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CONFERENCE 2026

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Effects of whiplash on ENT conditions

Giving an opinion on tinnitus, dizziness and hearing symptoms after a road traffic accident, and knowing when to refer and when not to

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SESSION OVERVIEW

What we will cover in thirty minutes

01

ENT symptoms after whiplash

The injuries outside the fixed tariff, and why your report is often the only evidence

02

Categorising tinnitus

The 2026 AAO-HNS framework: subjective or objective, pulsatile, persistent, bothersome

03

Somatosensory tinnitus

What it is, why the neck drives the ear after an RTA, and a four-question screen

04

When to refer

Asymmetry, unilateral and pulsatile tinnitus, sudden hearing loss and the other red flags

05

When not to refer

Giving the opinion yourself: causation, McCombe grading, prognosis and treatment costs

06

Dizziness, hearing and acoustic shock

BPPV and cervicogenic dizziness, the airbag and the ear, the acoustic shock cluster

Declaration. Substantive NHS consultant at Manchester University NHS Foundation Trust; medicolegal practice through Hobson ENT Ltd, with instructions from claimants, defendants and jointly, including through Premex. No commercial interest in any device, drug or treatment named in this session.

SETTING THE SCENE

ENT injuries are not whiplash injuries

Section 1 of the Civil Liability Act 2018 defines a whiplash injury as a sprain, strain, tear, rupture or lesser damage of a muscle, tendon or ligament in the neck, back or shoulder.

Injury to the ear, the balance system or the nose is **not a whiplash injury**. Each is a separate head of claim, valued outside the fixed tariff, and in the Official Injury Claim portal the medical report is usually the only evidence that it exists.

The expert is asked to say whether the symptom is present, whether it is attributable to the accident, how severe it is and how it is likely to behave. For most claimants that opinion does not need an ENT surgeon.

Tinnitus

Ringing, buzzing or hissing; the commonest ENT complaint after whiplash

Dizziness and imbalance

Positional vertigo, neck-related unsteadiness, labyrinthine concussion

Hearing loss

Airbag acoustic trauma, head impact, or pre-existing loss newly noticed

Aural fullness and pain

Eustachian tube dysfunction, TMJ strain, acoustic shock

Hyperacusis

Decreased sound tolerance after a sudden loud sound

Smell, taste and nasal injury

Olfactory loss after head impact; nasal fracture and septal deviation

THE EVIDENCE BASE

Whiplash is a recognised risk factor for tinnitus

10 to 15%

of people with a history of whiplash develop persistent tinnitus

Claussen and Constantinescu 1995

57%

of more than 400 German whiplash patients complained of tinnitus

Chetana and Claussen 2010

About 7%

of patients at a specialist tinnitus clinic attributed onset to head or neck injury

Folmer and Griest 2003

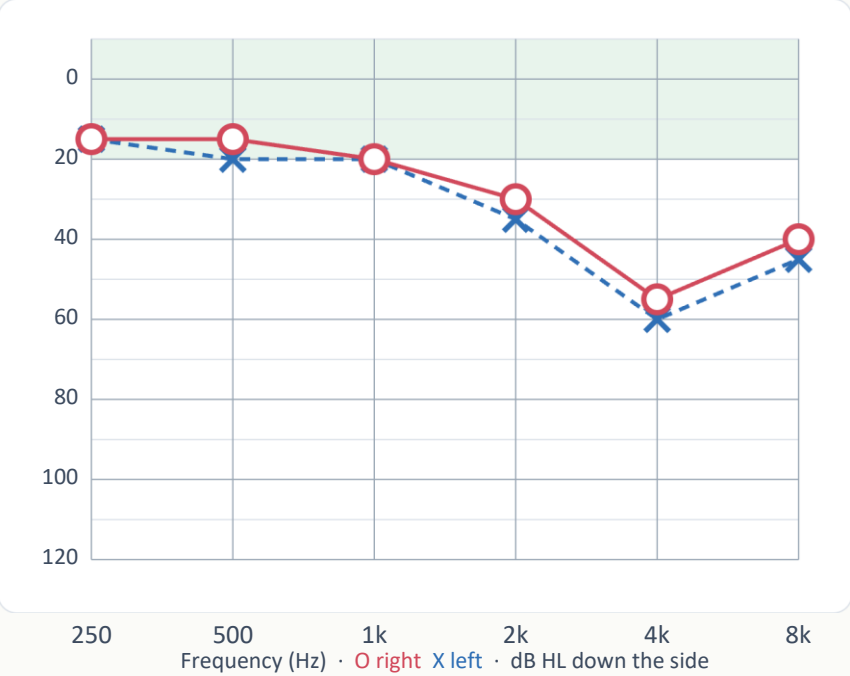
There is a significant association

A systematic review of non-otological risk factors found a significant association between whiplash and tinnitus (Deklerck 2020). The Blue Mountains Hearing Study of more than 3,600 adults confirmed whiplash as a risk factor (Sindhusake 2003).

But the background rate is high

Tinnitus affects 10 to 15% of adults, and 1 to 2% find it severely bothersome (Adams 2026). Always ask about tinnitus before the accident and look for it in the GP record; a pre-existing symptom made worse is a different opinion from a new one.

Hearing loss and tinnitus: the strongest association



A symmetrical 4 kHz notch: the signature of noise exposure, and a common incidental finding in claimants with an industrial or military history.

Hearing loss is the strongest and best-evidenced risk factor for tinnitus. In the Biswas meta-analysis the risk roughly doubled with any hearing loss (RR 1.94) and more than tripled with sensorineural loss (RR 3.68); occupational noise carried a 70% excess. Most people with tinnitus have some measurable loss.

The mechanism is deafferentation: when the cochlea sends less to the brain, central auditory gain rises and spontaneous activity is heard as sound. That is why the audiogram is the first investigation, and why an existing loss is the first alternative cause to exclude in an RTA claim.



Hearing aids: recommended where hearing difficulty and bothersome tinnitus coexist (KAS 8)



Noise: a pneumatic drill runs at about 100 to 110 dB; ask about work, service and loud leisure

THE EVIDENCE BASE

Biswas for the defence!

What Biswas et al. (2023) found

A systematic review of tinnitus risk factors that admitted only cohort and case-control studies reporting risk ratios: 25 of 374 articles. Pooled estimates: **whiplash RR 1.40 (95% CI 0.95 to 2.07)** and head injury RR 1.21 (0.97 to 1.52), each from no more than four studies with high heterogeneity. The authors list whiplash among exposures with “no association”, and defendants now cite it to argue the link is unproven.

Absence of evidence, not evidence of absence

The point estimate favours an association. A handful of heterogeneous studies cannot exclude a 40% increase; the authors themselves say the pooled estimates come from two or three sources and that most associations “remain inconclusive”.

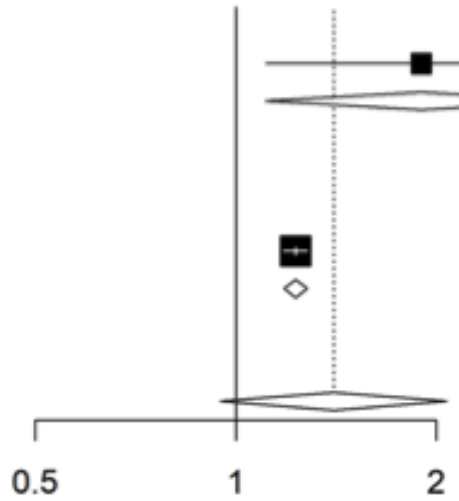
1.40

pooled relative risk of tinnitus after whiplash: a 40% excess, with a confidence interval that only just crosses 1 (p = 0.11)

Biswas R et al. J Assoc Res Otolaryngol 2023;24:81-94

Supplementary Figure 24. Traumatic head and neck injury – whiplash

Study Design	Author, year	Cases Whiplash	No history	RR [95% CI]
	AnyT			
Cohort	Gopinath et al., 2010b			1.90 [1.11; 3.26]
	Pooled estimate			**1.90 [1.11; 3.26]**
	Heterogeneity: not applicable			
	MedT			
Case-Control	Stohler et al., 2019	4948	104835	1.23 [1.18; 1.28]
	Pooled estimate	**4948**	**104835**	**1.23 [1.18; 1.28]**
	Heterogeneity: not applicable			
	Pooled estimate	**4948**	**104835**	**1.40 [0.95; 2.07]**
	Heterogeneity: I^2 = 60%, p = 0.11			



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It excluded the studies that show the link

Only cohort and case-control designs were admitted. The whiplash literature (Claussen, Folmer and Griest, the Blue Mountains survey, the Deklerck review) is largely cross-sectional and clinic-based, so it never entered the pool.

It confirmed the pathway

The same review found temporomandibular disorder a reliable risk factor (RR 2.06). TMJ and cervical dysfunction are the drivers of somatosensory tinnitus, which the 2026 guideline now defines with “tinnitus preceded by head or neck trauma” as a criterion.

Population risk is not individual causation

A risk factor study asks whether whiplash raises tinnitus rates across a population. The court asks whether this claimant’s tinnitus, with this onset, this neck and this record, was caused by this accident. The second question is answered clinically.

CATEGORISING TINNITUS

The 2026 AAO-HNS Clinical Practice Guidelines

Subjective or objective

Subjective: heard only by the patient (nearly all cases). Objective: generated by blood flow or muscle contraction and sometimes audible to the examiner.

Non-pulsatile or pulsatile

Rhythmic tinnitus. Pulse-synchronous suggests a vascular cause; asynchronous clicking suggests palatal or middle ear myoclonus. Both need investigation.

Recent onset or persistent

Recent onset: under 6 months. Persistent: 6 months or longer (some use 3). Spontaneous improvement is common early, which matters for prognosis.

Bothersome or non-bothersome

Bothersome: causes distress or affects function and the patient wants treatment. Non-bothersome: noticed but not intrusive. This split drives severity grading.

Primary or secondary

Primary: idiopathic, with or without sensorineural hearing loss. Secondary: an identifiable cause such as otosclerosis, Ménière's disease or a vestibular schwannoma.

Somatosensory subtype

Subjective tinnitus modulated by head, neck or jaw movement, or coexisting with neck pain or TMJ dysfunction. The usual pattern after whiplash; the next four slides.

Why the category matters. It decides whether you can give the opinion yourself, whether audiometry is enough, or whether ENT assessment and imaging are needed before causation and prognosis can be stated safely.

SOMATOSENSORY TINNITUS

What it is

A subtype of subjective tinnitus influenced by input from the musculoskeletal system, particularly the **cervical spine**, the **temporomandibular joint** and the **cranial musculature**.

Not a separate disease but a recognisable clinical subtype, defined by Delphi consensus (Michiels 2018) and adopted as a new key action statement in the 2026 AAO-HNS guideline: clinicians may inquire about somatosensory features in persistent, bothersome tinnitus.

69%

of tinnitus patients report some somatic modulation across studies

30 to 64%

of patients with temporomandibular disorder have tinnitus

DIAGNOSTIC CRITERIA (MICHIELS 2018)

Modulation

- Changes with voluntary head, neck, jaw or eye movement
- Changes with muscle contraction or somatic manoeuvres
- Changes with pressure on myofascial trigger points

Characteristics

- Tinnitus and neck or jaw pain began together and flare together
- Preceded by head or neck trauma
- Worse with poor posture; pitch, loudness or side vary
- Unilateral tinnitus not explained by the audiogram

Accompanying features

- Frequent neck, head or shoulder girdle pain
- Tender trigger points; suboccipital and cervical extensor tension
- TMJ disorder, clenching or bruxism, dental disease

SOMATOSENSORY TINNITUS

Why it occurs after a road traffic accident

1

Whiplash

Rapid flexion and extension strains cervical soft tissue; TMJ and jaw muscles are often strained too, and clenching follows

2

Pain and guarding

Protective muscle tension, trigger points, altered posture and restricted cervical movement persist for weeks to months

3

Altered somatic input

Upper cervical (C2) and trigeminal afferents carry increased and abnormal signals from the injured neck and jaw

4

Dorsal cochlear nucleus

The first auditory relay in the brainstem, where somatosensory and auditory inputs converge and are integrated

5

Cross-modal plasticity

Somatosensory input gains influence over auditory neurons; spontaneous firing and synchrony rise and are heard as sound

Why onset is often delayed

The tinnitus is a product of the neck dysfunction, not of the impact itself, so it typically appears days to a few weeks after the accident as guarding and posture change set in. Delayed onset within that window supports causation.

Why the audiogram is usually normal

The cochlea is not the generator. Hearing is often normal for age or shows pre-existing presbyacusis. A normal audiogram with a positive somatosensory screen is the expected picture and does not undermine the diagnosis.

Why it tracks the neck

Loudness rises and falls with neck pain, posture and fatigue, and improves as cervical function recovers. That is why treatment is aimed at the neck and jaw rather than at the ear.

Four screening questions (AAO-HNS 2026) and what to examine

Somatic modulation

"Does your tinnitus change when you move your jaw or neck, for example clenching, opening wide or turning your head?"

A yes suggests: Strong indicator of somatosensory modulation

Pressure modulation

"Does pressing on sore spots in your jaw or neck muscles change the sound?"

A yes suggests: Supports a myofascial or cervical contribution

TMJ and neck symptoms

"Do you have jaw problems (pain, clicking, locking) or frequent neck or shoulder pain?"

A yes suggests: Common coexisting musculoskeletal drivers

Temporal association

"Did your tinnitus start around the same time as the jaw or neck pain, or after the injury?"

A yes suggests: Temporal linkage consistent with somatosensory origin

In the examination

- Cervical range of movement and pain on movement
- Tenderness of paraspinal and suboccipital muscles; trigger points
- TMJ tenderness, clicking or restricted opening; wear facets from clenching
- Ask the claimant to clench, open wide and turn the head: does the tinnitus change?
- Otoscopy: expect a normal drum; note any abnormality
- Tuning forks or a screening audiogram if available

Record the answers in the report

A positive screen supports causation, because it ties the tinnitus to the injured neck rather than to the ear or to chance, and it directs treatment towards cervical physiotherapy rather than hearing aids. A negative screen with normal hearing still permits the opinion; it removes one strand of support.

Management and prognosis

Cervical physiotherapy

Eight randomised trials of multimodal cervical physiotherapy (manual therapy, posture, exercise) show consistent reductions in tinnitus handicap scores. A course is reasonable to recommend; its cost is similar to tinnitus therapy, so obtain a quote.

TMJ-directed treatment

A meta-analysis of four trials of bite splints, jaw exercises and occlusal adjustment in TMD-related tinnitus found reduced tinnitus severity alongside reduced jaw pain. Dental or maxillofacial referral where the jaw dominates.

Standard tinnitus care still applies

Education and counselling, sound enrichment, cognitive behavioural therapy (recommended), and a hearing aid evaluation where there is hearing loss. Medicines and supplements are not recommended for tinnitus itself.

Prognosis follows the neck

Most somatosensory tinnitus improves as cervical pain and guarding resolve, usually within twelve to eighteen months, and the majority habituate. Persistent neck dysfunction, a high initial grade and marked distress predict a longer course. No single therapy is universally effective, so treatment is individualised.

Suggested report wording

"The tinnitus is modulated by neck movement and began with the neck pain. On the balance of probabilities it is somatosensory tinnitus arising from the cervical injury. I recommend a course of cervical physiotherapy, for which a quote should be obtained."

WHEN TO REFER

The red flags: refer before you grade and prognose

Feature	Concern	Action
Unilateral tinnitus	Focal auditory pathway lesion, e.g. vestibular schwannoma	Audiometry and ENT assessment; MRI when indicated
Asymmetric or unilateral hearing loss	Vestibular schwannoma, Ménière's disease, otological disease	Audiometry and ENT assessment; MRI when indicated
Pulsatile tinnitus	Vascular lesion or tumour, dissection, intracranial hypertension	ENT referral and vascular imaging (next slide)
Sudden hearing loss	Sudden sensorineural loss: a treatment window of days	Same-day ENT or emergency department; do not wait
Vertigo or imbalance	Labyrinthine, retrocochlear or central cause	Vestibular assessment; urgent if central features
New neurological symptoms	Central pathology: vascular, demyelinating, degenerative	Neurology; emergent if sudden onset
Abnormal otoscopy	Perforation, haemotympanum, retrotympanic mass, ossicular injury	ENT referral; CT temporal bone if fracture suspected
Distress or thoughts of self-harm	Psychological morbidity is common and under-recognised	Screen (PHQ-9 item 9); mental health referral; urgent if at risk

WHEN TO REFER

Pulsatile tinnitus

Pulse-synchronous

Beats with the heart: usually vascular. Carotid or vertebral artery stenosis or dissection, dural arteriovenous fistula, glomus tumour, sigmoid sinus diverticulum, venous hum, idiopathic intracranial hypertension. After an RTA think specifically of dissection and of temporal bone injury.

Pulse-asynchronous

Clicking or fluttering out of time with the pulse: palatal or middle ear myoclonus, patulous Eustachian tube, or the tensor tympani in acoustic shock. Usually benign but still deserves assessment because it is easily confused with the vascular group.

What you can do in the room

- Ask: in time with the pulse, or a click? One ear or both? Changed by neck pressure or turning?
- Auscultate the neck, mastoid and orbit for a bruit
- Otoscopy for a red or blue retrotympanic mass
- Blood pressure; fundoscopy for papilloedema if headache or visual symptoms

Imaging (ACR appropriateness criteria 2023)

- Pulsatile, normal drum: CT angiography of head and neck, or MRI with MR angiography and venography
- Pulsatile with retrotympanic lesion: CT temporal bone
- Not for the generalist to order in a medicolegal setting: refer and say why

Always refer pulsatile tinnitus. Do not attribute it to the accident, grade it or give a prognosis until the cause is established.

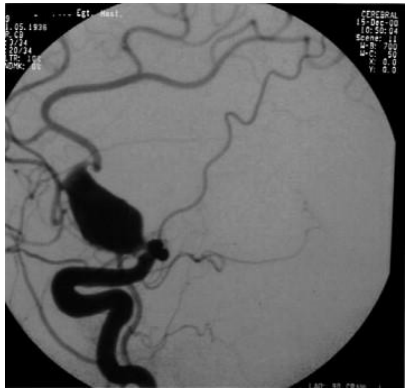
WHEN TO REFER

Pulsatile tinnitus: what may be behind it



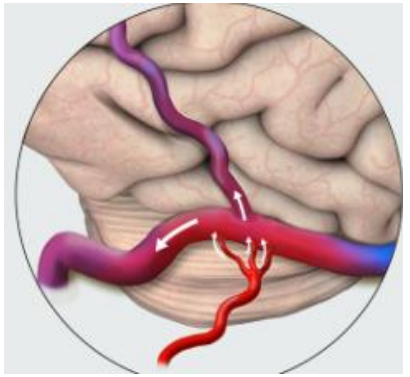
Glomus tympanicum

Otoscopic view: a red vascular mass behind the lower drum, the “rising sun” sign. Pulse-synchronous tinnitus, sometimes with conductive loss. The one cause otoscopy can find in the room; CT temporal bone next.



Aneurysm or dissection

Turbulent flow through a dilated or injured artery is heard as a pulse-synchronous hum. Carotid or vertebral dissection is the post-RTA concern, especially with neck pain, headache or a Horner’s syndrome. CT or MR angiography.



Dural arteriovenous fistula

A direct connection between a dural artery and a venous sinus, often the sigmoid or transverse sinus behind the ear. A bruit over the mastoid is characteristic. Angiographic imaging and neuro-interventional referral.

Schematic illustrations. Other vascular causes include sigmoid sinus diverticulum or dehiscence, aberrant carotid artery, high jugular bulb, venous hum and idiopathic intracranial hypertension. None of these is an accident injury and none can be graded or prognosed until identified.

WHEN TO REFER

Unilateral tinnitus and asymmetric hearing loss

Every tinnitus claimant should have an audiogram

The 2026 guideline recommends a comprehensive audiological evaluation for every patient with tinnitus, ideally within four to six weeks, whether or not they notice hearing difficulty. It defines the hearing loss, separates conductive from sensorineural, and is the only objective way to detect asymmetry. If none exists, recommend one in the report and say what it would decide.

What counts as asymmetric

Commonly used UK referral thresholds: a difference between the ears of **15 dB or more at two adjacent frequencies**, or **20 dB or more at a single frequency**, between 0.5 and 8 kHz. Unilateral tinnitus with symmetrical hearing has a low yield for schwannoma, but still needs ENT review. Check the records: asymmetry documented before the accident changes the causation question entirely.

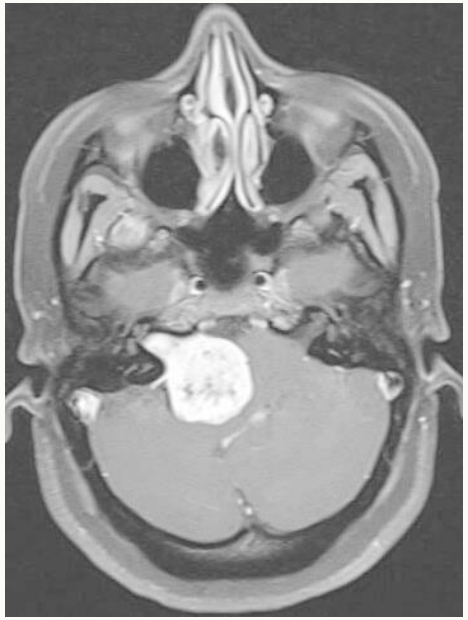
Imaging rule (AAO-HNS 2026, KAS 6, strong recommendation)

Do not obtain neuroradiological imaging to evaluate tinnitus unless there is one or more of: tinnitus that localises to one ear, pulsatile tinnitus, focal neurological abnormality, or asymmetric hearing loss. Bilateral non-pulsatile tinnitus with symmetrical hearing and no neurology needs no scan, and the report can say so.

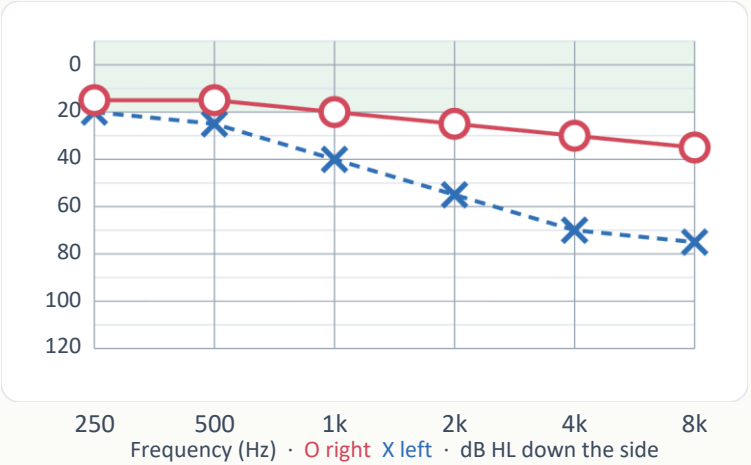
If referral is needed

Unilateral non-pulsatile tinnitus or asymmetry: MRI of the internal auditory meati. State in the report that causation and prognosis are provisional until the scan is reported, and offer an addendum.

Vestibular schwannoma: the diagnosis behind the asymmetry rule



Schematic axial view. A benign tumour of the vestibular nerve sheath grows slowly within the internal auditory canal and may extend into the cerebellopontine angle to press on the brainstem.



How it presents, and why it matters to the claim

Unilateral tinnitus, an asymmetric high-frequency sensorineural loss, sometimes imbalance; growth is slow. MRI of the internal auditory meati is the test, with a yield of a few per cent in asymmetric loss. An incidental schwannoma explains the tinnitus and defeats causation, so unilateral symptoms are imaged before the opinion is final. A symmetrical audiogram with bilateral tinnitus makes it very unlikely.

WHEN NOT TO REFER

A four-column decision for the RTA report

Give the opinion yourself

- Bilateral or symmetrical, non-pulsatile tinnitus
- Normal otoscopy
- No vertigo, no neurological symptoms
- No sudden hearing loss
- Onset within days to a few weeks of the accident
- Then: causation, somatosensory screen, McCombe grade, prognosis, treatment and costs

Recommend audiometry first

- Any hearing complaint, aural fullness or muffled hearing
- Airbag deployment or head impact
- Symptoms persisting beyond six months
- No audiogram in the records
- State what the result would change in your opinion

Recommend ENT referral

- Unilateral tinnitus
- Asymmetric hearing loss on audiometry
- Persistent bothersome tinnitus not improving at six to twelve months
- Vertigo beyond six to eight weeks, or positional vertigo not settling
- Hyperacusis or acoustic shock features
- Loss of smell; nasal obstruction after nasal injury

Refer urgently, do not wait

- Sudden hearing loss (same day)
- Pulsatile tinnitus, especially with headache or neurology
- New neurological deficit or facial weakness
- Clear fluid from nose or ear after head injury
- Thoughts of self-harm
- Tell the claimant and the GP in writing

Dizziness after whiplash

BPPV

The commonest vertigo after head or neck trauma: brief spinning on turning in bed or looking up. Dix-Hallpike confirms; repositioning (Epley) cures most. Good prognosis, recurrence possible.

Cervicogenic dizziness

Unsteadiness and light-headedness rather than spinning, linked to neck pain and movement, worse with fatigue. A diagnosis of exclusion that sits with the whiplash itself. Physiotherapy.

Labyrinthine concussion

Direct head impact: vertigo with hearing loss and tinnitus from inner ear injury. Needs audiometry and ENT assessment; CT if fracture suspected.

Vestibular migraine and PPPD

Migraine is often unmasked by trauma: episodic vertigo with headache or light sensitivity. PPPD: constant swaying unsteadiness for three months or more, worse upright, moving or in busy visual surroundings.

Refer when

Positional vertigo not settling with repositioning; vertigo with one-sided hearing loss or tinnitus; dizziness beyond six to eight weeks; falls. **Urgently** for central features (double vision, slurred speech, ataxia, new severe headache) or vertigo with neck pain soon after the accident, which raises vertebral artery dissection.

In the report

Describe the character (spinning or unsteadiness), triggers, duration and course, and record a Dix-Hallpike if performed. Most post-traumatic dizziness is BPPV or cervicogenic and settles within months; give the prognosis as a percentage and recommend vestibular physiotherapy where appropriate.

Hearing loss and the airbag

Airbag deployment

Airbag inflation produces an impulse noise with peak levels reported at around 160 to 170 dB in an enclosed cabin. A temporary threshold shift with tinnitus for hours to days is common; permanent acoustic trauma is less common but well documented, typically a high-frequency notch. Ask whether the airbag deployed.

Head impact

Labyrinthine concussion, temporal bone fracture, ossicular disruption (a conductive loss with a normal or bloodstained drum) and haemotympanum. Any hearing loss after head impact needs audiometry; a conductive loss or abnormal drum needs ENT assessment.

Pre-existing loss newly noticed

Presbyacusis and noise-induced loss are common in the claimant population. The audiogram pattern (symmetrical high-frequency slope, or a 4 kHz notch) usually tells the story and the records often confirm it. Attribute only what the accident changed.

Sudden sensorineural hearing loss is an emergency

A loss of 30 dB or more over three contiguous frequencies developing within 72 hours. Oral or intratympanic steroids are most effective in the first two weeks and of little value after a month. If a claimant describes a sudden drop in hearing in one ear at any point, including at the time you see them, advise same-day ENT or emergency department attendance and record that you did.

Acoustic shock: a distinct entity

Acoustic shock is a response to an unexpected loud sound perceived as highly threatening. Classically a tone through a telephone headset, but the same mechanism applies to an airbag report or a sudden impact noise in the cabin.

The proposed mechanism is **tonic tensor tympani syndrome**: an involuntary, anxiety-driven lowering of the tensor tympani reflex threshold, producing recurrent middle ear muscle spasm. Otoscopy, tympanometry and the audiogram are typically normal.

Tinnitus	**Aural pain or blockage**
Hyperacusis or phonophobia	**Fluttering, muffled hearing**
Numbness or burning round the ear	**Anxiety, trauma, low mood**

	Whiplash (somatosensory) tinnitus	Acoustic shock
Trigger	Mechanical neck and jaw injury	A sudden, unexpected, threatening loud sound
Hallmark	Tinnitus tracks neck pain; onset over days to weeks	Hyperacusis dominant; aural pain and flutter; immediate
Hearing	Normal, or pre-existing loss	Normal otoscopy, tympanometry and audiogram
Driver	Altered somatosensory input to the auditory pathway	Tonic tensor tympani syndrome; anxiety-driven
Refer?	Not for the typical bilateral symmetrical case	Yes: tinnitus and hyperacusis clinic for sound tolerance testing

THE OPINION

Grading tinnitus severity

Slight

Heard only in quiet surroundings; no interference with sleep or daily activities

Mild

Easily masked by environmental sound; readily forgotten during activity

Moderate

Noticed even with background noise; intrudes on sleep and quiet activities

Severe

Almost always heard; sleep disturbed; interferes with daily activity; medical consultation expected in the records

Catastrophic

All severe features and worse; documented consultation; psychological sequelae likely

Anchor the grade to evidence

The McCombe grading (Clin Otolaryngol 2001) is the accepted scale in UK medicolegal practice. Severe and catastrophic grades should be supported by documented medical consultation, so obtain the GP records before finalising. A claimant who first mentions tinnitus at the medicolegal examination is rarely more than mild to moderate.

Bothersome or not, in the guideline's words

The 2026 guideline splits bothersome from non-bothersome tinnitus and recommends a validated questionnaire (Tinnitus Handicap Inventory or Tinnitus Functional Index) to measure impact. Quoting a score alongside the McCombe grade strengthens the report and makes the severity reproducible for the other side.

THE OPINION

Causation and prognosis

Temporal coherence

Tinnitus emerging within days to a few weeks of the accident, in the setting of neck pain, supports causation on the balance of probabilities. Months of silence followed by a new complaint does not.

Documentary support

Look for the complaint recorded with the GP or physiotherapist before any medicolegal contact, particularly at higher grades. Record the date of first complaint and the interval from the accident.

Exclude alternatives

Pre-accident tinnitus, noise exposure, ototoxic drugs, otological disease and the red flags. The audiogram and the records do this work; the background prevalence of 10 to 15% is why it matters.

Frame future risk as a percentage

Most accident-related tinnitus improves. Symptoms commonly settle within twelve to eighteen months and the majority habituate, like ceasing to notice a dripping tap. Future risk is not decided on the balance of probabilities: state a best-estimate chance of recovery, for example a 75% chance of becoming largely asymptomatic within two years with treatment, leaving the corollary chance for the court to value. This is how the court assesses loss and it is correct in law.

Prognostic markers

- Mild tinnitus resolves about twice as often as severe (40% against 20%)
- Of persistent tinnitus, 18% resolved over four to five years in two large cohorts
- Neck dysfunction, hearing loss and distress predict a longer course

THE OPINION

Treatment and indicative costs

1 **Education, counselling and sound enrichment**

First line for everyone; recommended by the guideline. Sound enrichment is an option, not a cure.

2 **Treat the cause**

Cervical physiotherapy for somatosensory tinnitus; TMJ treatment where the jaw dominates; hearing aids where there is hearing loss.

3 **Cognitive behavioural therapy and tinnitus therapy**

CBT is recommended (KAS 11). Tinnitus retraining or therapy: usually four to six sessions, about £250 per session (NICE NG155).

4 **Masking devices and bimodal neuromodulation**

Sound generators if symptoms persist; bimodal stimulation (Lenire) is an option in the 2026 guideline, about £3,600.

Not **Medicines, supplements and rTMS**

Antidepressants, anticonvulsants, anxiolytics, supplements and repetitive transcranial magnetic stimulation are recommended against for tinnitus itself.

INDICATIVE PRIVATE COSTS

Item	Cost
Hearing and tinnitus assessment	£275
Tinnitus therapy, 4 to 6 sessions at £250	£1,500
Cervical physiotherapy course	similar; quote
Pair of masking devices (bilateral)	£4,500
Five-year consumables	£300
Bimodal neuromodulation device, if indicated	£3,600

Devices need renewal after about five years. Costs are dynamic: recommend a current quote. Treatment is supportive, not curative; an interim payment may be sought to fund it.

THE OPINION

Treatment and indicative costs


TINNITUS UK

Home » Tinnitus treatments » Lenire®

Lenire®

Here we look at the claims for Lenire®.

Version: 2.1
Last updated: November 2025
To be reviewed: November 2028

Sound therapy via headphones
Electrical stimuli to tongue

Changes brain plasticity

- **95%** of treatment-compliant participants reported an improvement in Tinnitus Handicap Inventory (THI) scores after 12 weeks. [3,4]
- **84%** reported improvement in Tinnitus Functional Index (TFI) scores. [4]
- Large trials (TENT-A1, TENT-A2) reported clinically meaningful average reductions in Tinnitus Handicap Inventory (THI) and Tinnitus Functional Index (TFI) during treatment. Benefits persisted for some participants at follow-up.[4]

4 **Masking devices and bimodal neuromodulation**

Sound generators if symptoms persist; bimodal stimulation (Lenire) is an option in the 2026 guideline, about £3,600.



THE OPINION

Treatment and indicative costs

1 **Education, counselling and sound enrichment**

First line for everyone; recommended by the guideline. Sound enrichment is an option, not a cure.

2 **Treat the cause**

Cervical physiotherapy for somatosensory tinnitus; TMJ treatment where the jaw dominates; hearing aids where there is hearing loss.

3 **Cognitive behavioural therapy and tinnitus therapy**

CBT is recommended (KAS 11). Tinnitus retraining or therapy: usually four to six sessions, about £250 per session (NICE NG155).

4 **Masking devices and bimodal neuromodulation**

Sound generators if symptoms persist; bimodal stimulation (Lenire) is an option in the 2026 guideline, about £3,600.

Not **Medicines, supplements and rTMS**

Antidepressants, anticonvulsants, anxiolytics, supplements and repetitive transcranial magnetic stimulation are recommended against for tinnitus itself.

INDICATIVE PRIVATE COSTS

Item	Cost
Hearing and tinnitus assessment	£275
Tinnitus therapy, 4 to 6 sessions at £250	£1,500
Cervical physiotherapy course	similar; quote
Pair of masking devices (bilateral)	£4,500
Five-year consumables	£300
Bimodal neuromodulation device, if indicated	£3,600

Devices need renewal after about five years. Costs are dynamic: recommend a current quote. Treatment is supportive, not curative; an interim payment may be sought to fund it.

A WORKED EXAMPLE

Case study: post-RTA tinnitus, end to end

The instruction (anonymised, illustrative)

Male, 42, rear-ended at a junction; airbag did not deploy. Neck pain from day one. Three weeks later, constant high-pitched ringing in both ears, louder when turning the head or clenching. No prior ear history; no noise exposure. Instructed to address causation, severity, prognosis and treatment.

Findings

Normal otoscopy. Symmetrical audiogram with a mild high-frequency tail-off from 4 kHz, consistent with age. Positive somatosensory screen: modulated by jaw clench and neck rotation; tender suboccipital trigger points.

Referral decision

Bilateral, symmetrical, non-pulsatile; no vertigo, neurology or sudden loss. Column one: no ENT referral and no imaging indicated (KAS 6). Audiogram already obtained.

Causation and grade

Onset within weeks, tracking neck pain, no alternative cause: on balance, somatosensory tinnitus caused by the accident. Intrudes on sleep and quiet, masked by ambient noise, GP record at week five: moderate.

Prognosis and treatment

Already improving with the neck. About 75% chance of becoming largely asymptomatic within two years. Recommend cervical physiotherapy and tinnitus therapy; masking devices if symptoms persist at twelve months.

Change one fact. The same claimant, but the ringing is in the left ear only and the audiogram shows a 25 dB difference at 2 and 4 kHz: this moves to column three. Recommend ENT referral and MRI of the internal auditory meati, give causation and prognosis as provisional, and offer an addendum.

KEY TAKEAWAYS

Five things to carry into the next report

HOBSON ENT

1. **ENT injuries sit outside the whiplash tariff**
   Tinnitus, dizziness and hearing loss are separate heads of injury; your report is usually the only evidence.

2. **Most post-RTA tinnitus is somatosensory**
   Four screening questions, examine the neck and jaw, record the answers: they support causation and direct treatment.

3. **Refer on the red flags, not on the diagnosis**
   Unilateral tinnitus, asymmetry, pulsatile tinnitus, sudden loss, central vertigo, neurology, abnormal drum, self-harm risk.

4. **Otherwise give the opinion yourself**
   Bilateral symmetrical non-pulsatile tinnitus with a normal drum needs no scan and no ENT surgeon.

5. **Get the audiogram and the GP records**
   They settle asymmetry, pre-existing loss, the date of first complaint and the evidence for any grade above mild.

Mr Jonathan Hobson | Consultant ENT and Head & Neck Surgeon | Hobson ENT Ltd | Questions welcome

EVIDENCE

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