Ramsay Hunt Syndrome (Herpes Zoster Oticus):

A Vestibular Physician's Deep Review of Mechanism, Diagnosis, and Management

Vestibular Medicine for Vestibular Physicians

Peripheral Vestibular Pathology — Module 2.7

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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 vestibular physiotherapists working at the deep end of peripheral 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 to 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

Ramsay Hunt syndrome (RHS), also termed herpes zoster oticus, is the reactivation of latent varicella-zoster virus (VZV) within the geniculate ganglion of the facial nerve, producing the clinical triad of acute peripheral facial palsy, ipsilateral otalgia, and a vesicular eruption of the auricle or oropharynx [2,3]. James Ramsay Hunt first localised the lesion to the geniculate ganglion in 1907 and described the syndrome that now carries his name — specifically Hunt type II, distinguishing it from his cerebellar (type I) and peripheral neuritis (type III) syndromes [1]. For the vestibular physician the defining interest of RHS lies not in the facial palsy, which is shared with Bell's palsy, but in its frequent and frequently under-recognised audiovestibular involvement [2,11].

RHS is the second most common cause of atraumatic peripheral facial paralysis after Bell's palsy, yet it is far less common in absolute terms, with an estimated annual incidence in the order of five cases per 100,000 [2,4]. It accounts for a variable proportion of facial palsies across series — figures from roughly 7 to 18 per cent are reported, with large cohort estimates near 12 per cent — and importantly a substantial minority of cases initially labelled Bell's palsy are in fact zoster sine herpete, RHS without a visible rash [12,14]. Herpes zoster as a whole is common and increasing with population ageing, with population incidence of around three to four per 1,000 person-years, of which facial or geniculate involvement represents only one to two per cent [38,39].

The disease is predominantly one of the sixth to eighth decades, mirroring the age-related decline in VZV-specific cell-mediated immunity that drives reactivation; it is rare in children, in whom incidence is approximately 2.7 per 100,000 per year, and shows no consistent sex predilection [5,43]. Within a dedicated vertigo clinic RHS is a rare presenting diagnosis — under 0.1 per cent of attendances in large series — but the prevalence of dizziness within RHS itself is high, reported in around 31 per cent of patients compared with under 9 per cent of Bell's palsy patients, underscoring that cochleovestibular symptoms are a genuine and discriminating feature rather than an incidental one [9].

Table 1. Epidemiology of Ramsay Hunt syndrome at a glance.

Parameter	Figure	Refs
Annual incidence	~5 per 100,000 (range 1–5)	[2,4]
Share of atraumatic facial palsies	Second to Bell's; ~7–18% (≈12% in large cohorts)	[2,14]
Zoster sine herpete	Up to one-third lack rash at presentation	[12]
Typical age	6th–8th decade; paediatric ~2.7/100,000/yr	[5,43]
Sex distribution	No consistent predilection	[4]
Dizziness frequency	~31% RHS vs ~9% Bell's palsy	[9]

□ **Key Point:** RHS is uncommon but clinically louder than Bell's palsy: vertigo, hearing loss and severe otalgia are intrinsic to the syndrome, and up to a third of cases present without the diagnostic rash.

Why RHS matters to the vestibular physician

Although the facial palsy dominates the clinical picture and drives the popular name, it is the audiovestibular limb of the disease that most often reaches the vestibular physician, and frequently after the acute window has closed. Patients may present with isolated acute vertigo and hearing loss in the days before the rash and palsy declare themselves, or with persistent imbalance and oscillopsia weeks after an episode that was treated as Bell's palsy elsewhere [2,9]. Recognising RHS within an undifferentiated acute vestibular syndrome therefore depends less on the dermatological signs and more on a disciplined search for the associated otalgia, sensorineural hearing loss and lower cranial nerve features that betray a zoster aetiology [3,11].

Epidemiological context and the ageing population

The epidemiology of RHS cannot be separated from that of herpes zoster itself, whose incidence rises steeply with age as varicella-zoster-virus-specific cell-mediated immunity wanes [4,39]. Population

studies place the incidence of zoster at roughly three to four episodes per 1,000 person-years, increasing several-fold beyond the seventh decade, with a lifetime risk approaching one in three [38,39]. Against this background the small fraction of episodes that localise to the geniculate region generates the observed RHS incidence of around five per 100,000 per year, and the same demographic forces that are increasing the zoster burden are expected to increase the absolute number of RHS cases despite the mitigating effect of vaccination [4,38].

The under-recognition of RHS has a measurable epidemiological footprint. Because zoster sine herpete is captured poorly by rash-based case definitions, and because audiovestibular symptoms are often attributed to coincidental causes, contemporary estimates that incorporate virological testing suggest varicella-zoster virus accounts for a larger share of acute peripheral facial palsy than the classical figures imply [12,14]. This matters for the vestibular physician because it reframes RHS from a rare curiosity into a recurring differential in the acute vertigo-and-hearing-loss presentation [9,11].

A note on nomenclature avoids confusion in the records. The eponym honours James Ramsay Hunt, who described three unrelated conditions; the syndrome of interest here is Hunt type II, herpes zoster oticus with facial palsy, and should not be conflated with his cerebellar syndrome (type I) or his peripheral neuritis of the extremities (type III) [1]. In contemporary usage the unqualified term Ramsay Hunt syndrome is taken to mean type II, and herpes zoster oticus is an acceptable synonym [2,3].

II. Pathophysiology — VZV Reactivation in the Geniculate Ganglion

Primary VZV infection (varicella) establishes lifelong latency in cranial nerve and dorsal root ganglia, including the geniculate ganglion — the sensory ganglion of the nervus intermedius lying at the first genu of the facial nerve within the temporal bone [1,15]. Reactivation, precipitated by immunosenescence, intercurrent illness, physiological stress or iatrogenic immunosuppression, produces an inflammatory ganglionitis characterised histologically by haemorrhagic necrosis and dense perivascular and intraneural lymphocytic infiltration [2,15]. The virus is transported anterogradely along the sensory fibres of the nervus intermedius to the cutaneous territory of the auricle — Hunt's zone — producing the characteristic vesicles [1,5].

The facial palsy is mechanistically a compartment phenomenon. Inflammatory oedema of the nerve within the rigid, non-distensible fallopian canal raises endoneurial pressure, compromises the vasa nervorum, and produces ischaemic demyelination and, in severe cases, axonal degeneration — a dual injury that is reflected in the worse prognosis of RHS relative to the predominantly oedematous neuropathy of Bell's palsy [24,29]. Intraoperative and imaging studies have confirmed swelling of the intratemporal segment in RHS, providing the rationale for decompression in selected refractory cases [24].

Audiovestibular involvement reflects two non-exclusive mechanisms. The first is contiguous inflammatory spread from the geniculate ganglion across the short distance separating the facial and vestibulocochlear nerves within the internal auditory canal [2,34]. The second, and arguably more important, is primary VZV reactivation within the spiral and vestibular ganglia themselves: VZV DNA has been demonstrated directly in these ganglia, and viral load correlates with the severity of cochleovestibular symptoms, explaining why hearing loss and vertigo can dominate or even precede the facial palsy [11,12,30]. VZV dormancy in non-neuronal satellite glial cells of these ganglia supports the concept of independent reactivation at multiple sites [11].

Wider cranial polyneuropathy is explained by Hunt's 'chain of ganglia' concept, in which the gasserian (V), geniculate (VII), petrous (IX) and jugular/plexiform (X) ganglia, together with the upper cervical dorsal root ganglia, form an interconnected network through which inflammation can spread either by contiguity or via a shared vascular supply [1,6]. VZV vasculopathy — the virus's well-documented tropism for vascular endothelium — is the unifying substrate, and in the immunocompromised can extend to a frank large- or small-vessel cerebral vasculopathy with stroke [10,16,42]. The order of cranial nerve involvement in descending frequency is VII, VIII, IX, X then V [6].

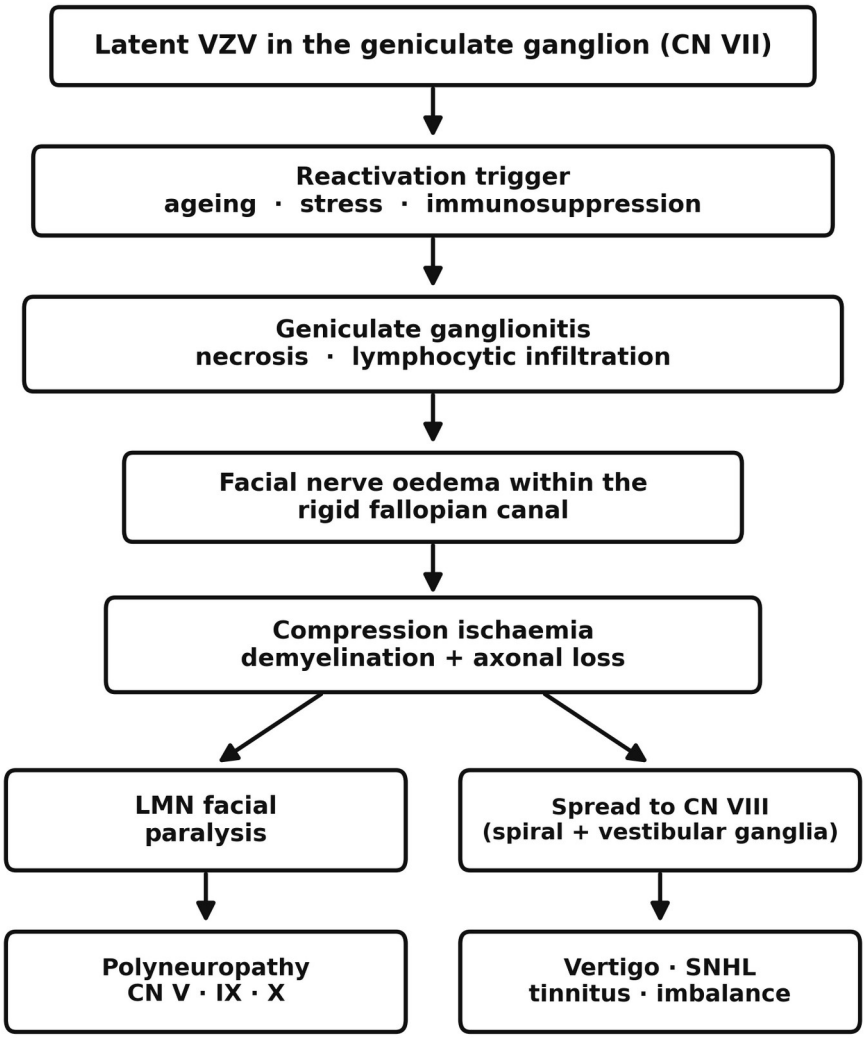
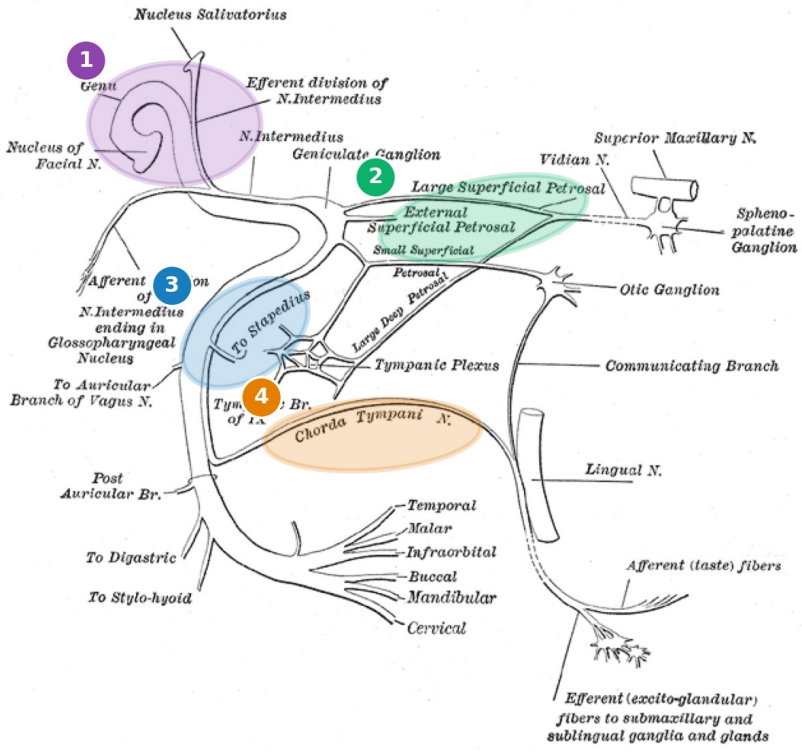


Figure 1. Pathophysiological cascade of Ramsay Hunt syndrome — from geniculate reactivation to audiovestibular involvement.

Source: Adapted from Sweeney and Gilden [2], Gilden et al. [15] and Kuhweide et al. [34].



Structures relevant to Ramsay Hunt syndrome

1. **Geniculate ganglion — VZV reactivation site**
2. **Greater petrosal n. — lacrimation (dry eye)**
3. **Nerve to stapedius — hyperacusis**
4. **Chorda tympani — taste & salivation**

Figure 2. The facial and intermediate (nervus intermedius) nerves, with the four structures most relevant to Ramsay Hunt syndrome highlighted: the geniculate ganglion (VZV reactivation site), greater petrosal nerve, nerve to stapedius and chorda tympani.

Source: Annotated by Australian Dizziness Clinics from Henry Gray, Anatomy of the Human Body (1918), Plate 788; original image public domain, via Wikimedia Commons.

□ **Clinical Insight:** Cochleovestibular symptoms in RHS are not merely 'spillover' from the facial nerve — they often reflect direct VZV reactivation in the spiral and vestibular ganglia, which is why vertigo and hearing loss can be the presenting complaint with the facial palsy following days later.

VZV vasculopathy and the immunocompromised host

Beyond the focal ganglionitis, VZV exhibits a marked tropism for the vascular endothelium of the small and large cerebral vessels, and this vasculopathy is the substrate for the most serious neurological complications of zoster [10,16]. In the immunocompetent it is usually subclinical, but in the immunocompromised it can produce a multifocal arteriopathy with ischaemic and haemorrhagic stroke, a presentation that should be considered whenever an RHS patient develops focal neurological deficits beyond the cranial nerves [10,42]. The same endothelial tropism plausibly contributes to the ischaemic component of the facial neuropathy itself, compounding the compressive injury within the fallopian canal [15].

The histopathological signature of the disease — haemorrhagic necrosis, perivascular cuffing and combined demyelination with axonal loss — explains why recovery is slower and less complete than in the predominantly oedematous Bell's palsy, and why electrophysiological evidence of dense axonal

degeneration carries such adverse prognostic weight [24,29]. This mechanistic asymmetry is the through-line that connects the pathology to the worse outcomes detailed later in this review [6,9].

The autonomic and secretomotor fibres carried by the nervus intermedius account for several of the less obvious features. Greater superficial petrosal nerve involvement reduces reflex tearing and contributes, with the motor lagophthalmos, to the dry and vulnerable eye, while chorda tympani involvement disturbs taste over the anterior tongue and alters submandibular and sublingual salivation [2,5]. Late aberrant regeneration of these autonomic fibres underlies gustatory lacrimation, the so-called crocodile-tears phenomenon, which can emerge months after the acute illness and is a visible reminder that reinnervation in RHS is frequently disordered rather than simply incomplete [46].

III. Clinical Features and Facial Nerve Topodiagnosis

The classic triad is acute ipsilateral lower motor neuron facial paralysis, severe otalgia, and a vesicular rash of the external ear, canal, tympanic membrane or oropharynx [2,3]. Unlike the upper motor neuron pattern of a hemispheric stroke, the forehead is involved, and patients cannot wrinkle the brow, fully close the eye (lagophthalmos) or maintain oral competence. The otalgia is characteristically deep, burning and disproportionate, frequently preceding the rash and the palsy by several days [2,5].

The temporal relationship of the triad is diagnostically treacherous. The rash may precede, coincide with or follow the palsy; vesicles appear after the onset of weakness in roughly one in seven cases and may be absent altogether in zoster sine herpete, a presentation that requires virological confirmation and that accounts for a meaningful share of apparent Bell's palsy [12,44]. Careful inspection of the concha, external canal, tympanic membrane, palate and anterior two-thirds of the tongue is therefore mandatory in every acute facial palsy.

The level of the lesion along the facial nerve can be inferred from the associated deficits, a topodiagnostic exercise familiar to the neuro-otologist. Involvement at or proximal to the geniculate ganglion produces otalgia and auricular vesicles; greater petrosal nerve involvement reduces lacrimation and causes a dry eye; nerve-to-stapedius involvement produces hyperacusis; chorda tympani involvement produces dysgeusia of the anterior tongue and altered salivation; and a purely distal lesion at the stylomastoid foramen yields isolated motor paralysis [2,5]. Severity is graded with the House-Brackmann scale (I to VI), supplemented where synkinesis and regional function matter by the Sunnybrook system and the Facial Nerve Grading System 2.0 [17,18,19,45].

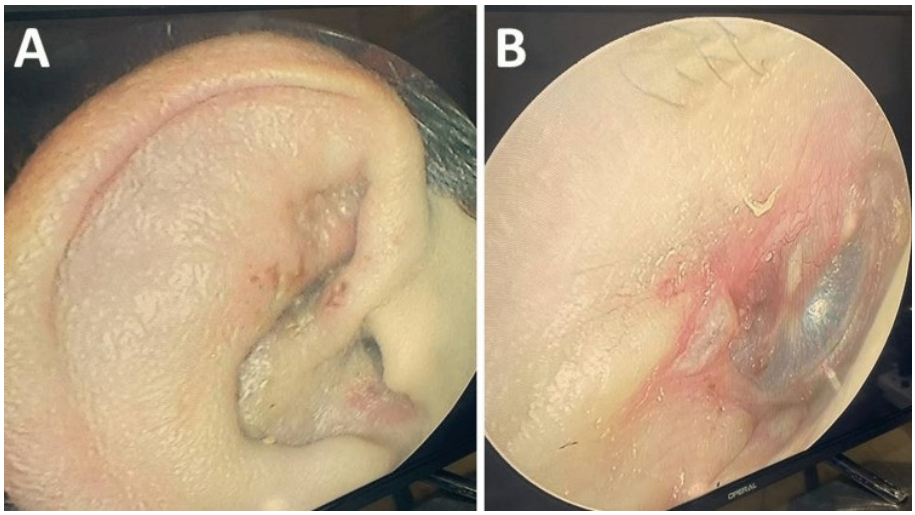


Figure 3. Herpes zoster oticus: erythematous vesicular eruption over the auricle, external auditory canal and tympanic membrane (Hunt's zone).

Source: Al-Ani RM. Ramsay Hunt syndrome with cranial polyneuropathy and delayed facial nerve palsy. Cureus. 2022;14(7):e27434; reproduced under CC BY 4.0.

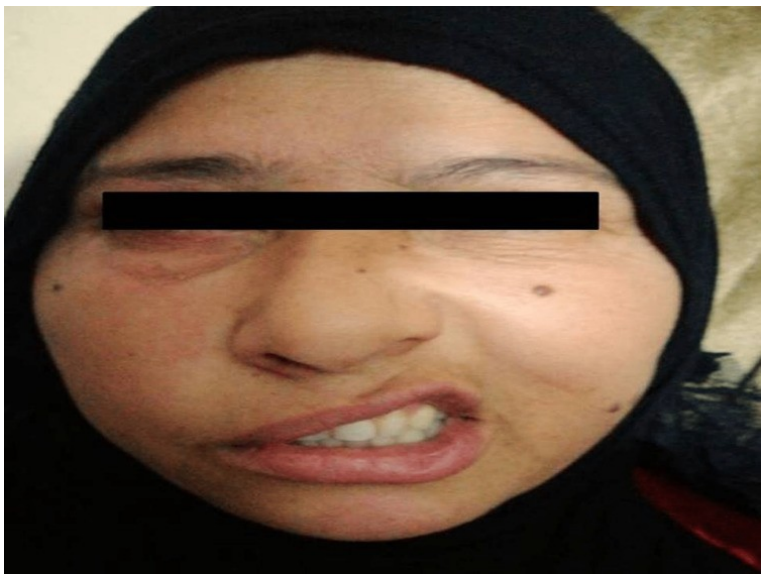


Figure 4. Right lower motor neuron facial paralysis in Ramsay Hunt syndrome: flattened nasolabial fold, drooping oral angle and loss of forehead wrinkling.

Source: Al-Ani RM. Cureus. 2022;14(7):e27434; reproduced under CC BY 4.0.

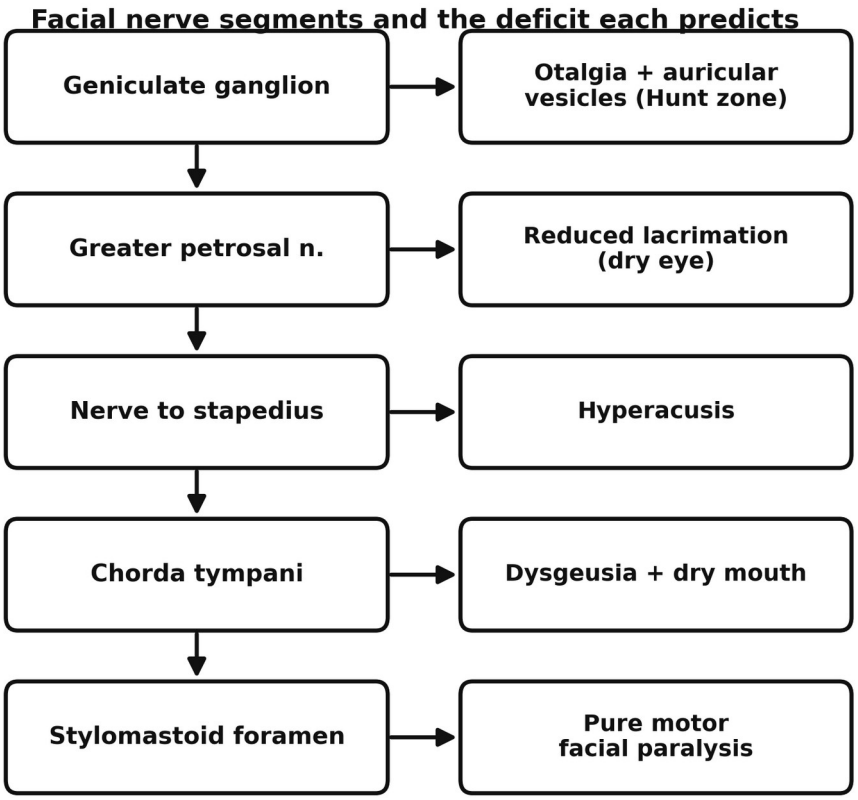


Figure 5. Facial nerve topodiagnosis — the lesion level and the deficit each segment predicts.

Source: Adapted from Sweeney and Gilden [2] and Coulson et al. [9].

Audiovestibular features comprise sensorineural hearing loss (mild to profound, high frequencies preferentially affected), tinnitus and true vertigo with nausea, vomiting and spontaneous nystagmus

[11,31]. Vestibular-evoked myogenic potential studies localise the lesion within the labyrinthine end-organs and their afferents, frequently demonstrating inferior vestibular nerve and saccular involvement in addition to the superior division [31,32]. Lower cranial nerve involvement (IX and X) may produce dysphagia, hoarseness and aspiration risk, and constitutes a cranial polyneuropathy that carries a worse prognosis for facial recovery [6].

Table 2. House-Brackmann facial nerve grading scale.

Grade	Severity	Clinical description
I	Normal	Normal facial function in all areas
II	Mild dysfunction	Slight weakness on close inspection; complete eye closure with effort
III	Moderate	Obvious but not disfiguring asymmetry; complete eye closure with effort
IV	Moderately severe	Disfiguring asymmetry; incomplete eye closure
V	Severe	Barely perceptible motion; incomplete eye closure
VI	Total paralysis	No movement

□ **Important:** Up to one-third of RHS presents with the rash appearing after the palsy, and a subset never develops a rash at all (zoster sine herpete). Severe otalgia, hearing loss or vertigo accompanying an apparent Bell's palsy should prompt VZV testing rather than reassurance.

Cranial polyneuropathy and the chain of ganglia

A substantial minority of patients have involvement beyond the seventh and eighth nerves, and recognising this polyneuropathic phenotype matters because it predicts poorer facial recovery [6]. Dysphagia, hoarseness and a depressed gag reflex point to ninth and tenth nerve involvement, facial sensory disturbance to the fifth, and the aggregate picture reflects inflammation tracking through Hunt's interconnected chain of cranial and upper cervical ganglia [1,6]. The vestibular physician assessing such a patient should examine the lower cranial nerves explicitly and consider the aspiration risk that accompanies a combined ninth and tenth nerve palsy [6].

Where the rash is absent, the diagnostic burden falls on virology, and saliva polymerase chain reaction has emerged as a practical non-invasive route to demonstrating VZV reactivation in zoster sine herpete [12]. A negative result does not wholly exclude the diagnosis when clinical suspicion is high, and paired serology or cerebrospinal fluid analysis may be required in equivocal or central presentations [12,42].

Quantifying the vestibular lesion has both diagnostic and rehabilitative value. The video head impulse test interrogates each semicircular canal individually and commonly demonstrates a broader, multi-canal pattern of vestibulo-ocular reflex loss than caloric irrigation alone, consistent with reactivation distributed across the vestibular ganglion rather than confined to the superior division [33]. Cervical and ocular vestibular-evoked myogenic potentials add saccular and utricular information and, taken together with the head impulse and caloric data, allow the clinician to map which afferents are affected and to set realistic expectations for compensation [31,32]. This functional mapping is the kind of detail that distinguishes a vestibular-physician work-up from a purely otological one [2].

IV. Diagnosis — Clinical, Virological and Audiovestibular

When acute peripheral facial palsy coexists with an ipsilateral zoster oticus rash, the diagnosis is clinical and requires no confirmatory testing [2,3]. The diagnostic challenge is the incomplete or rashless presentation. Here the combination of severe otalgia, sensorineural hearing loss, tinnitus or vertigo should raise suspicion and trigger virological confirmation [12,44]. Detection of VZV DNA by polymerase chain reaction — from vesicle fluid where present, or non-invasively from saliva, and from cerebrospinal fluid when central involvement is suspected — is highly sensitive and specific and is the investigation of choice for zoster sine herpete [12]. Where PCR is unavailable, a fourfold or greater rise in anti-VZV IgG across paired acute and convalescent sera, or detectable IgM, provides supporting evidence [12].

Audiovestibular assessment is integral rather than optional. Pure-tone and speech audiometry document the type and severity of the typically high-frequency sensorineural loss and establish a baseline for counselling and rehabilitation [11]. Caloric or video-nystagmography testing demonstrates unilateral canal paresis and any spontaneous or positional nystagmus, while the video head impulse test (vHIT) quantifies vestibulo-ocular reflex gain across all six semicircular canals and frequently reveals more widespread, multi-canal impairment than caloric testing alone [31,33]. Cervical and ocular VEMPs localise saccular and utricular dysfunction and help distinguish inferior from superior vestibular nerve involvement [31,32].

A pivotal point for the vestibular physician is the dissociation between functional and radiological findings: patients with objective vestibular deficits on nystagmography may show facial nerve enhancement on MRI with no discernible vestibulocochlear nerve abnormality [35]. Functional vestibular testing must therefore not be omitted on the basis of a normal-appearing eighth nerve on imaging. Electroneuronography and needle electromyography quantify the degree of axonal degeneration and are prognostic rather than diagnostic, with greater than 90 per cent degeneration within the first fortnight signalling a poor outlook and prompting consideration of surgery in some centres [37].

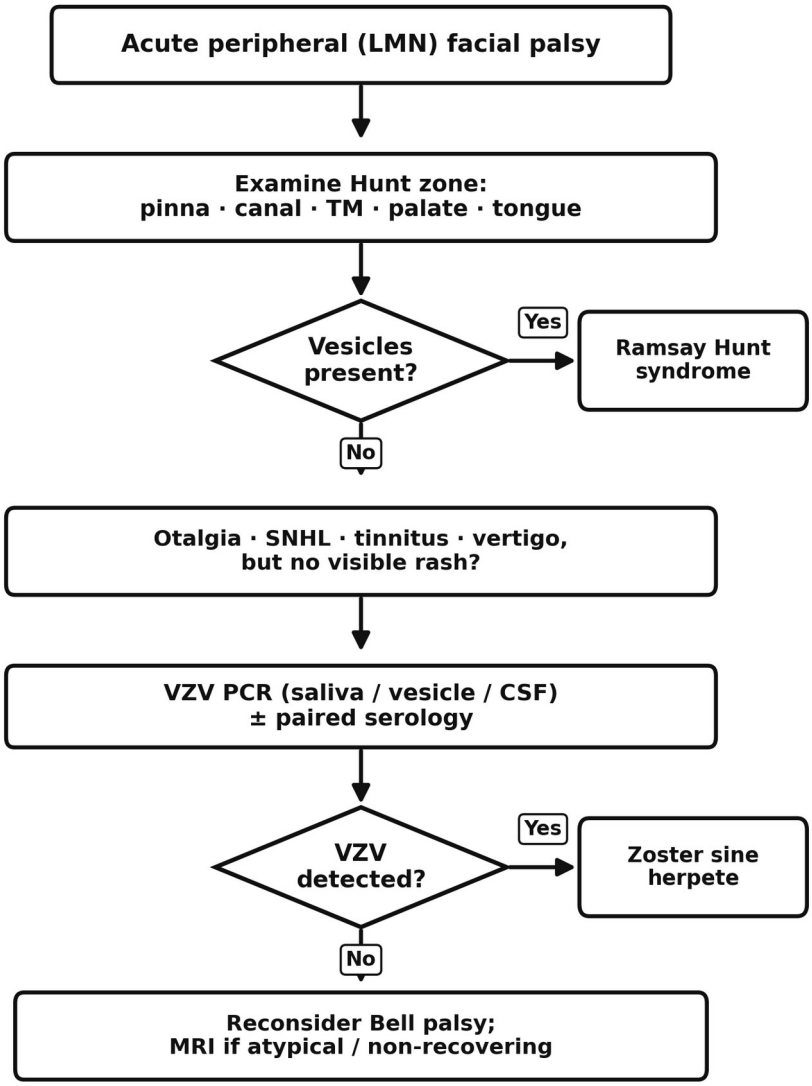


Figure 6. Diagnostic algorithm for suspected Ramsay Hunt syndrome, including the zoster sine herpete pathway.
Source: Adapted from Crouch et al. [3] and Furuta et al. [12].

□ **Clinical Pearl:** In a facial palsy with prominent vertigo or hearing loss but no rash, send a saliva VZV PCR. A positive result reclassifies the patient as zoster sine herpete and mandates antiviral therapy that pure Bell's palsy would not receive.

A pragmatic testing sequence

In practice the investigations serve three distinct purposes — confirming aetiology, characterising the audiovestibular deficit, and stratifying prognosis — and ordering them with that logic avoids both under- and over-investigation [2,12]. Aetiological confirmation by VZV polymerase chain reaction is reserved for the rashless or atypical case; audiometry, video head impulse testing and vestibular-evoked myogenic potentials characterise the end-organ and nerve involvement and establish a rehabilitation baseline; and electroneuronography is added only where the palsy is complete and the result might influence a decision about decompression [31,33,37]. Routine gadolinium MRI is unnecessary in the typical rash-positive patient but is indispensable when a structural or vascular mimic is plausible [35,36].

Cerebrospinal fluid and central involvement

Lumbar puncture is not part of the routine work-up but becomes important when the clinical picture suggests central nervous system involvement — depressed conscious level, meningism, multifocal deficits or disproportionate headache — particularly in the immunocompromised [10,42]. The expected cerebrospinal fluid profile is a lymphocytic pleocytosis with mildly elevated protein and preserved glucose, and the diagnosis of central varicella-zoster virus disease is secured by detecting viral DNA on polymerase chain reaction or by demonstrating intrathecal synthesis of anti-varicella-zoster-virus antibody [12,42]. Identifying a varicella-zoster virus vasculopathy is consequential, as it mandates a prolonged course of intravenous antiviral therapy that differs materially from the management of uncomplicated RHS [10,16].

V. Investigations and the Role of Imaging

Imaging is not required to make the diagnosis in a typical rash-positive case, but gadolinium-enhanced MRI is valuable when the presentation is atypical, when recovery stalls, or when a structural mimic must be excluded [35,36]. The characteristic finding is enhancement of the facial nerve, most marked at the geniculate ganglion and labyrinthine segment, sometimes accompanied by enhancement of the vestibulocochlear nerve within the internal auditory canal and, in polyneuropathic cases, of the lower cranial nerves [35]. Enhancement is supportive but neither necessary nor sufficient, and its absence does not exclude the diagnosis [36].

The principal role of imaging is to exclude alternative pathology: a vestibular schwannoma or other cerebellopontine angle mass, a pontine or anterior inferior cerebellar artery territory infarct, demyelination, or neoplastic and granulomatous infiltration of the temporal bone [16,35]. In the immunocompromised patient with neurological signs, MRI and cerebrospinal fluid examination are indicated to identify VZV vasculopathy, meningitis or encephalitis, where cerebrospinal fluid typically shows a lymphocytic pleocytosis with elevated protein, and VZV PCR with intrathecal antibody synthesis confirms central nervous system disease [10,42].

Table 3. Investigations in Ramsay Hunt syndrome and their expected findings.

Investigation	Purpose	Expected finding in RHS
VZV PCR (saliva, vesicle, CSF)	Confirm aetiology, especially ZSH	VZV DNA detected
Paired VZV serology	Support diagnosis when PCR unavailable	≥4-fold IgG rise or detectable IgM
Pure-tone / speech audiometry	Quantify hearing loss	Ipsilateral SNHL, high-frequency
Caloric / videonystagmography	Assess horizontal canal / superior nerve	Unilateral canal paresis; nystagmus
Video head impulse test	Assess all six semicircular canals	Reduced VOR gain; corrective saccades
cVEMP / oVEMP	Localise saccular / utricular afferents	Absent or reduced responses
MRI with gadolinium	Exclude mimics; demonstrate neuritis	CN VII (geniculate) ± CN VIII enhancement

ENoG / EMG	Prognosis; decompression decision	Over 90% degeneration = poor prognosis

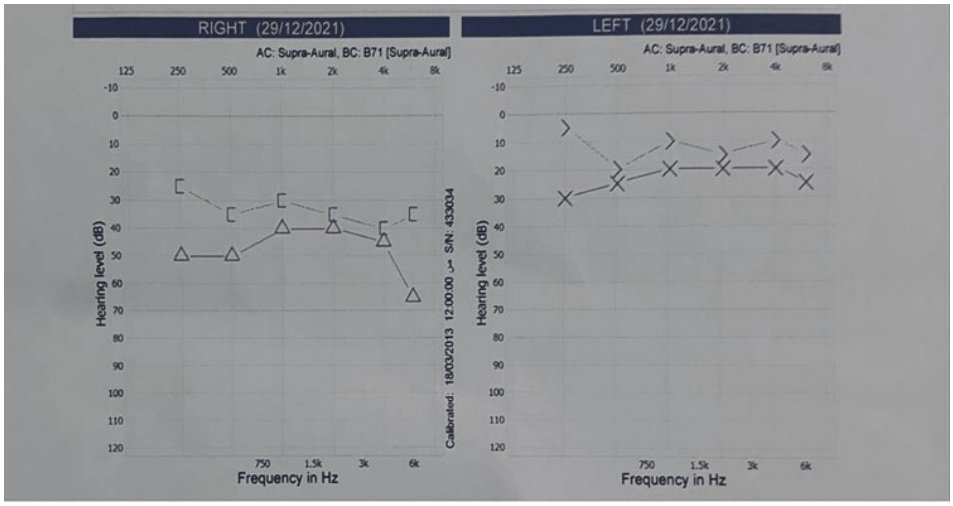


Figure 7. Pure-tone audiogram in Ramsay Hunt syndrome demonstrating ipsilateral sensorineural hearing loss.
Source: Al-Ani RM. Cureus. 2022;14(7):e27434; reproduced under CC BY 4.0.

□ **Key Point:** vHIT and VEMP frequently reveal multi-canal and otolith involvement that caloric testing alone misses, and functional deficits can exist despite a normal-appearing eighth nerve on MRI — test function, do not rely on imaging.

Resolving the Bell's palsy overlap

Because a meaningful share of apparent Bell's palsy is occult zoster, the practical question is not whether the two can always be separated clinically but how aggressively to test [12,29]. A defensible position for the vestibular physician is to treat any facial palsy accompanied by significant otalgia, hearing loss or vertigo as RHS until proven otherwise, and to send a saliva VZV polymerase chain reaction in that setting, since the therapeutic consequence — adding an antiviral within the treatment window — is both cheap and potentially decisive [12,22]. The corollary is that a purely motor facial palsy with none of these features can reasonably be managed as Bell's palsy, with steroids and watchful follow-up [13,23].

The central mimics deserve particular vigilance. A lateral pontine or anterior inferior cerebellar artery territory infarct can produce facial weakness, hearing loss and vertigo together, superficially resembling RHS but without vesicles and usually with additional crossed or cerebellar signs, and the distinction is made by attention to the pattern of associated deficits and by MRI with diffusion-weighted imaging [16,35]. The reassuring corollary is that the forehead-sparing of a true central facial palsy, when present, is a useful bedside discriminator from the forehead-involving lower motor neuron palsy of RHS [16].

VI. Differential Diagnosis

The single most important differential is Bell's palsy, which shares the lower motor neuron facial paralysis but lacks vesicles, produces only mild retro-auricular discomfort, rarely causes true vertigo or hearing loss, and carries a substantially better prognosis [13,14]. The distinction is not academic: the landmark randomised evidence in Bell's palsy supports corticosteroids while finding no benefit from aciclovir, whereas in RHS antiviral therapy is central — and the considerable overlap created by zoster sine herpete means a proportion of apparent Bell's palsy is in truth occult zoster [22,29]. A low threshold for VZV testing in the painful or audiovestibular case is the practical resolution of this overlap [12].

Other mimics include the vestibular schwannoma, in which facial weakness is late and the course progressive; acute otitis media or mastoiditis, with a bulging or perforated drum, fever and conductive loss; Lyme disease, with erythema migrans and tick exposure; and a central facial palsy from a pontine or AICA-territory infarct, in which the forehead may be relatively spared and other brainstem signs coexist [3,16,35]. Sarcoidosis, Guillain-Barre syndrome, temporal bone trauma and malignant otitis externa complete the list. Table 4 summarises the discriminating features most useful at the bedside.

Table 4. Differentiating Ramsay Hunt syndrome from common mimics.

Condition	Otalgia / pain	Key discriminator
Bell's palsy	Mild or absent	No vesicles; ~85% reach HB I; HSV-implicated
Zoster sine herpete	Severe	RHS features without rash; VZV PCR/serology positive
Vestibular schwannoma	Absent	Progressive SNHL; late facial weakness; MRI mass
AOM / mastoiditis	Present	Bulging/perforated TM, fever, conductive loss
Lyme disease	Variable	Erythema migrans; tick exposure; serology
Central (AICA/pontine)	Variable	Forehead may be spared; brainstem signs; MRI

Eye protection and pain in practice

The eye is the organ most likely to suffer lasting harm, and the combination of lagophthalmos, reduced lacrimation from greater petrosal nerve involvement and corneal hypoaesthesia creates a real risk of exposure keratitis and ulceration [40]. A simple regimen of preservative-free drops through the day, ointment and taping or a moisture chamber overnight, and a low threshold for ophthalmology review prevents most complications, and patients generally tolerate lubrication better than continuous taping [40]. Severe otalgia frequently outlasts the acute illness as postherpetic neuralgia, and early use of a gabapentinoid alongside conventional analgesia is reasonable in the patient with disproportionate pain [25].

The timing of rehabilitation matters as much as its content. Vestibular suppressants have a place only in the first few days of incapacitating vertigo, after which they impede the central compensation that drives recovery, and structured vestibular rehabilitation should take over with gaze-stabilisation and habituation exercises [47]. Facial neuromuscular retraining is best introduced once early reinnervation appears, typically with mime therapy and biofeedback aimed at restoring symmetry and pre-empting synkinesis, and meta-analytic data support a benefit from such physical therapy in peripheral facial palsy [46].

The choice of antiviral agent is guided as much by pharmacokinetics and adherence as by comparative efficacy data, which in RHS specifically are sparse [20]. Valaciclovir and famciclovir achieve substantially higher and more reliable plasma concentrations than oral aciclovir with a far less burdensome dosing schedule, and are therefore preferred for oral therapy, while intravenous aciclovir is reserved for severe, disseminated or immunocompromised disease and for patients who cannot reliably absorb oral medication [41,48]. Whichever agent is chosen, the consistent message of the observational literature is that the benefit is front-loaded into the first 72 hours, so the operational priority is to start treatment promptly rather than to agonise over the choice between equivalent oral agents [7,8].

Corticosteroid therapy is given on the rationale of reducing the inflammatory oedema that compresses the nerve within its bony canal, mirroring its established role in Bell's palsy, even though disease-specific randomised evidence in RHS is lacking [21,22]. A typical course is prednisolone at approximately one milligram per kilogram per day for five to seven days followed by a taper, with the usual cautions regarding diabetes, hypertension and other steroid-sensitive comorbidities that are common in the older patients who predominate in this disease [13,23].

VII. Management — Antivirals, Steroids and Supportive Care

The governing principle is early combination antiviral and corticosteroid therapy, ideally started within 72 hours of onset, when the prospect of facial recovery is greatest [7]. First-line oral therapy is valaciclovir 1 g three times daily for seven to ten days, with famciclovir 500 mg three times daily as an alternative; aciclovir 800 mg five times daily is effective but limited by poor bioavailability, and intravenous aciclovir 10 mg/kg eight-hourly is reserved for severe disease, the immunocompromised, or patients unable to

tolerate oral therapy [8,20,41,48]. Corticosteroid is given as prednisolone approximately 1 mg/kg/day, capped near 60 to 80 mg, tapered over one to two weeks [21].

The evidence base deserves honest framing. The 2008 Cochrane reviews found insufficient randomised evidence to establish the efficacy of antivirals in RHS specifically, and no randomised trials of corticosteroids as an adjuvant in this disease, so practice is extrapolated from herpes zoster generally and from Bell's palsy [20,21,23]. Nonetheless the observational signal is consistent and clinically compelling: Murakami and colleagues showed that combination therapy begun within three days achieved complete recovery in around three-quarters of patients, and meta-analytic data favour the addition of antiviral to corticosteroid for facial recovery [7,8]. The American Academy of Neurology and Bell's palsy guidance inform the steroid component [13,23].

Supportive care is not ancillary. Exposure keratopathy from lagophthalmos is the most avoidable serious complication, and demands preservative-free artificial tears through the day, lubricating ointment and eyelid taping or a moisture chamber at night, and ophthalmology review for any corneal compromise [40]. Severe otalgia and subsequent neuropathic pain are managed with simple analgesia escalating to gabapentinoids, carbamazepine or low-dose tricyclics [25]. Vestibular suppressants are appropriate only for the acute days, as prolonged use retards central compensation; thereafter, structured vestibular rehabilitation with gaze-stabilisation and balance retraining is the mainstay, and facial neuromuscular retraining improves symmetry and limits synkinesis [46,47].

Table 5. Pharmacotherapy of Ramsay Hunt syndrome.

Agent	Dose	Duration	Notes
Valaciclovir	1 g PO three times daily	7–10 days	First-line oral; start within 72 h
Famciclovir	500 mg PO three times daily	7 days	Oral alternative
Aciclovir (oral)	800 mg PO five times daily	7–10 days	Lower bioavailability
Aciclovir (IV)	10 mg/kg every 8 h	7–10 days	Severe / immunocompromised / vomiting
Prednisolone	~1 mg/kg/day (max 60–80 mg)	Taper over 7–14 days	Adjuvant; extrapolated evidence

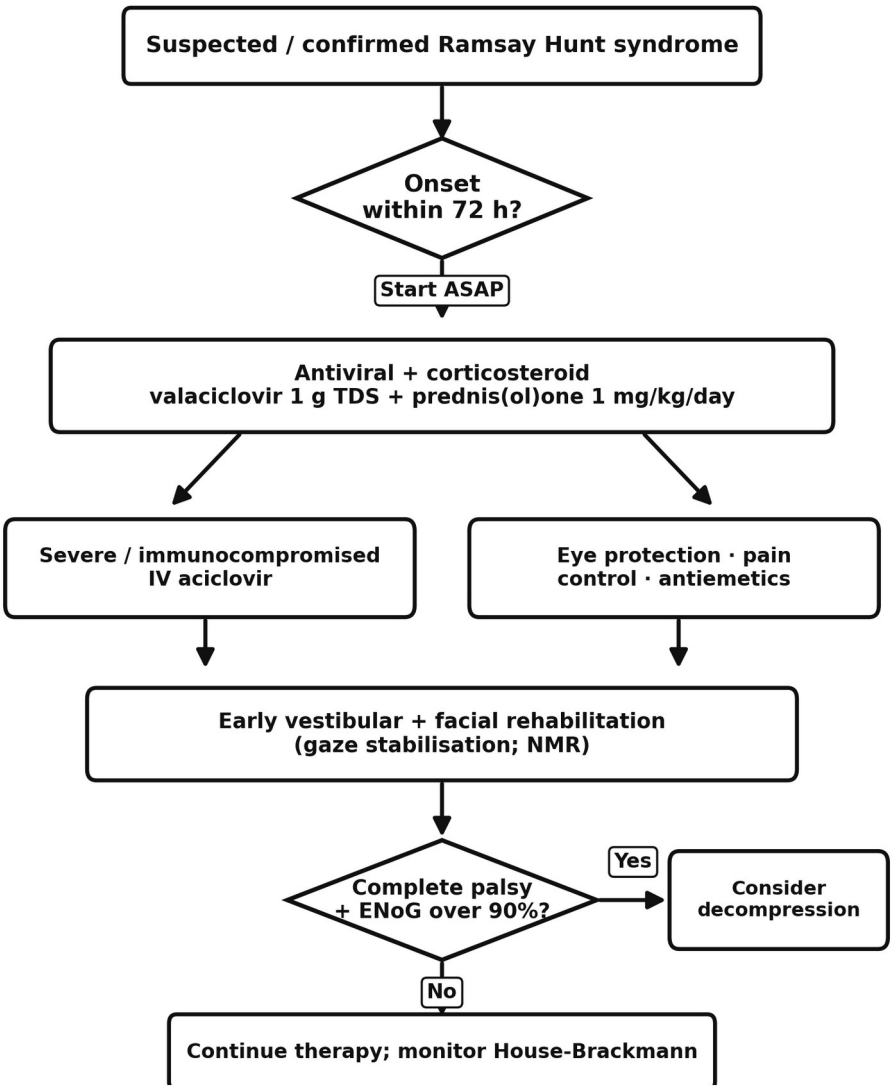


Figure 8. Management algorithm anchored on the 72-hour antiviral and corticosteroid window.
Source: Adapted from Murakami et al. [7] and the Cochrane reviews [20,21].

□ **Clinical Insight:** Treat empirically on clinical suspicion rather than waiting for virological confirmation — the 72-hour window is the strongest modifiable determinant of facial recovery, and the cost of treating a Bell's palsy as RHS is low.

Counselling about residual deficit

Patients value an honest account of the likely trajectory, and the vestibular physician is well placed to give it. Most facial recovery occurs over the first three to six months, but improvement can continue for up to a year, and synkinesis — most visibly oral-ocular twitching and gustatory lacrimation — tends to appear as a late marker of aberrant reinnervation rather than as a treatment failure [46]. Framing botulinum toxin and targeted retraining as planned management of expected reinnervation, rather than as rescue of a poor outcome, helps patients engage with a rehabilitation programme that may extend over many months [46].

The evidence around decompression illustrates the wider difficulty of generating high-quality data in a rare disease [37]. The reported series are small, retrospective and subject to selection, and although some describe favourable outcomes in neurophysiologically complete palsies operated within a defined early window, the absence of randomised comparison means the procedure cannot be recommended as a standard intervention and is best confined to carefully counselled patients in experienced centres

[24,37]. For the great majority, optimised medical therapy and structured rehabilitation remain the entire substance of management [46,47].

VIII. Refractory Disease, Surgery and Sequelae

Facial nerve decompression remains controversial and is offered in few centres. The rationale rests on the demonstrable intratemporal swelling of the nerve, and the procedure is generally restricted to complete paralysis (House-Brackmann V to VI) that fails to recover after at least two weeks of medical therapy and shows greater than 90 to 95 per cent degeneration on electroneuronography within the first one to two weeks [24,37]. No high-quality randomised data support it, and the operation carries risks of hearing loss, vertigo and further nerve injury, so it should be discussed only within a neuro-otology multidisciplinary setting [37].

Persisting deficits are managed expectantly and rehabilitatively. Synkinesis from aberrant regeneration — including oral-ocular synkinesis and gustatory lacrimation — typically emerges around three months and responds to neuromuscular retraining, mime therapy and targeted botulinum toxin [46]. Postherpetic neuralgia, the most troublesome sensory sequela, is managed with gabapentinoids and tricyclics and occasionally interventional nerve blocks [25]. Late reconstructive options for the incompletely recovered face include eyelid weight implantation, hypoglossal-facial or masseteric-facial transfer, and static slings, but these lie beyond the scope of acute vestibular management [31].

□ **Important:** Decompression is a last resort for neurophysiologically complete, non-recovering palsy and must never delay the time-critical medical therapy that determines most of the outcome.

The audiovestibular trajectory

The audiovestibular and facial limbs of the disease recover on different timescales and to different extents, a distinction worth making explicit when counselling [11]. Vertigo, driven by an acute unilateral vestibular deficit, typically settles over weeks as central compensation proceeds, and is the symptom most responsive to rehabilitation, whereas the sensorineural hearing loss is comparatively refractory, with complete recovery in only around one in ten patients and a tendency to persist at high frequencies [11]. Residual chronic imbalance, particularly in older patients with reduced compensatory reserve, is a recognised long-term outcome and an appropriate target for ongoing vestibular physiotherapy [47].

Prognostic stratification is increasingly quantitative. Beyond the clinical predictors of complete initial paralysis, advanced age and comorbidity, the degree of axonal degeneration measured by electroneuronography provides an objective marker, with dense degeneration in the first fortnight signalling a poor outlook, and emerging inflammatory indices such as the neutrophil-to-lymphocyte ratio have been explored as accessible adjuncts [6,9]. The presence of a cranial polyneuropathy is a clinically obvious and powerful adverse sign that should temper optimism at the first consultation [6].

Counselling on recurrence and prevention closes the loop with the patient. True recurrence of RHS is uncommon in the immunocompetent, but a single episode is a reminder of the broader lifetime risk of zoster, and recovered patients in the eligible age groups are reasonable candidates for the recombinant zoster vaccine once the acute illness has resolved [28,38]. In the immunocompromised, where both recurrence and severe disease are more likely, the case for vaccination and for vigilance is correspondingly stronger [41,42].

Among the special populations, pregnancy poses the most frequent practical questions. Facial palsy in pregnancy is more often Bell's palsy, but RHS must be considered when there is severe otalgia or a vesicular eruption, and management balances maternal benefit against fetal safety [13,22]. Corticosteroids, with their long record of use in pregnancy, remain first-line, and aciclovir and valaciclovir have a reassuring safety profile, so the combination can be offered with appropriate obstetric and infectious-diseases input rather than withheld [22]. Children, by contrast, are affected rarely but generally enjoy better facial recovery than adults, reflecting their greater regenerative capacity [43].

IX. Prognosis, Recurrence and Special Populations

RHS carries a worse facial prognosis than Bell's palsy. Untreated, only around one in five patients recovers completely; with early combination therapy complete recovery rises to roughly 70 to 75 per cent, and the timing of treatment is the dominant modifiable factor — approximately 75 per cent recover fully when treated within three days against around 30 per cent when treatment is delayed beyond a week [7,9]. In direct comparison, two-year recovery to House-Brackmann grade I is reached by about 58 per cent of RHS patients versus 86 per cent of those with Bell's palsy [14]. Adverse prognostic factors include complete paralysis at onset, advanced age, diabetes, a high degree of axonal loss on electroneuronography, and the presence of cranial polyneuropathy [6,9].

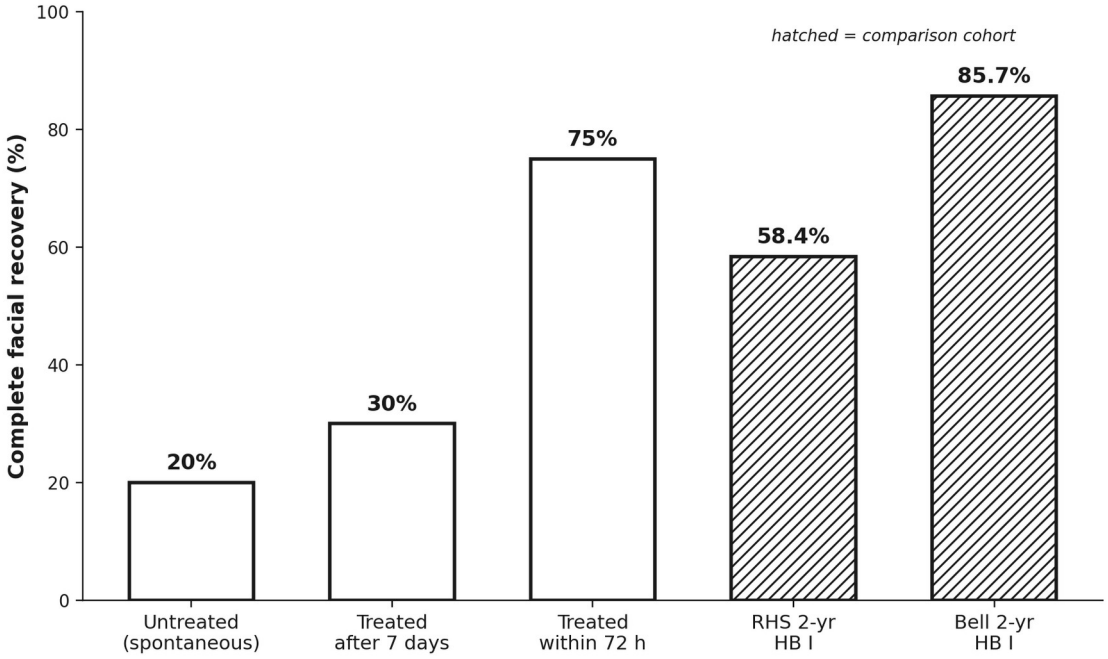


Figure 9. Complete facial recovery by treatment timing, and 2-year House-Brackmann grade I outcomes versus Bell's palsy.

Source: Data from Murakami et al. [7], Coulson et al. [9] and Zainine et al. [14].

Hearing recovery is notably poorer than facial recovery, with complete resolution of the sensorineural loss in only around one in ten patients, while vertigo usually settles over weeks through central compensation, assisted by rehabilitation [11]. Other complications include synkinesis, chronic ocular exposure, persistent dysgeusia and postherpetic neuralgia, the last reported across a wide range in the broader zoster population [25,39,42]. True recurrence of RHS is uncommon in the immunocompetent — under about five per cent — and is more likely in the immunosuppressed [38].

Table 6. Prognosis and recovery — key numbers for counselling.

Outcome	Figure
Complete recovery, treated within 72 h	~75%
Complete recovery, treated after 7 days	~30%
Complete recovery, untreated	~20%
2-year HB grade I (RHS vs Bell's)	58% vs 86%
Hearing recovery rate	~11%
Postherpetic neuralgia (zoster population)	2.6–46.7%
Recurrence (immunocompetent)	under 5%

Special populations modify management. The immunocompromised are at higher risk of severe, disseminated or polyneuropathic disease and of VZV vasculopathy, and generally warrant intravenous antiviral therapy, longer courses and closer surveillance [41,42]. In pregnancy, corticosteroids remain first-line and aciclovir and valaciclovir have a reassuring safety record, with management shared with obstetric and infectious-diseases colleagues [22]. Paediatric RHS is rare but recognised, and children

tend to recover better than adults [43]. The elderly carry the highest incidence, the poorest regenerative capacity and the greatest postherpetic neuralgia risk [4,39].

Implications for vestibular service design

The practical lessons of RHS for a vestibular service are about systems rather than science. Because outcome is so sensitive to the speed of treatment, the rate-limiting step is usually recognition at first contact, which argues for educating referrers and front-line clinicians to test for VZV and start antivirals in the painful or audiovestibular facial palsy rather than awaiting specialist review [2,12]. Equally, because the audiovestibular sequelae often outlast the facial palsy, a service that can offer timely audiometry, vestibular function testing and rehabilitation adds value well beyond the acute episode [11,47].

The contrast between the strength of the preventive evidence and the weakness of the treatment evidence is the defining tension in the field [20,21,26]. Prevention now rests on two large, high-quality randomised trials of the recombinant zoster vaccine, whereas acute management rests on extrapolation and observational data, an asymmetry that should shape both research priorities and the emphasis clinicians place on vaccination in their older patients [22,27]. Until disease-specific trials are feasible, the pragmatic standard remains early empirical combination therapy paired with proactive prevention [7,28].

X. Guidelines, Controversies and Future Directions

RHS sits in an awkward evidentiary position: a disease in which the standard of care — early combination antiviral and corticosteroid — rests largely on extrapolation rather than disease-specific randomised trials [20,21]. The open questions are whether the treatment window can be meaningfully extended beyond 72 hours, whether all acute facial palsy should be tested for VZV given the burden of zoster sine herpete, and what role, if any, decompression retains [12,23,37]. These uncertainties argue for adequately powered trials, which the rarity of the condition makes difficult.

The most consequential development is preventive. The adjuvanted recombinant zoster vaccine has shown efficacy exceeding 90 per cent against herpes zoster in adults over 50 and over 70 in the ZOE-50 and ZOE-70 trials, and is now recommended in preference to the live vaccine; widespread uptake is expected to reduce the incidence of zoster and, by extension, of RHS, although the vaccine is not absolutely protective and RHS is still reported in vaccinated individuals [26,27,28]. Future directions include better non-invasive virological diagnostics for zoster sine herpete, prognostic stratification using electrophysiology and inflammatory markers, neuroprotective adjuncts, and a clearer understanding of VZV vasculopathy as the unifying mechanism of its neurological complications [10,16,33]. For the vestibular physician, the enduring message is that RHS is a treatable, time-critical audiovestibular disease that is too often recognised late [2,3].

□ **Key Point:** Prevention now matters as much as treatment: the recombinant zoster vaccine substantially reduces zoster and therefore RHS, and post-recovery vaccination is reasonable to counsel in eligible older adults.

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