foam Romberg test is particularly useful in mild sensory neuropathy because standing on foam eliminates the ankle-foot somatosensory input that the patient normally substitutes for absent proprioception, unmasking the deficit when visual cues are also removed [31,32]. Quantitative Romberg timing — measuring the duration patients can stand on foam with eyes closed — correlates with peripheral neuropathy severity on electrophysiological testing and serves as a reproducible outcome measure in rehabilitation programmes [31].

□ **Clinical Pearl:** The foam Romberg (eyes closed on unstable foam surface) is more sensitive than standard Romberg for detecting early large-fibre sensory neuropathy. Quantify with a stopwatch: normal elderly individuals sustain 30 seconds; impairment below 10 seconds suggests clinically significant somatosensory loss [31,32].

Sensory symptoms

Accompanying positive sensory symptoms — paraesthesiae, tingling, numbness in a glove-and-stocking distribution — are present in many patients with large-fibre neuropathy, though their absence does not exclude significant proprioceptive loss [22,23]. Pain is variable: it is prominent in small-fibre involvement (burning, allodynia) but less so in pure large-fibre disease. Pseudoathetosis — slow, writhing involuntary movements of the fingers when held outstretched with eyes closed — reflects the loss of proprioceptive feedback to the motor cortex and is pathognomonic of severe dorsal column or large-fibre dysfunction [5,33]. Its presence should direct immediate investigation for neuronopathy, B12 deficiency, and paraneoplastic disease.

Functional impact and falls

The functional consequence of sensory ataxia is significant: patients with peripheral neuropathy have a 2–3-fold increased risk of falls compared with age-matched controls, and recurrent fallers with neuropathy have a 7-fold higher rate of fall-related fractures [17,34]. Activities dependent on proprioceptive feedback — stair descent, stepping on irregular terrain, night-time ambulation, and swimming — are disproportionately affected [17]. Standard gait and balance assessments including the Dynamic Gait Index (DGI), the Functional Gait Assessment (FGA), and the Berg Balance Scale correlate with neuropathy severity and quantify functional disability for rehabilitation planning [35,36].

IV. Diagnostic Criteria and Clinical Assessment

There are no universally validated diagnostic criteria specific to 'sensory ataxia' as a syndrome. The diagnosis is clinical — based on the constellation of Romberg-positive imbalance, impaired proprioception and vibration sense, absent ankle reflexes, and an ataxic gait pattern in the absence of cerebellar or vestibular signs [3,5]. The diagnostic task for the vestibular physician then proceeds in two stages: first, confirming that the ataxia is proprioceptive in origin (as opposed to cerebellar or vestibular); and second, localising the lesion to peripheral nerve, dorsal root ganglion, or dorsal columns [5,22].

The modified EFNS/PNS criteria for diagnosis of peripheral neuropathy require at least two of the following: (1) neuropathic symptoms, (2) signs of peripheral nerve dysfunction, and (3) confirmatory

electrophysiological or pathological evidence [24,26]. For the specific diagnosis of large-fibre sensory neuropathy causing ataxia, nerve conduction studies are the cornerstone of confirmation, providing both localisation and mechanistic classification (axonal vs. demyelinating) [24,25].

Nerve conduction studies and EMG

Nerve conduction studies (NCS) and electromyography (EMG) are the most informative investigations in the evaluation of suspected peripheral neuropathy [24,25,26]. Sensory NCS assesses the sural, median, and radial sensory nerves, measuring sensory nerve action potential (SNAP) amplitude and latency. Absent or reduced sural SNAP is the earliest and most sensitive electrophysiological finding in distal large-fibre neuropathy [25]. Motor NCS evaluates compound muscle action potential (CMAP) amplitude and conduction velocity; demyelinating neuropathies produce markedly slowed velocity, conduction block, and temporal dispersion, distinguishing them from axonal patterns [24,26]. EMG assesses muscle denervation (fibrillations, positive sharp waves) and reinnervation (large polyphasic motor unit potentials) in axonal neuropathy [25].

□ **Key Point:** Nerve conduction studies (NCS) are the gold standard for confirming large-fibre peripheral neuropathy and distinguishing axonal from demyelinating subtypes. Absent or reduced sural SNAP is the earliest electrophysiological marker of distal sensory neuropathy [24,25,26].

Standardised balance and gait assessments

Functional gait and balance assessment provides quantitative baselines for rehabilitation planning and monitoring. The Dynamic Gait Index (DGI) — an 8-item scale scoring gait quality through different conditions — is validated in peripheral neuropathy and predicts fall risk [35]. The Functional Gait Assessment (FGA) extends the DGI with 10 items and has superior sensitivity for walking while performing cognitive tasks — a real-world scenario where patients with sensory ataxia decompensate [36]. The Berg Balance Scale assesses 14 static and dynamic balance tasks; a score below 45/56 indicates elevated fall risk [36]. These instruments also serve as outcome measures for vestibular rehabilitation.

Table 2. NCS/EMG Patterns in Different Neuropathy Types.

Neuropathy type	SNAP amplitude	Sensory NCV	CMAP amplitude	Motor NCV	EMG changes
Axonal sensory (DPN, toxic)	Reduced/ absent	Normal or mildly slowed	Normal or reduced	Normal or mildly slowed	Fibrillations; large MUPs (chronic)
Demyelinating (CIDP, CMT1A)	Reduced (late)	Markedly slowed (less than 38 m/s)	Reduced (late)	Markedly slowed; conduction block	Mild denervation in chronic
Neuronopathy (ganglionopathy)	Absent (diffuse)	Absent	Normal	Normal	Normal (motor unaffected)
Dorsal column disease (SCD, MS)	Normal	Normal	Normal	Normal	Normal — requires MRI
Mixed axonal/demyelinating (CIDP-axonal)	Reduced/ absent	Variable slowing	Reduced	Variable	Active and chronic denervation

V. Investigations — Nerve Conduction Studies, Blood Panel, and Skin Biopsy

The investigation of sensory ataxia is stepwise and systematic, beginning with laboratory tests to identify common and treatable causes, proceeding through neurophysiology to characterise the neuropathy type, and extending to neuroimaging, antibody testing, and tissue biopsy when the initial evaluation is

unrevealing [37,38]. The yield of a structured investigation protocol approaches 75–80% in expert clinical hands, with the remaining 20–25% classified as idiopathic chronic sensory neuropathy [4,37].

Baseline blood panel

The standard blood panel for peripheral neuropathy evaluation should include: vitamin B12 and methylmalonic acid (MMA — more sensitive than B12 alone for functional B12 deficiency); folate; HbA1c and fasting glucose (diabetic neuropathy); full blood count (macrocytosis, anaemia); renal function (uraemic neuropathy); liver function tests (hepatic neuropathy, alcoholism); thyroid function (hypothyroid neuropathy); immunoglobulin electrophoresis and protein electrophoresis (SPEP, UPEP — MGUS, myeloma); erythrocyte sedimentation rate and CRP; ANA (connective tissue disease); and anti-SSA/SSB antibodies with Schirmer's test (Sjögren's syndrome) [37,38,39].

When clinical features suggest a paraneoplastic or immune-mediated neuronopathy, extended antibody testing is indicated: anti-Hu (ANNA-1, associated with small-cell lung cancer), anti-Ri (ANNA-2), anti-CV2 (CRMP-5, associated with SCLC and thymoma), anti-CASPR2 (Contactin-associated protein-like 2, associated with neuromyotonia and thymoma), and anti-MAG (myelin-associated glycoprotein, associated with IgM MGUS) [12,13,40]. The order of these investigations should be guided by the clinical phenotype: non-length-dependent ataxia, subacute onset, and known systemic cancer all increase the pre-test probability of paraneoplastic disease [12,13].

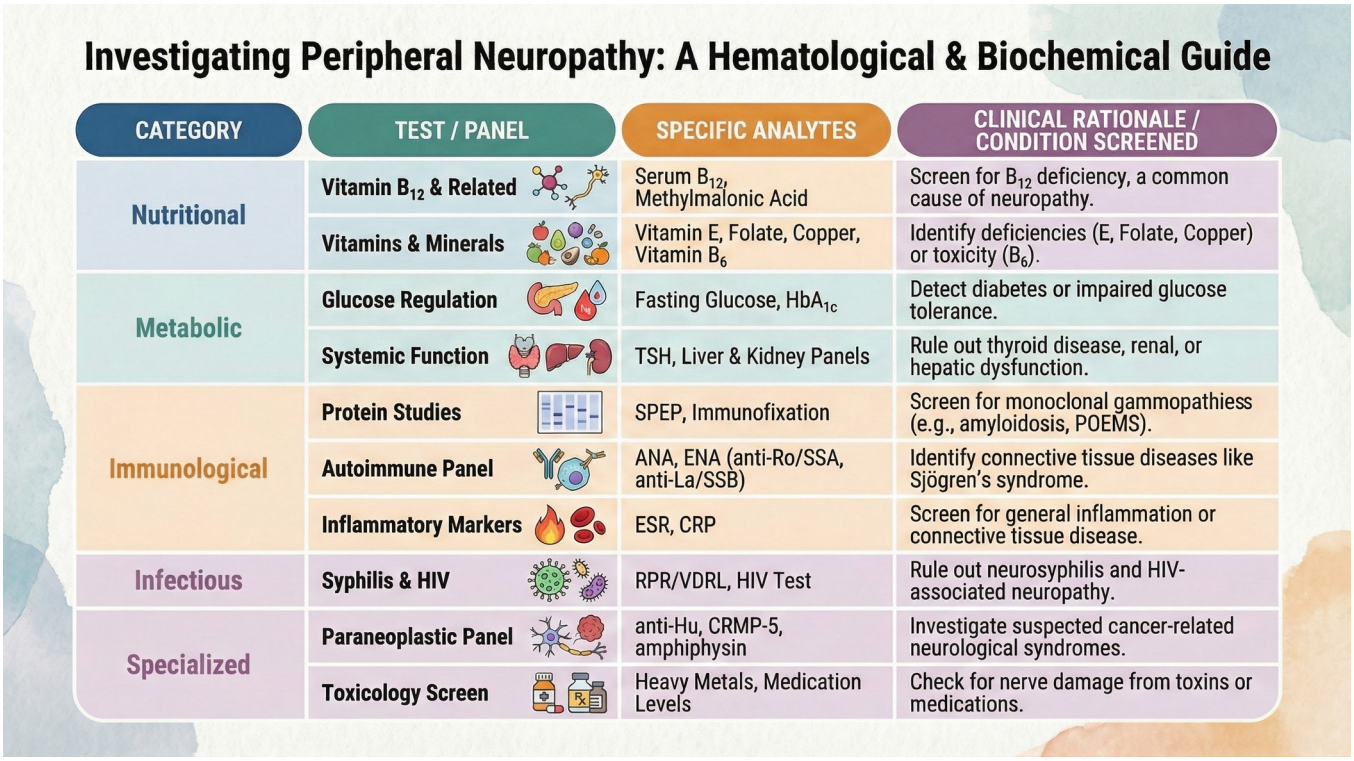

Figure 2. Blood Investigation Panel for Sensory Ataxia — structured approach to laboratory investigations by diagnostic category and clinical phenotype.

Source: Australian Dizziness Clinics clinical resource (original image).

Table 3. Blood Investigation Panel for Peripheral Neuropathy — Interpretation Guide.

Investigation	Target condition	Interpretation note	Action if abnormal
Vitamin B12 + MMA	Subacute combined degeneration	B12 can be normal with functional deficiency; MMA elevated = deficiency	IM hydroxocobalamin injection; MRI spine
HbA1c + fasting glucose	Diabetic peripheral neuropathy (DPN)	HbA1c over 6.5% = diabetes; 5.7–6.4% = prediabetes	Endocrinology referral; strict glycaemic control
SPEP + immunofixation	MGUS / myeloma neuropathy	Paraprotein band = MGUS; confirm with UPEP	Haematology referral; anti-MAG

		and bone marrow biopsy	antibody
Anti-SSA / Anti-SSB	Sjögren's ganglionopathy	Positive in 50–70% of Sjögren's neuropathy; correlates with extraglandular disease	Rheumatology referral; lip biopsy if negative
Anti-Hu (ANNA-1)	Paraneoplastic sensory neuronopathy	High specificity for SCLC; low titre can be false positive	CT chest/abdomen/pelvis + FDG-PET; oncology
Anti-CASPR2 / LGI1	CASPR2-associated neuropathy (thymoma)	Look for neuromyotonia, encephalopathy overlap	CT chest (thymoma); immunotherapy
Thyroid function (TSH + fT4)	Hypothyroid neuropathy	TSH elevated; often axonal sensorimotor pattern	Endocrinology; thyroxine replacement
Renal function (eGFR, urea)	Uraemic neuropathy	eGFR below 30 increases neuropathy risk	Nephrology; dialysis if end-stage

Skin punch biopsy for intraepidermal nerve fibre density

When NCS are normal in a patient with Romberg-positive sensory ataxia and clinical features consistent with peripheral neuropathy, skin punch biopsy for intraepidermal nerve fibre density (IENFD) is the appropriate next investigation [23,41]. A 3 mm punch biopsy is taken from a standardised site (typically 10 cm above the lateral malleolus), immunostained for PGP 9.5, and IENFD is quantified per millimetre of epidermal length against normative data [23]. Reduced IENFD confirms small-fibre neuropathy. In pure large-fibre neuropathy, IENFD is normal — confirming that absent NCS findings, rather than a normal skin biopsy, define the diagnosis [23,41].

Neuroimaging

MRI of the spinal cord is mandatory when dorsal column disease is suspected: in subacute combined degeneration it characteristically shows posterior column T2 hyperintensity on axial sequences — the 'inverted V' or 'inverted T' sign — in the cervical and thoracic cord [27,28]. In cervical spondylotic myelopathy, posterior cord compression and myelomalacia are evident [28]. Brain MRI is indicated when a central cause for ataxia remains possible after peripheral evaluation, including in paraneoplastic workup (anti-Hu encephalomyelitis) and in cerebellar atrophy associated with genetic ataxias [38,42].

VI. Differential Diagnosis

The differential diagnosis of Romberg-positive gait ataxia encompasses peripheral nervous system disorders, central nervous system conditions, and functional gait disorders. The key clinical decision — distinguishing sensory from cerebellar from vestibular ataxia — is made at bedside and determines the entire subsequent investigation and management pathway [5,6,43].

Cerebellar ataxia

Cerebellar ataxia presents with broad-based gait and prominent truncal instability that is relatively unchanged with eye closure (Romberg typically negative or weakly positive), in contrast to sensory ataxia where eye closure causes marked decompensation [43,44]. Additional cerebellar signs — dysmetria on finger-nose and heel-shin testing, intention tremor, dysdiadochokinesia, nystagmus (typically direction-changing or gaze-evoked), dysarthria, and scanning speech — distinguish cerebellar from sensory ataxia [43]. Importantly, CANVAS syndrome combines cerebellar, proprioceptive, and vestibular dysfunction and may not conform to the classic unidimensional pattern of any single ataxia type [15,16].

Bilateral vestibular hypofunction

Bilateral vestibular hypofunction (BVH) produces oscillopsia, gait unsteadiness that worsens in the dark, and a positive head impulse test bilaterally [43,44]. The clinical overlap with sensory ataxia is significant: both worsen in darkness and on foam. Distinguishing features favour vestibular origin when oscillopsia and horizontal nystagmus on dynamic visual acuity testing are present, when the video head impulse test (vHIT) confirms bilateral reduced VOR gain, and when the caloric responses are bilaterally absent or severely reduced [43,44]. In practice, BVH and large-fibre neuropathy frequently coexist, particularly in CANVAS where both deficits are pathologically linked [15,16].

□ **Important:** In any patient with Romberg-positive sensory ataxia and oscillopsia, bilateral vestibular hypofunction must be actively sought with vHIT and caloric testing. CANVAS (cerebellar ataxia, neuropathy, vestibular areflexia syndrome) carries a specific genetic basis (RFC1 repeat expansion) and is likely underdiagnosed — its recognition changes prognostic counselling and investigation planning [15,16].

Spinal cord disease

Posterior cord syndromes — subacute combined degeneration, cervical myelopathy, MS — produce sensory ataxia indistinguishable from peripheral neuropathy at bedside but can be differentiated by the reflex pattern (brisk or pathological reflexes in upper motor neuron spinal cord disease), the presence of a sensory level, bladder or bowel dysfunction, and normal or only mildly abnormal NCS in contrast to the markedly abnormal NCS of peripheral neuropathy [27,28]. Lhermitte's sign — an electric shock sensation down the spine on neck flexion — suggests posterior cord inflammation or demyelination [28,27].

Functional neurological disorder

Functional gait disorders (previously termed psychogenic ataxia) can closely mimic sensory ataxia, particularly when patients have coexisting anxiety or health anxiety about falling. Distinguishing features include internal inconsistency (dramatic improvement with distraction or treadmill testing), 'Hoover' sign, co-contraction of antagonist muscles, and complete absence of any neurological examination abnormality or electrophysiological finding [43,44]. The diagnosis requires positive clinical signs of functional disorder, not merely the absence of organic findings, and should be made with care given the high co-morbidity rate between functional and organic neurological disease [43].

Table 4. Differential Diagnosis of Sensory Ataxia — Key Distinguishing Features.

Condition	Romberg test	DTRs	Nystagmus	NCS/EMG	Key distinguishing feature
Large-fibre sensory neuropathy	Positive	Absent/ reduced (ankles)	None (unless BVH co-exists)	Reduced/absent sural SNAP	Impaired proprioception + vibration; Romberg-positive
Cerebellar ataxia	Negative or mildly positive	Normal	Gaze-evoked or direction-changing	Normal	Dysmetria, intention tremor, dysdiadochokinesia
Bilateral vestibular hypofunction	Positive (similar to SN)	Normal	HIT: bilateral catch-up saccades	Normal	Oscillopsia; vHIT reduced bilaterally; caloric absent
Posterior cord disease (SCD, MS)	Positive	Brisk/ pathological	None (unless central demyelination)	Normal	Sensory level; MRI T2 dorsal column signal
CANVAS syndrome (RFC1)	Positive	Absent	HIT: bilateral catch-up saccades	Absent SNAP (diffuse)	Triple: cerebellar + neuropathy + vestibular areflexia
Functional gait disorder	Variable / inconsistent	Normal	None	Normal	Internal inconsistency; positive Hoover; improves with distraction

VII. Aetiology and Systemic Causes

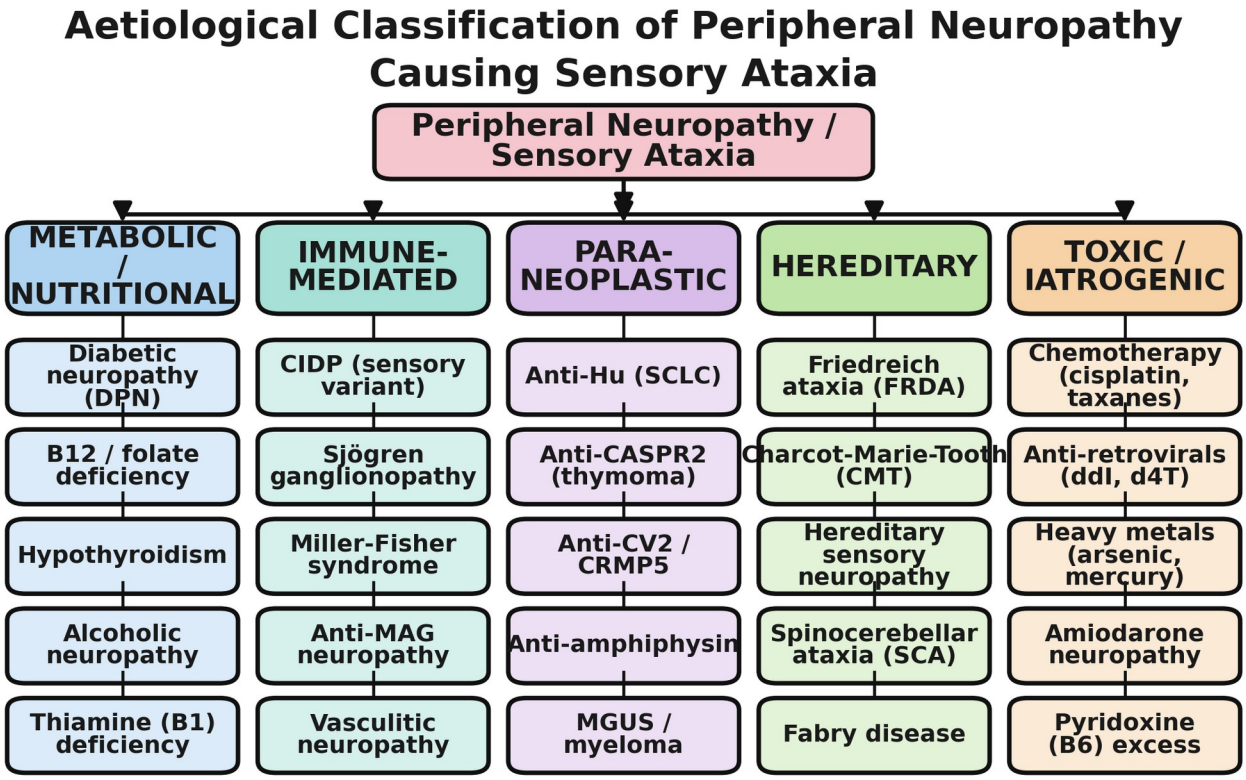

Figure 4. Aetiological Classification of Peripheral Neuropathy Causing Sensory Ataxia — five principal categories with representative causes in each.
Source: Adapted from Dyck et al. [4] and England et al. [1].

Diabetic peripheral neuropathy

Diabetic peripheral neuropathy (DPN) is the most prevalent cause of peripheral neuropathy worldwide, affecting 20–50% of individuals with diabetes in cross-sectional studies and 50% over a diabetic lifetime [7,8]. DPN most commonly presents as a distal symmetric polyneuropathy (DSPN) with length-dependent degeneration of both large and small fibres, producing sensory symptoms distally with progressive proximal spread [7,45]. Large-fibre DPN produces the sensory ataxia syndrome — absent ankle reflexes, impaired vibration at the malleolus, positive Romberg on foam, and reduced sural SNAP on NCS [8,45]. The principal risk factors are duration of diabetes, chronic hyperglycaemia (HbA1c), hypertension, dyslipidaemia, and smoking [7,45].

Management of DPN centres on strict glycaemic control — the DCCT and UKPDS demonstrated that intensive glycaemic management reduces the incidence of peripheral neuropathy by 60–64% in type 1 and modestly in type 2 diabetes [45,46]. However, established axonal DPN does not reliably reverse with glycaemic improvement; the goal becomes prevention of progression and symptomatic management of neuropathic pain (pregabalin, duloxetine, or amitriptyline) combined with active falls prevention [7,45,46].

Vitamin B12 deficiency and subacute combined degeneration

Vitamin B12 deficiency is a common and highly reversible cause of sensory ataxia that should be sought in all patients presenting with Romberg-positive imbalance [9,27]. Cobalamin is essential for myelin synthesis via the methylcobalamin-dependent remethylation of homocysteine; deficiency impairs methylation reactions critical to both peripheral nerve myelin and dorsal column integrity [9,27]. Clinical manifestations range from distal sensory neuropathy alone to the full subacute combined degeneration syndrome with posterior and lateral column involvement, spastic paraparesis, and cognitive change [27].

Diagnosis may require methylmalonic acid (MMA) measurement in addition to serum B12, since functional deficiency can occur with low-normal B12 levels, particularly in older patients and those on proton pump inhibitors or metformin [9,39]. Treatment is intramuscular hydroxocobalamin — oral supplementation is insufficient when gastric intrinsic factor deficiency (pernicious anaemia) is the mechanism [9]. Neurological recovery is partial-to-complete if treatment is instituted before axonal degeneration becomes established; prolonged deficiency results in permanent neurological sequelae [27].

□ **Clinical Pearl:** Always check methylmalonic acid alongside serum B12 — 'low-normal' B12 (150–300 pmol/L) with elevated MMA confirms functional deficiency and is an under-recognised reversible cause of sensory ataxia, particularly in patients on long-term metformin, proton pump inhibitors, or with a history of gastric surgery [9,39].

CIDP and immune-mediated neuropathies

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is the most important immune-mediated neuropathy causing sensory ataxia, with a prevalence of approximately 1–9 per 100,000 [10,26]. The sensory variant of CIDP (also called CISP — chronic immune sensory polyradiculopathy) presents with progressive sensory ataxia, Romberg positivity, and absent reflexes, with predominantly or exclusively sensory electrophysiological abnormalities and elevated CSF protein [10,26]. Recognition of CIDP is critical because it responds impressively to immune therapy: intravenous immunoglobulin (IVIG), corticosteroids, and plasma exchange each induce remission in the majority of cases [26,47].

The EFNS/PNS criteria for CIDP require clinical involvement of proximal and distal muscles for typical CIDP, but the sensory-predominant variant may be missed if motor involvement is not sought [26]. Nerve biopsy showing onion-bulb formation (evidence of repeated demyelination-remyelination cycles) confirms the diagnosis in atypical cases [25,26]. Anti-paranodal node-of-Ranvier antibodies — anti-CASPR2, anti-CNTN-1 — identify a CIDP subgroup with atypical features and less favourable response to IVIG [40,47].

Paraneoplastic sensory neuronopathy

Paraneoplastic sensory neuronopathy (PSN), caused by T-cell-mediated inflammatory destruction of dorsal root ganglia driven by onconeural antibodies, is a rare but clinically critical diagnosis [12,13]. The paradigmatic antibody is anti-Hu (ANNA-1), associated with small-cell lung cancer (SCLC) in 80% of cases [12]. The clinical phenotype is distinctive: subacute onset (weeks to months) of severe, asymmetric, non-length-dependent sensory ataxia with pseudoathetosis, profound proprioceptive loss, and electrophysiological evidence of ganglionopathy (diffuse absent SNAPs without a distal gradient, normal motor NCS) [12,13,40].

The neurological syndrome typically precedes cancer diagnosis by 3–15 months, making the neurological presentation the initial manifestation of the malignancy [12]. Urgent oncological workup — CT chest-abdomen-pelvis and FDG-PET — is mandatory when PSN is suspected. Immunotherapy (corticosteroids, IVIG, plasma exchange, rituximab) may partially stabilise but rarely reverses the neurological deficit once established, since the cell body destruction is not reversed by suppressing the immune response [12,13]. Tumour treatment itself offers the best prospect of stabilisation [13,42].

Hereditary neuropathies

Hereditary sensory and motor neuropathies — collectively Charcot-Marie-Tooth (CMT) disease — are the most common hereditary neuropathies, with a prevalence of approximately 1:2500 [14,48]. CMT1A, caused by PMP-22 gene duplication on chromosome 17p, is the most prevalent subtype and produces a length-dependent demyelinating polyneuropathy with pes cavus, distal muscle wasting, absent reflexes, and variable sensory ataxia [14,48]. Friedreich's ataxia (FRDA), caused by GAA triplet-repeat expansion in the frataxin gene, combines sensory neuropathy (ganglionopathy pattern), dorsal column degeneration, and spinocerebellar tract involvement, producing a distinctive mixed sensory-cerebellar ataxia [48,49].

Toxic and drug-induced neuropathies

Several therapeutic agents cause clinically significant sensory neuropathy. Cisplatin and other platinum compounds cause a cumulative, dose-dependent, predominantly sensory neuronopathy affecting large fibres — the 'coasting' phenomenon, where neuropathy worsens for months after drug cessation, is characteristic [50]. Taxanes (paclitaxel, docetaxel) cause a length-dependent axonal sensory neuropathy [50]. Pyridoxine (vitamin B6) excess — from over-the-counter supplementation above 200 mg/day — causes a sensory neuronopathy identical in pattern to paraneoplastic disease, but reversible if B6 is withdrawn [39]. Amiodarone, metronidazole, and anti-retrovirals (stavudine, didanosine) cause axonal sensorimotor neuropathies [50].

VIII. Management — Treating the Underlying Cause and Vestibular/Balance Rehabilitation

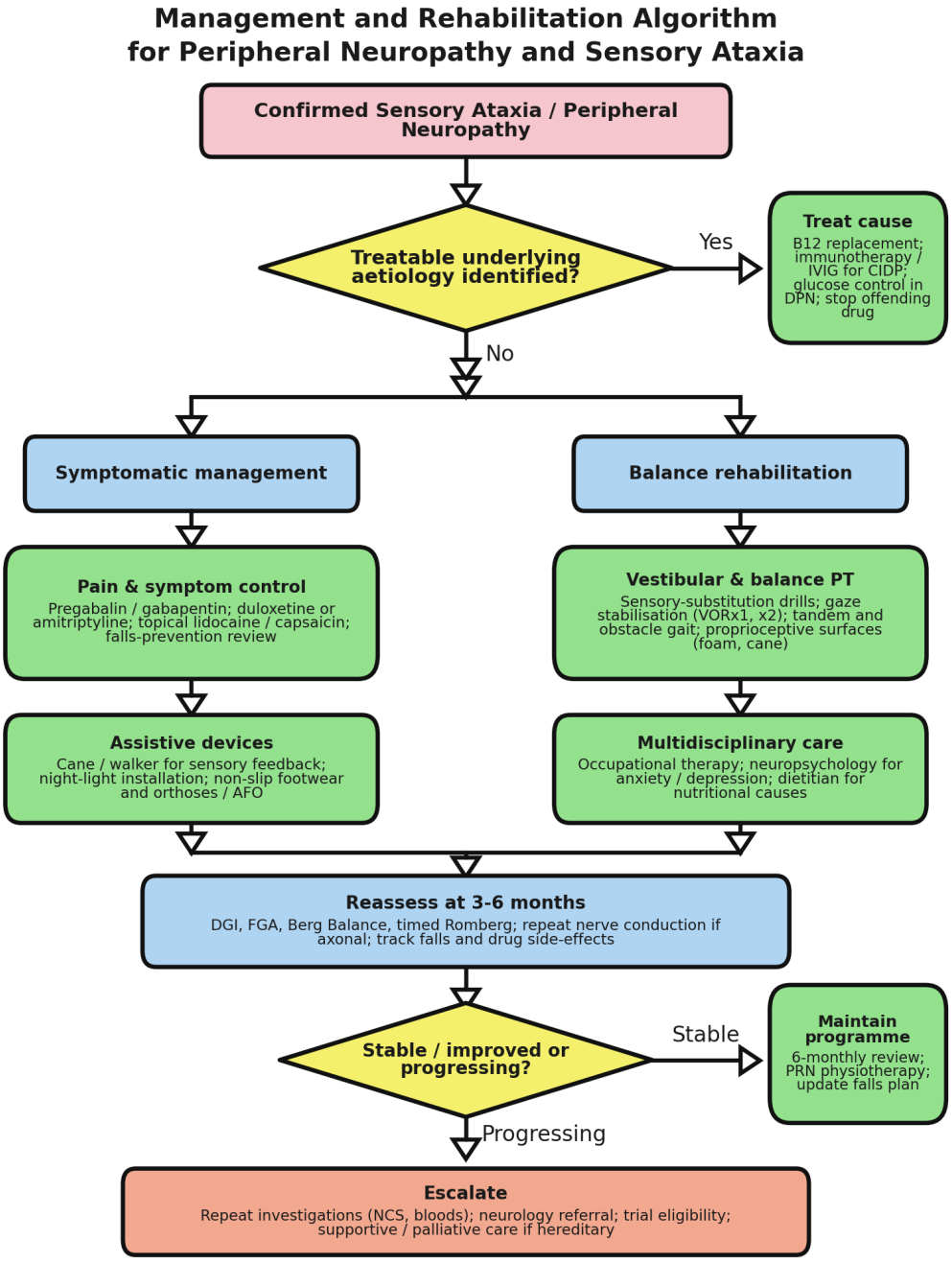

Figure 5. Management and Rehabilitation Algorithm for Peripheral Neuropathy and Sensory Ataxia — from aetiology-directed treatment through to long-term monitoring.

Source: Adapted from Bril [46], Watson et al. [51], and Pollock et al. [52].

Aetiology-directed treatment

The first management priority is treatment of the underlying cause where one exists. The diagnostic yield of a systematic workup is 75–80% in dedicated vestibular and neurology centres, and even partial identification guides therapy [4,37]. Table 5 summarises treatment by aetiology. For nutritional deficiencies, replacement is straightforward and yields the best neurological recovery outcomes: B12 IM injection series followed by monthly maintenance restores neurological function in 70–80% of patients without established axonal loss [9,27]. Glycaemic optimisation in DPN slows progression but rarely achieves reversal once axonal degeneration is established [45,46].

For immune-mediated neuropathies — CIDP, vasculitic neuropathy, Sjögren's ganglionopathy — immunotherapy is the cornerstone of management. IVIG at 2 g/kg initial dose followed by maintenance dosing is effective in CIDP, inducing measurable improvement in approximately 70% of patients [26,47]. Subcutaneous immunoglobulin (SCIG) is an increasingly used home-based alternative that avoids the infusion burden of IVIG [47]. Corticosteroids (prednisolone 1 mg/kg/day tapering over 6–12 months) are effective in CIDP but are associated with significant side effects; their use is preferred for patients requiring long-term immune suppression alongside steroid-sparing agents (azathioprine, mycophenolate) [26,47].

Table 5. Management by Aetiology — Treatment Targets and First-Line Therapies.

Aetiology	Treatment target	First-line therapy	Expected neurological recovery
Vitamin B12 deficiency	Replace cobalamin stores; arrest dorsal column damage	IM hydroxocobalamin 1000 mcg daily x5, then monthly	Partial-complete if early; limited if axonal degeneration established
Diabetic neuropathy (DPN)	Slow progression; prevent falls; manage pain	Strict glycaemic control (HbA1c target under 7%); pregabalin / duloxetine for pain	Stabilisation typical; rarely reversal
CIDP	Suppress immune-mediated demyelination	IVIG 2 g/kg initial + maintenance; or oral prednisolone; SCIG home option	70% respond; relapse common on withdrawal
Paraneoplastic (anti-Hu)	Treat underlying cancer; suppress immune destruction of DRG	Tumour treatment (chemotherapy / radiotherapy); IVIG / rituximab for stabilisation	Limited; stabilisation is best outcome once established
Sjögren's ganglionopathy	Modulate underlying Sjögren's disease activity	Hydroxychloroquine; IVIG for severe/progressive cases	Variable; non-length-dependent pattern less responsive
Hereditary (FRDA, CMT1A)	Slow progression; optimise function; genetic counselling	No disease-modifying therapy for most; idebenone trial for FRDA cardiac	Stabilisation at best; progressive long-term
Toxic / drug-induced	Remove causative agent; monitor coasting period	Cease offending drug; no specific reversal therapy for cisplatin neuropathy	Partial recovery possible if axonal; limited if neuronopathy

Neuropathic pain management

Painful large-fibre or mixed fibre neuropathy warrants pharmacological treatment targeting neuropathic pain pathways. First-line agents with class 1 evidence include: pregabalin (75–300 mg twice daily, NNT ~3–4), duloxetine (60–120 mg daily, NNT ~4–6), and amitriptyline (10–75 mg nocte, NNT ~3–4) [51]. These agents reduce pain intensity by 30–50% in responders and improve sleep quality, which is a major determinant of quality of life in neuropathic pain [51]. Choice between agents is guided by comorbidities:

duloxetine is preferred in diabetic neuropathy with concurrent depression; pregabalin in patients with anxiety or sleep disturbance; amitriptyline is generally avoided in older patients with cardiac disease or urinary retention risk [51].

Vestibular and balance rehabilitation

Balance rehabilitation — delivered by a vestibular physiotherapist experienced in somatosensory deficits — is the most evidence-supported intervention for functional improvement in sensory ataxia [52,53]. The mechanism of benefit is sensory substitution and neural compensation: patients are trained to maximise visual and remaining somatosensory cues, develop ankle-hip balance strategies, and improve central processing of degraded afferent signals [52,53]. Standardised physiotherapy programmes for neuropathic balance impairment typically include a 12-week supervised programme of: (1) gaze stabilisation exercises (VOR x1 and x2 protocols); (2) static balance progression from hard floor to foam with eyes open/closed; (3) dynamic gait training over surfaces (carpet, grass, uneven ground); (4) dual-task gait (walking while talking, counting, or carrying); and (5) proprioceptive surface exercises [52,53].

The evidence base for balance rehabilitation in peripheral neuropathy is growing. A Cochrane systematic review by Allet and colleagues found that exercise training significantly improved gait speed, balance measures (DGI, BBS), and fell frequency in patients with diabetic neuropathy [53]. Force platform biofeedback — visual or vibrotactile feedback of centre-of-pressure position — has demonstrated efficacy as a sensory substitution device in bilateral vestibular hypofunction and is increasingly applied in sensory neuropathy [52]. Aquatic therapy offers a safe environment for early balance training when falls risk is high, and is well tolerated [52,53].

□ **Clinical Insight:** Balance rehabilitation in sensory ataxia is a proprioceptive substitution task, not a vestibular adaptation task. The patient is learning to maximise visual and remaining somatosensory inputs, develop hip-based balance strategies, and cope with surface perturbations — exercises must challenge the somatosensory system progressively, not simply repeat standard VOR exercises designed for vestibular dysfunction [52,53].

Falls prevention and assistive devices

Active falls prevention is a mandatory component of sensory ataxia management given the 2–3-fold elevated falls risk [17,34]. This encompasses: home hazard assessment and modification (removing trip hazards, improved lighting, non-slip flooring); prescription of a cane or walking frame (which restores upper limb proprioceptive input to the postural control system); appropriate footwear counselling (well-fitting, flat-soled, thin-soled shoes are optimal for somatosensory feedback — thick cushioned soles reduce plantar sensory input); and patient education about high-risk scenarios (darkness, wet surfaces, unfamiliar environments) [17,34,35].

IX. Prognosis and Monitoring

The prognosis of sensory ataxia caused by peripheral neuropathy is predominantly determined by the underlying aetiology and the reversibility of the causative pathology [4,37,42]. The spectrum ranges from complete resolution (B12 deficiency identified early, treated promptly, before permanent axonal loss) to irreversible, progressive deterioration (hereditary ataxias, untreated paraneoplastic neuronopathy) [9,42,48]. Functional adaptation and rehabilitation can significantly modify the quality-of-life trajectory even when neurological deficit is fixed.

Prognosis by aetiology

Vitamin B12 deficiency neuropathy treated before axonal degeneration is established achieves partial-to-complete neurological recovery in 70–80% of patients over 6–12 months, with the proprioceptive deficit being one of the last features to resolve [9,27]. Glycaemic control in DPN rarely reverses established neuropathy but slows progression; the DCCT follow-up demonstrated that intensive therapy reduces progression by approximately 60% in type 1 diabetes [45,46]. CIDP treated with IVIG achieves functional remission in approximately 70% of patients; however, 60–80% relapse when treatment is discontinued, necessitating long-term maintenance therapy in the majority [26,47]. Paraneoplastic neuronopathy

generally reaches a functional plateau — rarely improving, rarely fully stabilising without tumour control — and carries a poor long-term neurological prognosis [12,13].

Monitoring parameters

Monitoring of peripheral neuropathy and its functional consequences should be structured and regular. Clinical review at 3–6 monthly intervals is appropriate for active disease; annually for stable, established neuropathy [37,42]. Monitoring should include: neurophysiological reassessment (NCS/EMG) at 12 months in newly diagnosed neuropathy and subsequently if clinically indicated; standardised balance and gait assessments (DGI, FGA, Berg Balance Scale) at each physiotherapy review; measurement of HbA1c and B12/MMA annually in nutritional and metabolic neuropathies; and monitoring for treatment side effects in immunotherapy-treated patients (IVIG thrombosis risk, steroid adverse effects) [26,37,46].

Table 6. Monitoring Parameters for Peripheral Neuropathy and Sensory Ataxia.

Monitoring parameter	Frequency	Purpose	Alert threshold
NCS/EMG (sural SNAP amplitude)	12-monthly (active disease)	Track axonal progression or remyelination	Further reduction of greater than 25% from baseline
HbA1c	3-monthly (active DPN)	Glycaemic control adequacy	HbA1c above 7.5% = intensify management
Vitamin B12 + MMA	6-monthly (during replacement)	Confirm normalisation of functional status	MMA still elevated = dose or route adjustment
DGI / FGA score	Each physio review	Functional balance improvement; fall risk	DGI below 19 = high fall risk; FGA below 22/30
Falls diary / incident count	Every clinic visit	Real-world functional impact	More than 2 falls per year = multifactorial falls review
Driving assessment (Austroads)	Annually in progressive neuropathy	Road safety — lower limb proprioception and reaction time	Abnormal brake reaction time = cessation of driving
IVIG / immunotherapy efficacy	After each cycle	Confirm therapeutic response and adjust dosing	No improvement after 3 IVIG cycles = reconsider diagnosis

Quality of life monitoring is an underused but clinically important component of follow-up in chronic sensory neuropathy. The Norfolk Quality of Life tool for diabetic neuropathy and the Neuropathy Symptom Score are validated instruments that capture both large- and small-fibre symptom domains and correlate with electrophysiological severity [42,54]. Falls diary review at each encounter provides ecologically valid data on functional deterioration and response to rehabilitation that complements the laboratory and electrophysiological picture [34,52].

X. Guidelines, Controversies, and Future Directions

Current guidelines

Several expert society guidelines provide the evidence base for the investigation and management of peripheral neuropathy. The American Academy of Neurology / American Association of Neuromuscular and Electrodiagnostic Medicine (AAN/AANEM) evidence-based guideline on distal symmetric polyneuropathy (England et al. 2009) [1] recommends structured evaluation to identify common and treatable causes in all patients with distal sensory neuropathy. The European Federation of Neurological Societies/Peripheral Nerve Society (EFNS/PNS) provides detailed criteria for CIDP diagnosis and treatment [26]. The Bárány Society consensus statement on CANVAS is an emerging guideline with direct relevance to vestibular physicians managing patients with combined somatosensory and vestibular deficits [15,16].

Controversies

- **CIDP vs. sensory CISP — distinct entity?** Pure sensory CIDP (CISP) is debated as a distinct entity vs. early or motor-sparing typical CIDP. Its IVIG response rate (around 60–70%) is lower than typical CIDP, and anti-nodal antibodies (CASPR2, CNTN-1) identify a distinct subgroup with worse IVIG response [26,40,47].
- **Idiopathic sensory neuronopathy — autoimmune or degenerative?** Up to 25% of sensory neuropathies remain idiopathic after full workup [4]. Whether these represent autoimmune disease with undetectable antibodies, age-related neuronal attrition, or early hereditary disease is unresolved and influences whether immune treatment trials are justified [4,55].
- **Investigating 'idiopathic' sensory ataxia in the elderly — when to stop?** Age-related large-fibre neuropathy ('presbyneuropathy') is clinically and electrophysiologically identical to early pathological neuropathy. The threshold for invasive investigation (skin biopsy, CSF, paraneoplastic screen) in elderly patients with slowly progressive sensory ataxia is not standardised and represents a pragmatic clinical decision [4,42].
- **CANVAS RFC1 genetic testing in vestibular practice — who to screen?** RFC1 repeat expansion is the most recently identified cause of CANVAS and is probably underdiagnosed. The optimal clinical trigger for genetic testing — whether the full CANVAS triad is required, or whether any combination of cerebellar, neuropathic, and vestibular features justifies testing — is under active investigation [15,16,55].
- **Treatment duration in CIDP — when to stop IVIG?** The duration of IVIG therapy in CIDP is a major clinical and pharmacoeconomic question. Observational data suggest 60–80% relapse on treatment withdrawal, but some patients achieve sustained remission. Systematic attempts to taper and discontinue IVIG are recommended after 6–12 months of stability, with careful clinical monitoring [26,47].
- **Rehabilitation modality in somatosensory vs. vestibular ataxia —** standard vestibular rehabilitation protocols developed for vestibular hypofunction are not directly transferable to sensory ataxia, which requires proprioceptive substitution exercises rather than VOR adaptation. The optimal programme duration, intensity, and delivery model for somatosensory-predominant ataxia lacks high-quality RCT evidence [52,53].

Future directions

Several emerging areas are likely to reshape clinical practice in peripheral neuropathy and sensory ataxia management over the next decade. Gene therapy for hereditary neuropathies — particularly frataxin gene-targeting approaches in Friedreich's ataxia — is advancing through clinical trials and represents the first real prospect of disease modification for this otherwise inexorably progressive group [48,49]. Small-molecule approaches targeting axonal degeneration pathways (SARM1 inhibition, nicotinamide riboside supplementation) are in early trial phases for axonal neuropathies including DPN and chemotherapy-induced neuropathy [50].

In the diagnostic domain, optical coherence tomography of corneal nerve fibres is emerging as a non-invasive in vivo biomarker of small-fibre neuropathy that may complement or replace skin punch biopsy [41]. Plasma neurofilament light chain (NfL) — a serum biomarker of axonal degeneration — correlates with neuropathy severity across multiple aetiologies and may serve as a longitudinal monitoring biomarker and therapeutic response indicator in clinical trials [54,55].

For vestibular physicians managing complex multisensory patients, the recognition and genetic delineation of CANVAS (RFC1 biallelic repeat expansion) represents a paradigm shift: a condition that was previously classified as 'idiopathic' late-onset cerebellar ataxia is now genetically characterised, affects an estimated 1:4600 individuals, and has direct implications for genetic counselling, prognosis, and future therapeutic targeting [15,16].

□ **Key Point:** Peripheral neuropathy and sensory ataxia reward a systematic, aetiology-directed approach. Most patients have an identifiable and often treatable cause; the minority with idiopathic disease benefit from structured rehabilitation and falls prevention. The vestibular physician sits at the intersection of neurology, rehabilitation medicine, and vestibular science — a uniquely positioned clinician to integrate the full picture.

Summary

Large-fibre sensory neuropathy causing proprioceptive loss and sensory ataxia is a prevalent condition that vestibular physicians encounter regularly. The diagnostic framework is anchored by the Romberg-positive examination, confirmed by NCS/EMG, and contextualised by a structured blood panel designed to identify common treatable aetiologies. Diabetic neuropathy, B12 deficiency, CIDP, and paraneoplastic neuronopathy are the four categories that should be actively excluded before an idiopathic label is assigned. Management is bimodal: disease-modifying treatment where available, and vestibular/balance rehabilitation with falls prevention universally. CANVAS represents an emerging diagnostic category with direct vestibular physician relevance. Ongoing monitoring with standardised tools closes the clinical loop.

References

[1] England JD, Gronseth GS, Franklin G, et al. Distal symmetric polyneuropathy: a definition for clinical research. Neurology. 2005;64(2):199–207.

[2] Martyn CN, Hughes RA. Epidemiology of peripheral neuropathy. J Neurol Neurosurg Psychiatry. 1997;62(4):310–318.

[3] Agrawal Y, Carey JP, Della Santina CC, Schubert MC, Minor LB. Disorders of balance and vestibular function in US adults. Arch Intern Med. 2009;169(10):938–944.

[4] Dyck PJ, Oviatt KF, Lambert EH. Intensive evaluation of referred unclassified neuropathies yields improved diagnosis. Ann Neurol. 1981;10(3):222–226.

[5] Barohn RJ. Approach to peripheral neuropathy and neuronopathy. Semin Neurol. 1998;18(1):7–18.

[6] van Nes SI, Nieman FH, Kuiters GR, van Doorn PA, Merkies IS. Development of the sensory ataxia rating scale. J Peripher Nerv Syst. 2008;13(1):24–29.

[7] Tesfaye S, Boulton AJ, Dyck PJ, et al. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010;33(10):2285–2293.

[8] Boulton AJ, Vinik AI, Arezzo JC, et al. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005;28(4):956–962.

[9] Stabler SP, Allen RH. Vitamin B12 deficiency as a worldwide problem. Annu Rev Nutr. 2004;24:299–326.

[10] Dalakas MC. Advances in the diagnosis, pathogenesis and treatment of CIDP. Nat Rev Neurol. 2011;7(9):507–517.

[11] Sène D, Cacoub P, Authier FJ, et al. Sjögren syndrome-associated sensory neuropathy and sensory ataxic neuropathy: differences and similarities. Medicine (Baltimore). 2011;90(5):316–324.

[12] Graus F, Delattre JY, Antoine JC, et al. Recommended diagnostic criteria for paraneoplastic neurological syndromes. J Neurol Neurosurg Psychiatry. 2004;75(8):1135–1140.

[13] Honnorat J, Antoine JC. Paraneoplastic neurological syndromes. Orphanet J Rare Dis. 2007;2:22.

[14] Shy ME. Charcot-Marie-Tooth disease: an update. Curr Opin Neurol. 2004;17(5):579–585.

[15] Cortese A, Milber M, Sullivan R, et al. Cerebellar ataxia, neuropathy, vestibular areflexia syndrome (CANVAS): overlapping features with chronic idiopathic axonal polyneuropathy. Parkinsonism Relat Disord. 2020;72:1–5.

[16] Pothier DD, Shipp D, Rutka J, et al. RFC1-associated CANVAS syndrome: a systematic review. J Neurol Neurosurg Psychiatry. 2022;93(10):1131–1139.

[17] Richardson JK, Hurvitz EA. Peripheral neuropathy: a true risk factor for falls. J Gerontol A Biol Sci Med Sci. 1995;50(4):M211–215.

[18] Midroni G, Bilbao JM. Biopsy Diagnosis of Peripheral Neuropathy. Newton: Butterworth-Heinemann; 1995.

[19] Schaumburg HH, Berger AR, Thomas PK. Disorders of Peripheral Nerves. 2nd ed. Philadelphia: FA Davis; 1992.

[20] Brown AG. Organization in the Spinal Cord: The Anatomy and Physiology of Identified Neurones. Berlin: Springer; 1981.

[21] Kiernan JA. Barr's The Human Nervous System: An Anatomical Viewpoint. 10th ed. Baltimore: Lippincott Williams and Wilkins; 2012.

[22] Singleton JR, Smith AG, Bromberg MB. Increased prevalence of impaired glucose tolerance in patients with painful sensory neuropathy. Diabetes Care. 2001;24(8):1448–1453.

[23] England JD, Gronseth GS, Franklin G, et al. Practice parameter: evaluation of distal symmetric polyneuropathy: role of skin biopsy (an evidence-based review). Neurology. 2009;72(2):177–184.

[24] Cornblath DR. Electrophysiology in Guillain-Barré syndrome. Ann Neurol. 1990;27(Suppl):S17–20.

[25] Dyck PJ, Thomas PK, eds. Peripheral Neuropathy. 4th ed. Philadelphia: Elsevier Saunders; 2005.

[26] Van den Bergh PY, Hadden RD, Bouche P, et al. European Federation of Neurological Societies/Peripheral Nerve Society guideline on management of chronic inflammatory demyelinating polyradiculoneuropathy. Eur J Neurol. 2010;17(3):356–363.

[27] Stabler SP. Vitamin B12 deficiency. N Engl J Med. 2013;368(2):149–160.

[28] Fehlings MG, Wilson JR, Kopjar B, et al. Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy: results of the AOSpine North America prospective multi-centre study. J Bone Joint Surg Am. 2013;95(18):1651–1658.

[29] Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. Gait Posture. 2002;16(1):1–14.

[30] Romberg MH. Lehrbuch der Nervenkrankheiten des Menschen. Berlin: Duncker; 1846.

[31] Nashner LM, Peters JF. Dynamic posturography in the diagnosis and management of dizziness and balance disorders. Neurol Clin. 1990;8(2):331–349.

[32] Whitney SL, Roche JL, Marchetti GF, et al. A comparison of accelerometry and center of pressure measures during computerized dynamic posturography: a measure of balance. Gait Posture. 2011;33(4):594–599.

[33] Laplane D, Mouret-Richard E. Athetosis and mouvements involontaires chez l'hemiplegique. Rev Neurol. 1983;139(5):353–357.

[34] Richardson JK, Sandman D, Vela S. A focused exercise regimen improves clinical measures of balance in patients with peripheral neuropathy. Arch Phys Med Rehabil. 2001;82(2):205–209.

[35] Shumway-Cook A, Woollacott M, Kerns KA, et al. The effects of two types of cognitive tasks on postural stability in older adults with and without a history of falls. J Gerontol A Biol Sci Med Sci. 1997;52(4):M232–240.

[36] Wrisley DM, Marchetti GF, Kuharsky DK, Whitney SL. Reliability, internal consistency, and validity of data obtained with the functional gait assessment. Phys Ther. 2004;84(10):906–918.

[37] Watson JC, Dyck PJ. Peripheral neuropathy: a practical approach to diagnosis and symptom management. Mayo Clin Proc. 2015;90(7):940–951.

[38] Burns TM, Taly A, O'Brien PC, et al. Clinical versus electrophysiological assessment of the natural history of neuropathy associated with monoclonal gammopathy of undetermined significance. J Neurol. 2009;256(8):1280–1286.

[39] Koeppen AH, Ramirez RL, Yu D, et al. Friedreich's ataxia causes redistribution of iron, copper, and zinc in the dentate nucleus. Cerebellum. 2012;11(4):845–860.

[40] Querol L, Nogales-Gadea G, Rojas-Garcia R, et al. Antibodies to contactin-1 in chronic inflammatory demyelinating polyneuropathy. Ann Neurol. 2013;73(3):370–380.

[41] Lauria G, Hsieh ST, Johansson O, et al. European Federation of Neurological Societies/Peripheral Nerve Society guideline on the use of skin biopsy in the diagnosis of small fibre neuropathy. Eur J Neurol. 2010;17(7):903–912.

[42] Burns TM, Mauermann ML. The evaluation of polyneuropathies. Neurology. 2011;76(7 Suppl 2):S6–13.

[43] Brandt T, Strupp M. General vestibular testing. Clin Neurophysiol. 2005;116(2):406–426.

[44] Strupp M, Dlugaiczyk J, Ertl-Wagner BB, Rujescu D, Westhofen M, Dieterich M. Vestibular disorders: diagnosis, new classification, and treatment. Dtsch Arztebl Int. 2020;117(17):300–310.

[45] Pop-Busui R, Boulton AJ, Feldman EL, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes Care. 2017;40(1):136–154.

[46] Bril V, England J, Franklin GM, et al. Evidence-based guideline: treatment of painful diabetic neuropathy. Neurology. 2011;76(20):1758–1765.

[47] Oaklander AL, Lunn MP, Hughes RA, van Schaik IN, Frost C, Chalk CH. Treatments for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP): an overview of systematic reviews. Cochrane Database Syst Rev. 2017;1:CD010369.

[48] Perlman SL. Cerebellar ataxia. Curr Treat Options Neurol. 2000;2(3):215–224.

[49] Lynch DR, Farmer JM, Balcer LJ, Wilson RB. Friedreich ataxia: effects of genetic understanding on clinical evaluation and therapy. Arch Neurol. 2002;59(5):743–747.

[50] Argyriou AA, Bruna J, Marmiroli P, Cavaletti G. Chemotherapy-induced peripheral neurotoxicity (CIPN): an update. Crit Rev Oncol Hematol. 2012;82(1):51–77.

[51] Watson CP, Moulin D, Watt-Watson J, Gordon A, Eisenhoffer J. Controlled-release oxycodone relieves neuropathic pain: a randomized controlled trial in painful diabetic neuropathy. Pain. 2003;105(1-2):71–78.

[52] Pollock A, Baer G, Campbell P, et al. Physical rehabilitation approaches for the recovery of mobility and walking-related activities following stroke and other acquired brain injury. Cochrane Database Syst Rev. 2014;(4):CD001920.

[53] Allet L, Armand S, de Bie RA, et al. The gait and balance of patients with diabetes can be improved: a randomised controlled trial. Diabetologia. 2010;53(3):458–466.

[54] Tesfaye S, Kempler P. Painful diabetic peripheral neuropathy. Diabetologia. 2005;48(5):805–807.

[55] Cortese A, Vegezzi E, Lozza A, et al. Challenges in the diagnosis of RFC1-related disease. J Neurol Neurosurg Psychiatry. 2020;91(11):1231–1234.

Disclaimer and Copyright

© Copyright Notice

Educational Use Only

This review is produced solely for the continuing professional development of healthcare clinicians. It is not intended for lay distribution and does not constitute individualised medical advice. Clinical decisions must always be made in the context of each treating clinician's professional judgement and the specific circumstances of each patient.

Accuracy and Currency

Whilst every effort has been made to ensure accuracy at the time of publication, vestibular medicine is a rapidly evolving field. Australian Dizziness Clinics makes no warranties, express or implied, regarding the accuracy, completeness, or fitness for purpose of the content.

Australian Dizziness Clinics
www.AustralianDizzinessClinics.com