The Ears – Part 3: When Sound & Balance Misfire

Exploring the complex world of auditory distortion and vestibular dysfunction

- PART 1 OF 2 -

Session Focus

An in-depth look at:

- **Tinnitus and other auditory perception disturbances**
- **Vestibular dysfunction and balance disorders**
- How inner ear and brain signaling can misfire
- Why symptoms persist even when imaging is “normal”

I. When Sound Misfires:

Tinnitus & Auditory Distortions

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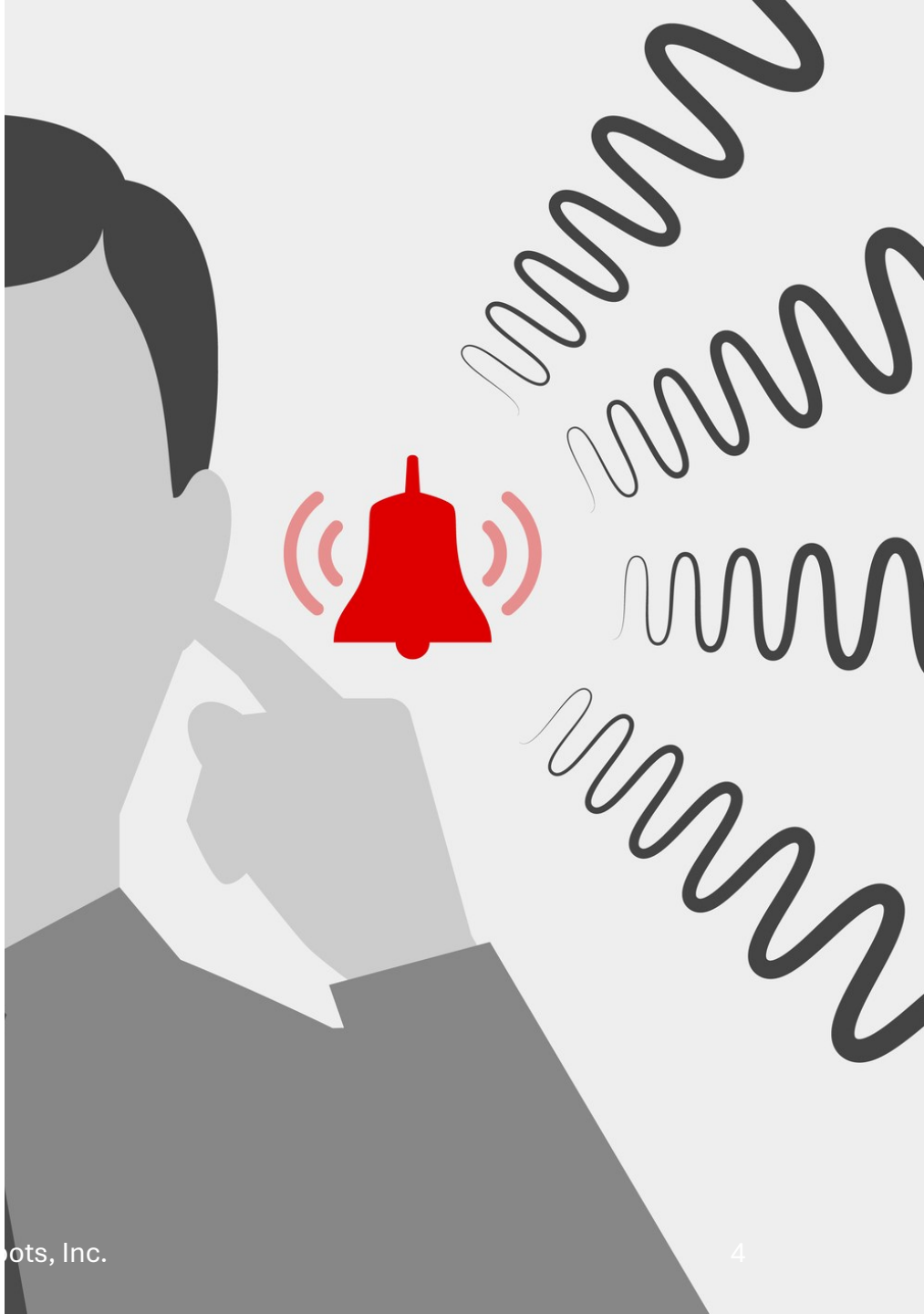
1. What Is Tinnitus?

- Perception of sound without external stimulus
- Ringing, buzzing, hissing, roaring, clicking, pulsatile
- Unilateral or bilateral
- Intermittent or constant

Distinction:

Tinnitus is a symptom, not a specific disease



2. Peripheral vs. Central Origins

Peripheral (Cochlear-Driven)

- Hair cell damage
- Noise-induced injury
- High-frequency hearing loss
- Ototoxic exposure

Central (Neural Gain)

- Brain compensates for reduced input
- Increased auditory cortex activity
- Maladaptive signal amplification

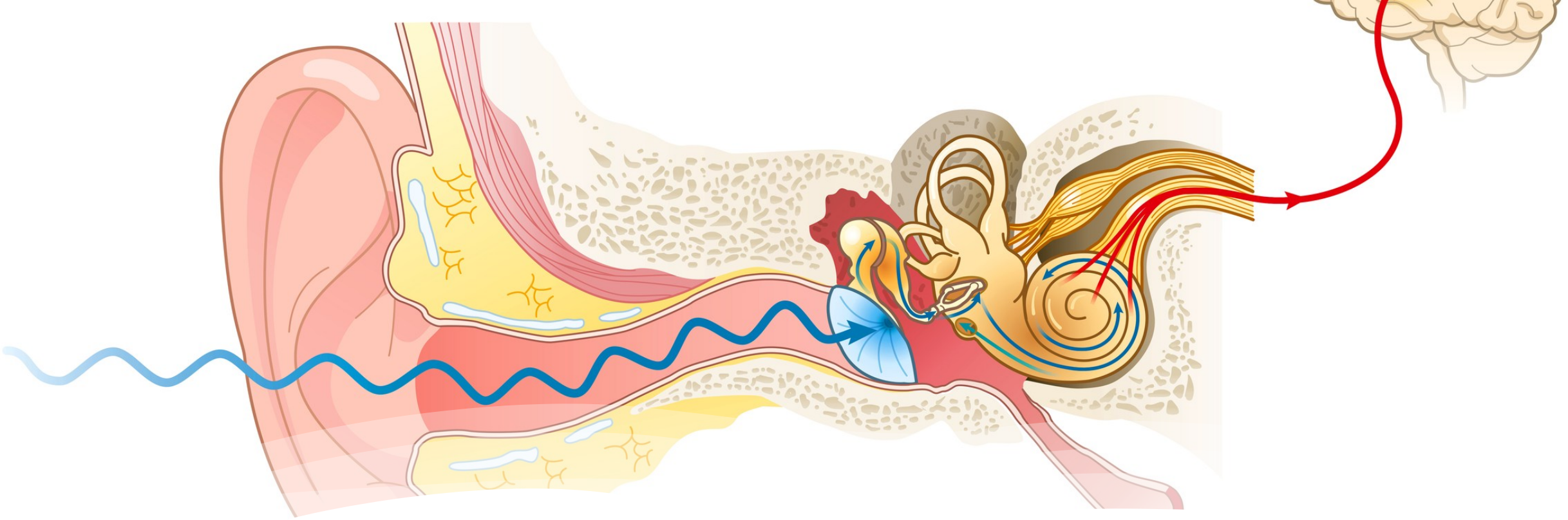
Key concept: Loss of input → brain turns up the volume.

Types of Tinnitus

There are two broad types of tinnitus:

Subjective tinnitus is tinnitus in which only the particular person can hear. This is the most common type of tinnitus. It can be caused by ear problems in your outer, middle or inner ear.

Objective tinnitus is tinnitus in which doctor can hear when he or she does an examination. This is rare type and it may be caused due to blood vessel problem, an inner ear bone condition or muscle contractions.



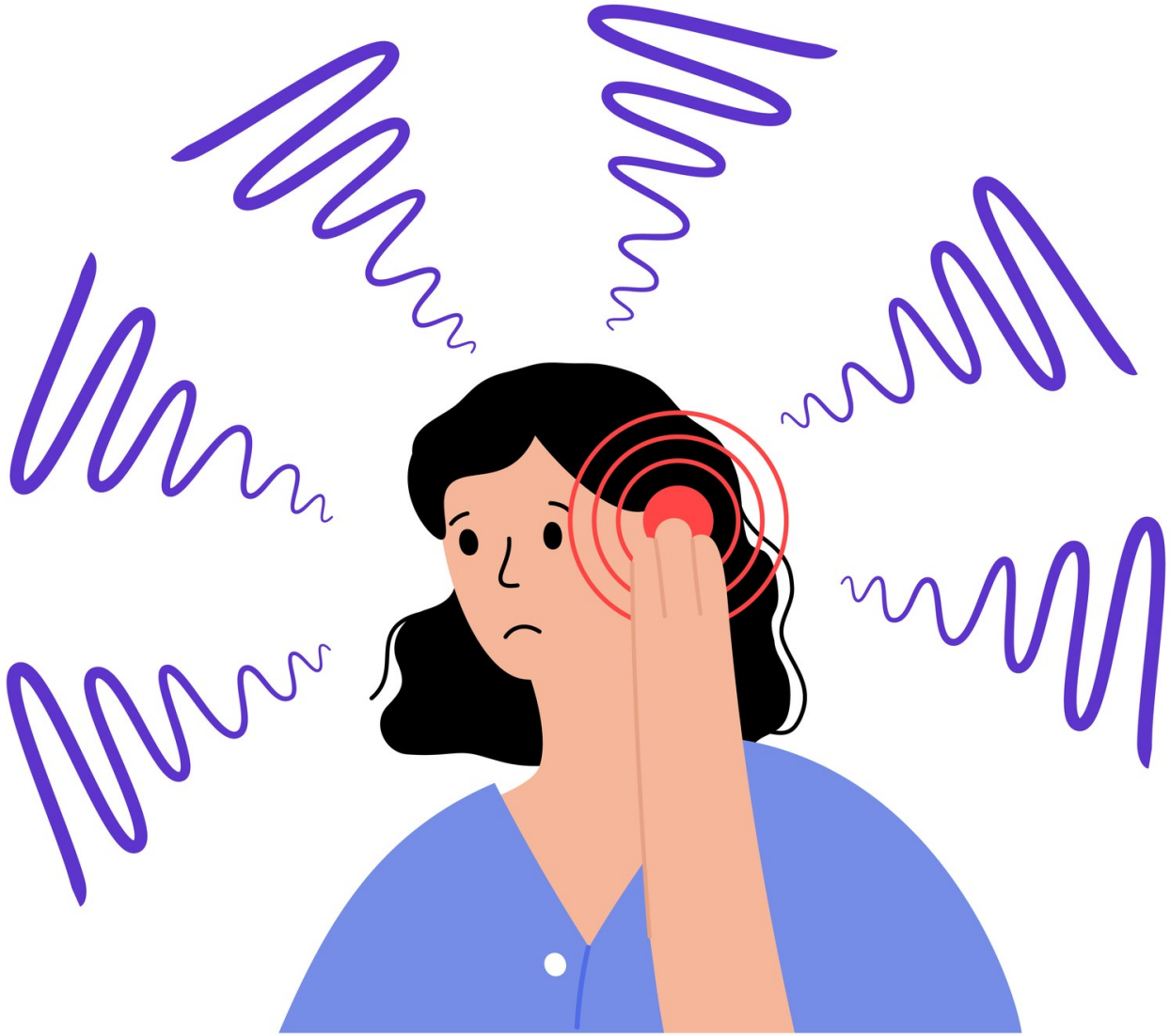
3. Sub-Types of Tinnitus

- Tonal tinnitus
- Pulsatile tinnitus (vascular origin)
- Somatic tinnitus (neck/jaw influence)
- Intermittent neural activation

Tonal Tinnitus

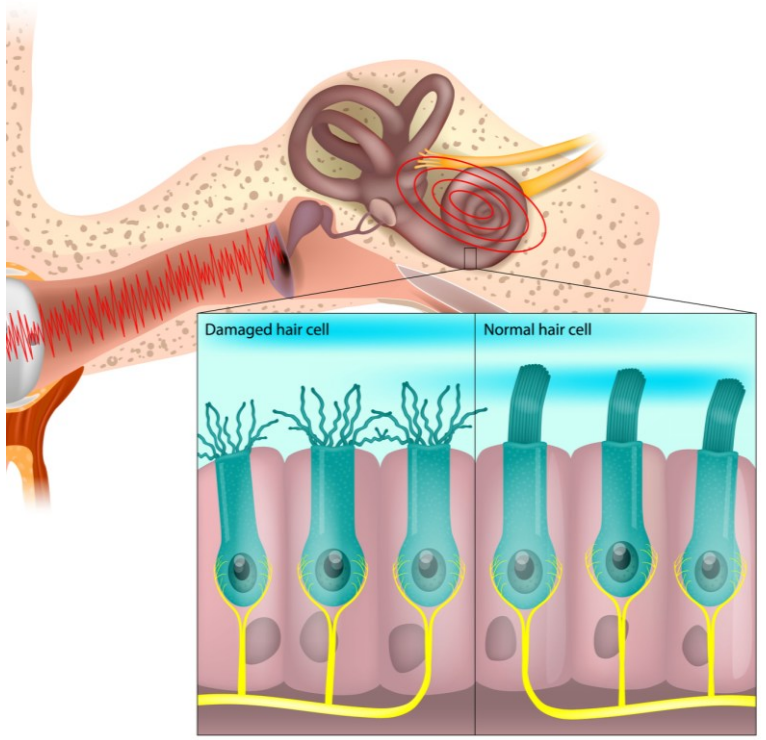
- Continuous internal tone (ringing, buzzing, hissing)
- Most common type
  Often linked to high-frequency hearing loss

Mechanism: Reduced input → brain increases neural gain → perceived sound

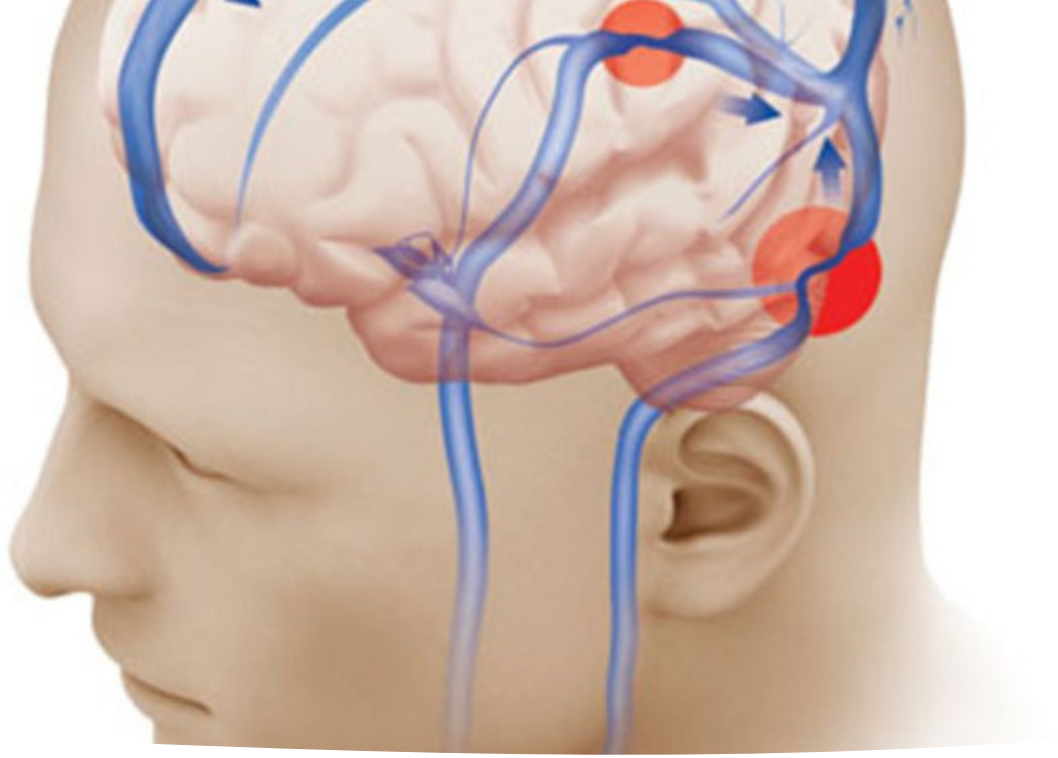


Tonal Tinnitus - What's happening?

When the cochlea is damaged — particularly the outer hair cells responsible for detecting high-frequency sound — the amount of auditory input reaching the brain decreases. The central auditory system does not passively accept this reduced input. Instead, it compensates by increasing neural gain, essentially "turning up the volume," in an attempt to detect missing signals. This heightened spontaneous firing in the auditory cortex is interpreted as sound, even though no external sound is present. In tonal tinnitus, that internally generated neural activity is perceived as a continuous ringing, buzzing, or high-pitched tone.

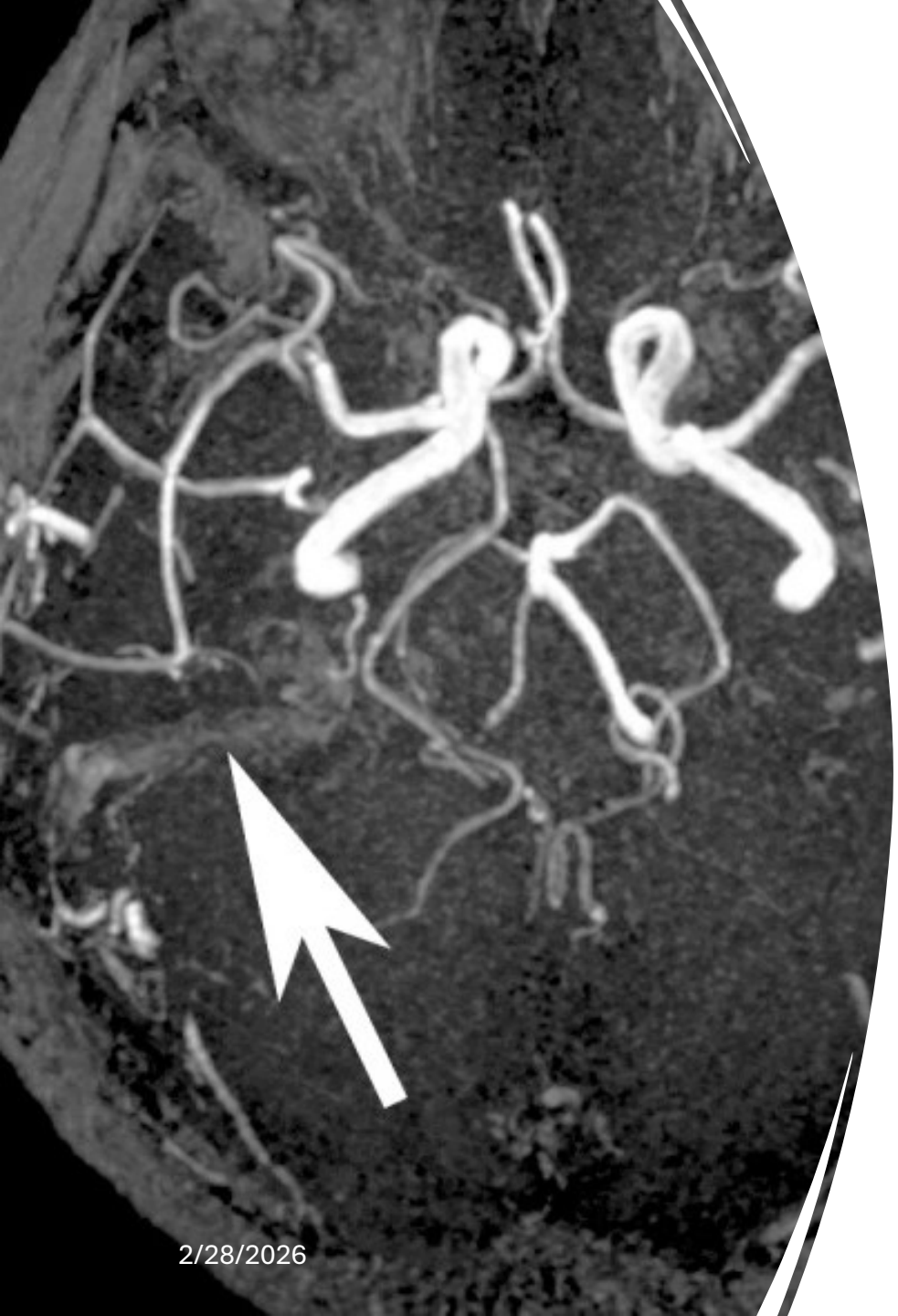


PULSATILE TINNITUS



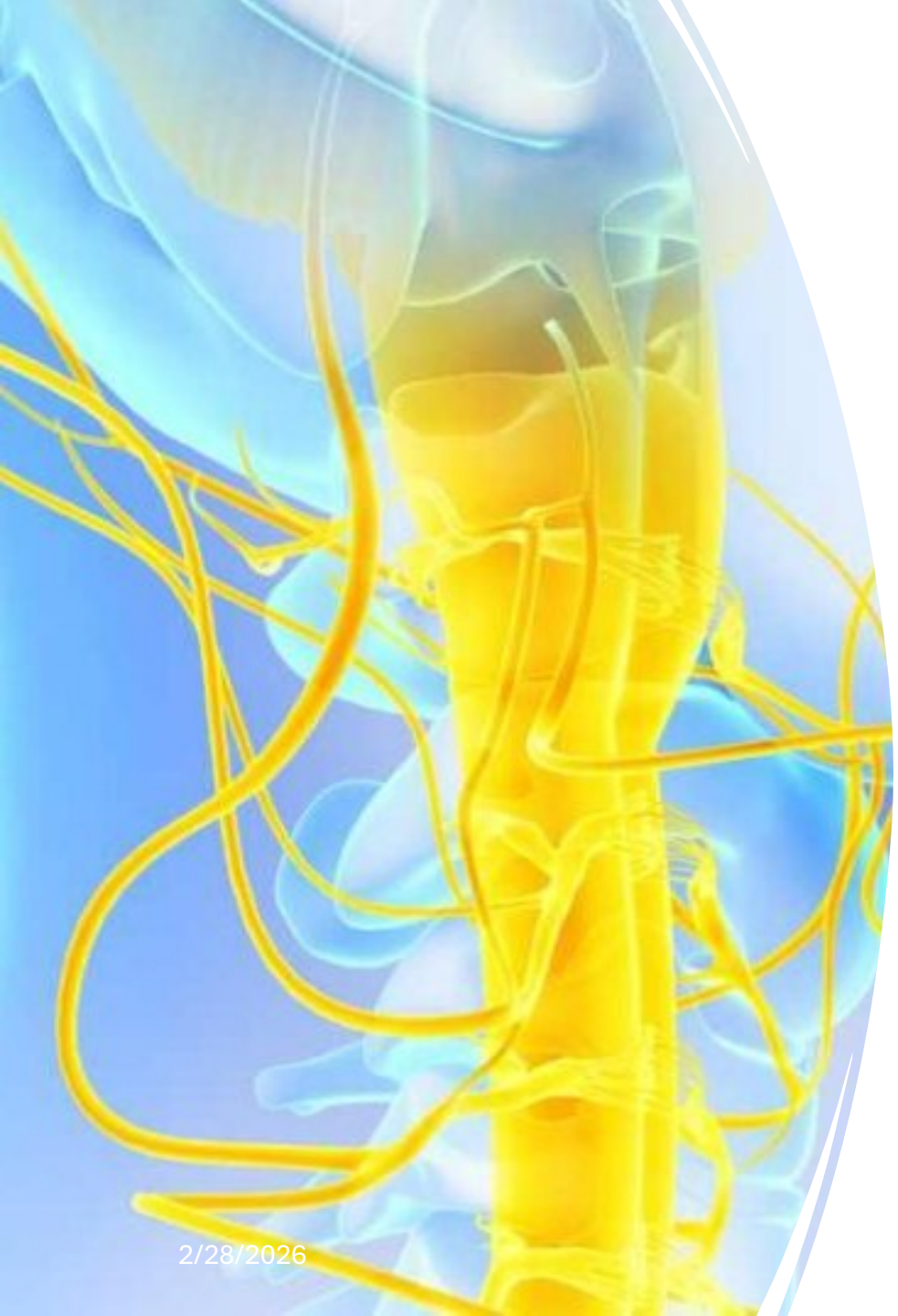
Pulsatile Tinnitus (Vascular)

- Rhythmic sound
  Often matches heartbeat
- Usually vascular in origin
  (turbulent blood flow)
- May indicate structural cause → warrants evaluation



Pulsatile Tinnitus – an inside look

Pulsatile tinnitus differs from **tonal tinnitus** in that it usually has a physical source. Rather than being generated by altered neural firing alone, pulsatile tinnitus is often caused by turbulent blood flow near the ear. Because the inner ear sits close to major arteries and venous sinuses, changes in blood velocity or vessel structure can create rhythmic vibrations that are transmitted to the auditory system. Patients often describe a whooshing or thumping sound that matches their heartbeat. In many cases, pulsatile tinnitus is benign and related to normal vascular flow that has simply become more noticeable. However, it can occasionally indicate a more significant underlying issue — such as carotid artery narrowing, vascular malformations, or rare tumors — which is why new or persistent pulsatile tinnitus warrants medical evaluation.



Somatic Tinnitus

- Changes with jaw or neck movement
- Linked to TMJ or cervical tension
- Reflects cross-talk between sensory and auditory pathways
- Not purely cochlear

Somatic Tinnitus – What's Happening?

Somatic tinnitus occurs when tinnitus changes in intensity or pitch with movements of the head, neck, jaw, or even pressure on certain muscles. Unlike purely cochlear-driven tinnitus, this form reflects interaction between the somatosensory system and the auditory pathways in the brainstem.

Nerves from the cervical spine and temporomandibular joint (TMJ) converge with auditory nuclei, particularly in the dorsal cochlear nucleus. When there is neck tension, jaw dysfunction, or musculoskeletal imbalance, these inputs can alter auditory signaling and modulate tinnitus perception. In other words, the sound is not coming from the ear alone — it is influenced by cross-talk between structural and auditory neural pathways.

Intermittent Neural Activation

- Occasional internal sound
  Comes and goes
- Not constant or progressive
- More consistent with central signaling fluctuation than new structural damage

Intermittent Neural Activation – learn more

Intermittent neural activation refers to **occasional** internal sound perception that comes and goes rather than remaining constant. Unlike classic tonal tinnitus, which is often tied to stable cochlear damage and persistent central gain, intermittent auditory phenomena are more consistent with transient fluctuations in central auditory signaling. The brain's sensory gating systems continuously regulate background neural activity, and small shifts in neuroinflammation, autonomic tone, stress levels, or cholinergic signaling can temporarily alter how auditory signals are processed. In these cases, the perception reflects dynamic central pathway activity rather than new structural injury within the ear itself.

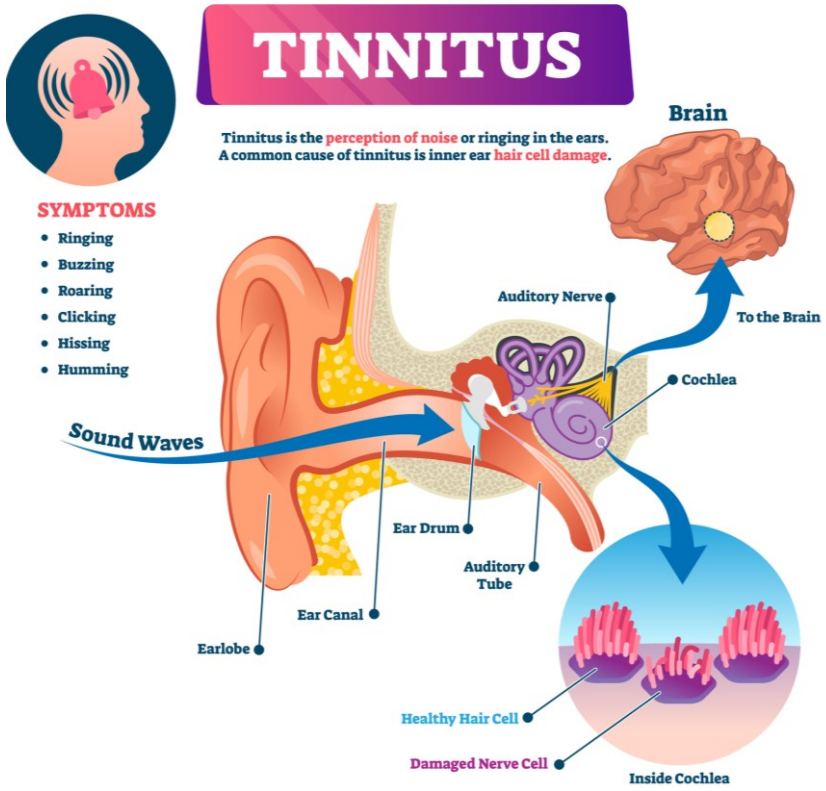
Tinnitus Types – In Summary

Tinnitus is a symptom with multiple origins, not a single condition. The most common form is **subjective tonal tinnitus**, typically associated with sensorineural hearing loss and increased central neural gain.

Less commonly, **pulsatile tinnitus** reflects vascular turbulence and may warrant medical evaluation.

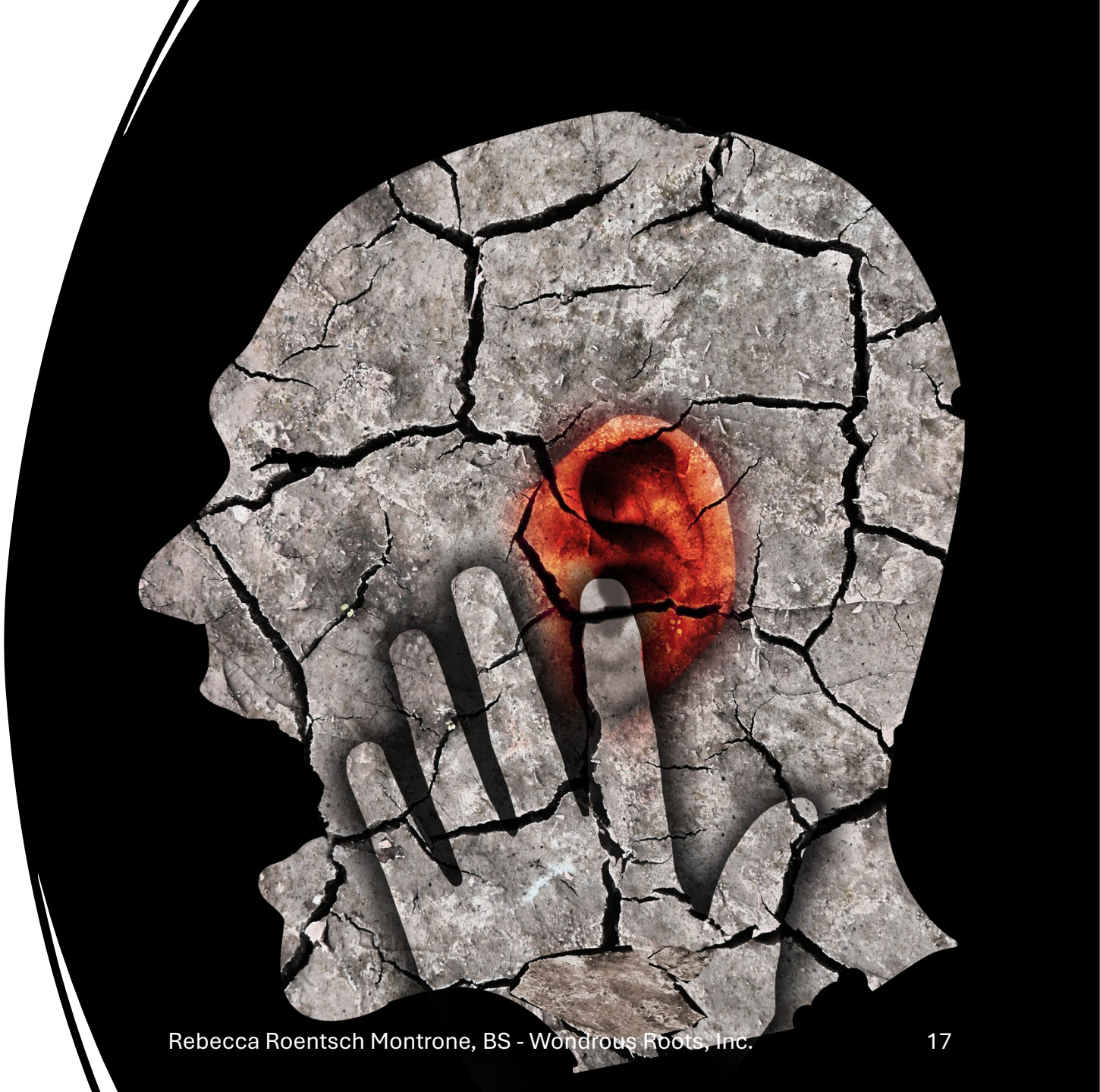
Somatic tinnitus arises from interaction between musculoskeletal and auditory pathways, while **intermittent neural activation** represents transient fluctuations in central signaling rather than fixed structural damage.

Recognizing the mechanism behind tinnitus helps distinguish benign neural patterns from those requiring further assessment and allows for more precise, individualized management.



4. Why Tinnitus Is So Distressing

- Linked to limbic system activation
- Emotional tagging of sound
- Sensory gating disruption
- Hypervigilance



The Terribleness of Tinnitus

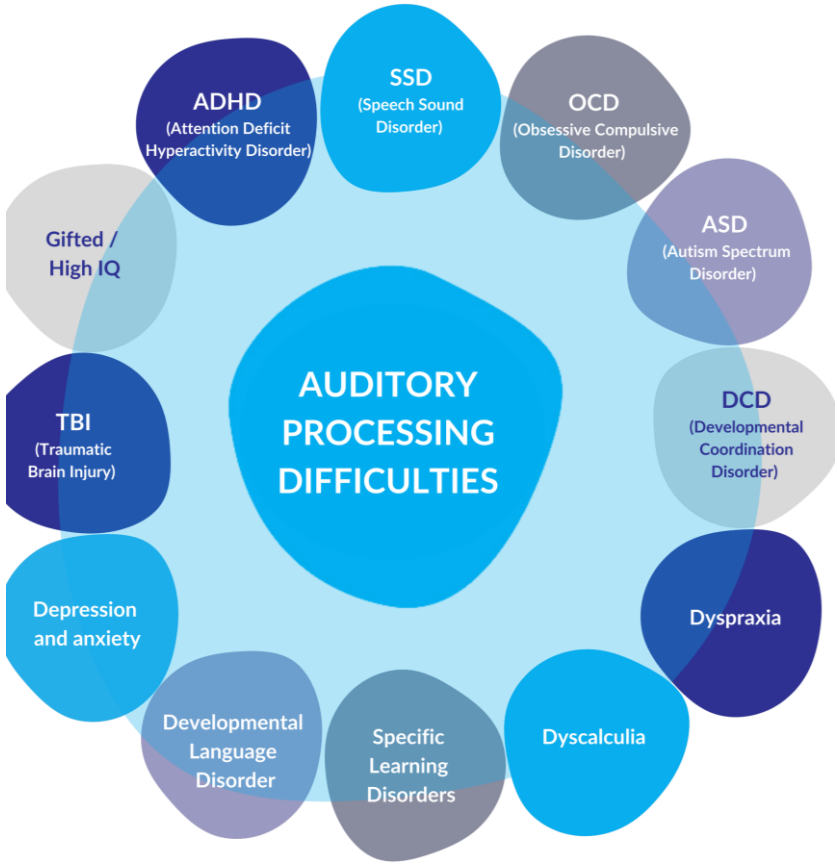
Tinnitus is often distressing not simply because of the sound itself, but because of how the brain processes it. The auditory system is closely linked to the limbic system, which governs emotion and threat perception. When an internal sound is interpreted as unfamiliar or concerning, it becomes emotionally “tagged,” increasing attention and amplifying awareness. At the same time, normal sensory gating — the brain’s ability to filter out background stimuli — can become disrupted, making the sound difficult to ignore. This heightened vigilance reinforces the perception, creating a feedback loop in which attention, anxiety, and neural amplification sustain one another.

5. Other Auditory Perception Disturbances

- **Hyperacusis (sound sensitivity)**
  Ordinary sounds are perceived as disproportionately loud due to increased central auditory gain.
- **Distorted sound perception**
  Sounds may seem warped, metallic, echoing, or unclear when neural signal processing is altered.
- **Sound-induced pain**
  Normal or high-pitched sounds trigger physical discomfort due to auditory pathway sensitization.
- **Intermittent internal auditory phenomena**
  Brief, occasional internal sounds arise from transient fluctuations in central auditory signaling.

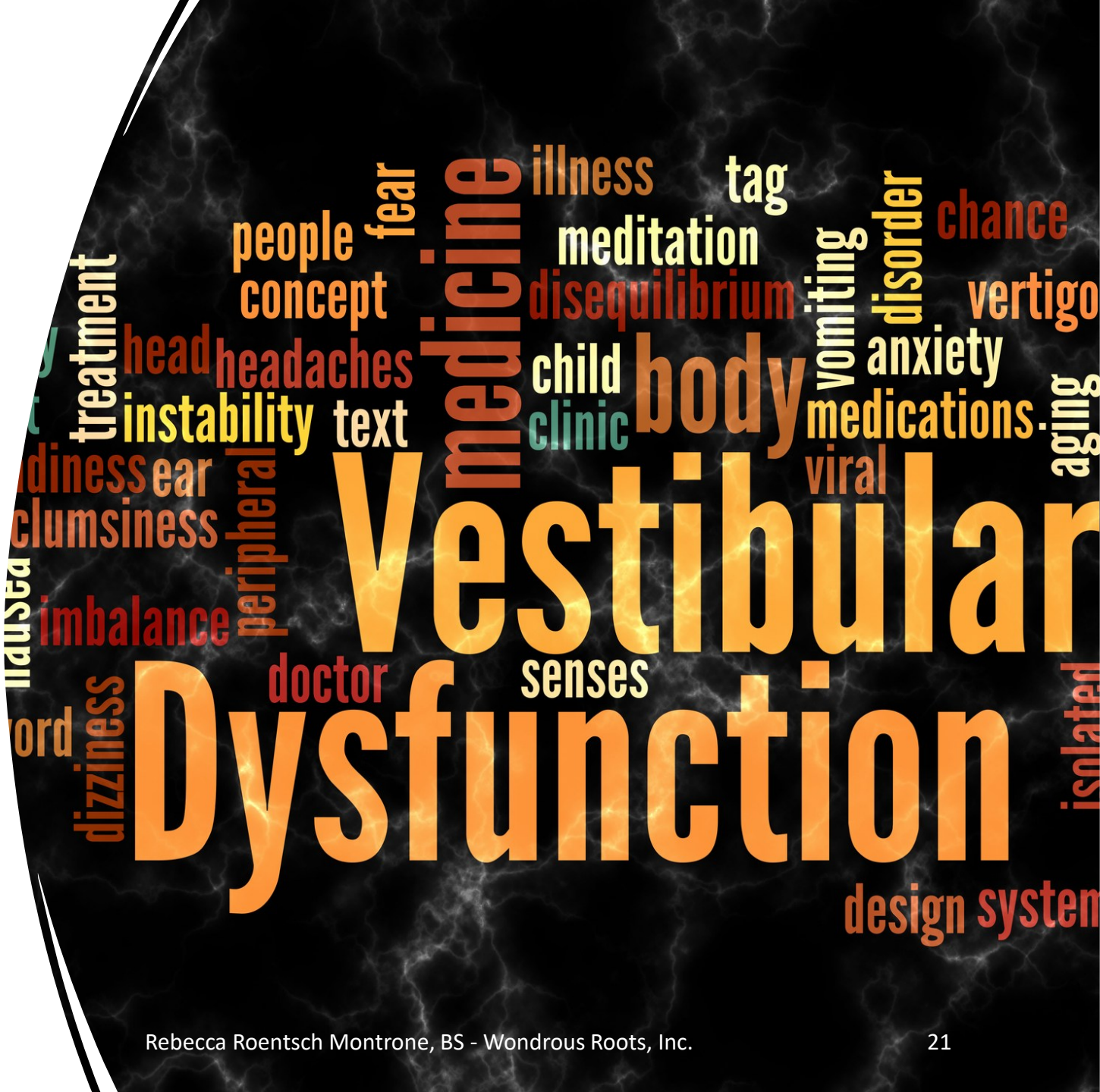
Some Causes of Auditory Processing Difficulties

Auditory processing difficulties are not caused by the ear itself, but by how the brain interprets sound. They are often associated with neurodevelopmental conditions such as ADHD, autism spectrum disorder, speech and language disorders, dyslexia, and dyspraxia, as well as learning disorders, traumatic brain injury, and mood or anxiety conditions. In each case, broader neural differences affect attention, sensory filtering, and signal integration, making it harder to accurately process and interpret auditory information.



II. When Balance Misfires:

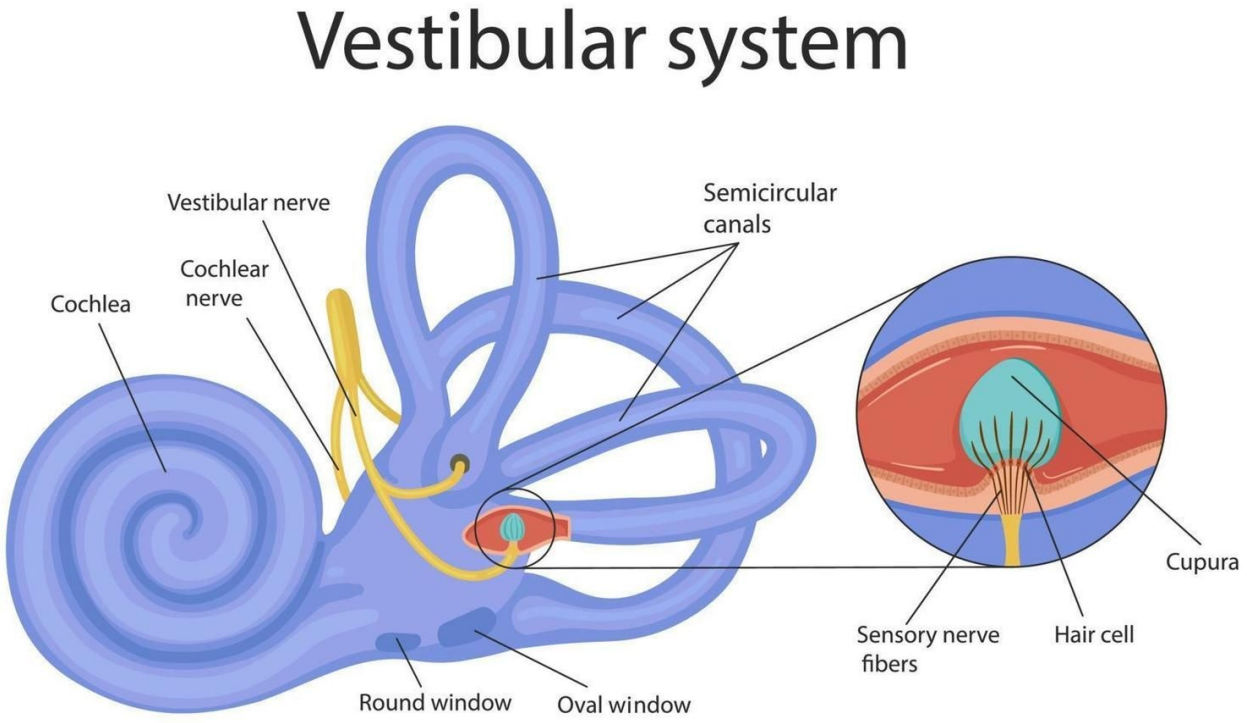
Vestibular Dysfunction



1. Overview of the Vestibular System

Overview of the Vestibular System

- Semicircular canals (rotation)
- Utricle and saccule (linear motion)
- Vestibular nerve (branch of CN VIII)
- Integration with vision and proprioception



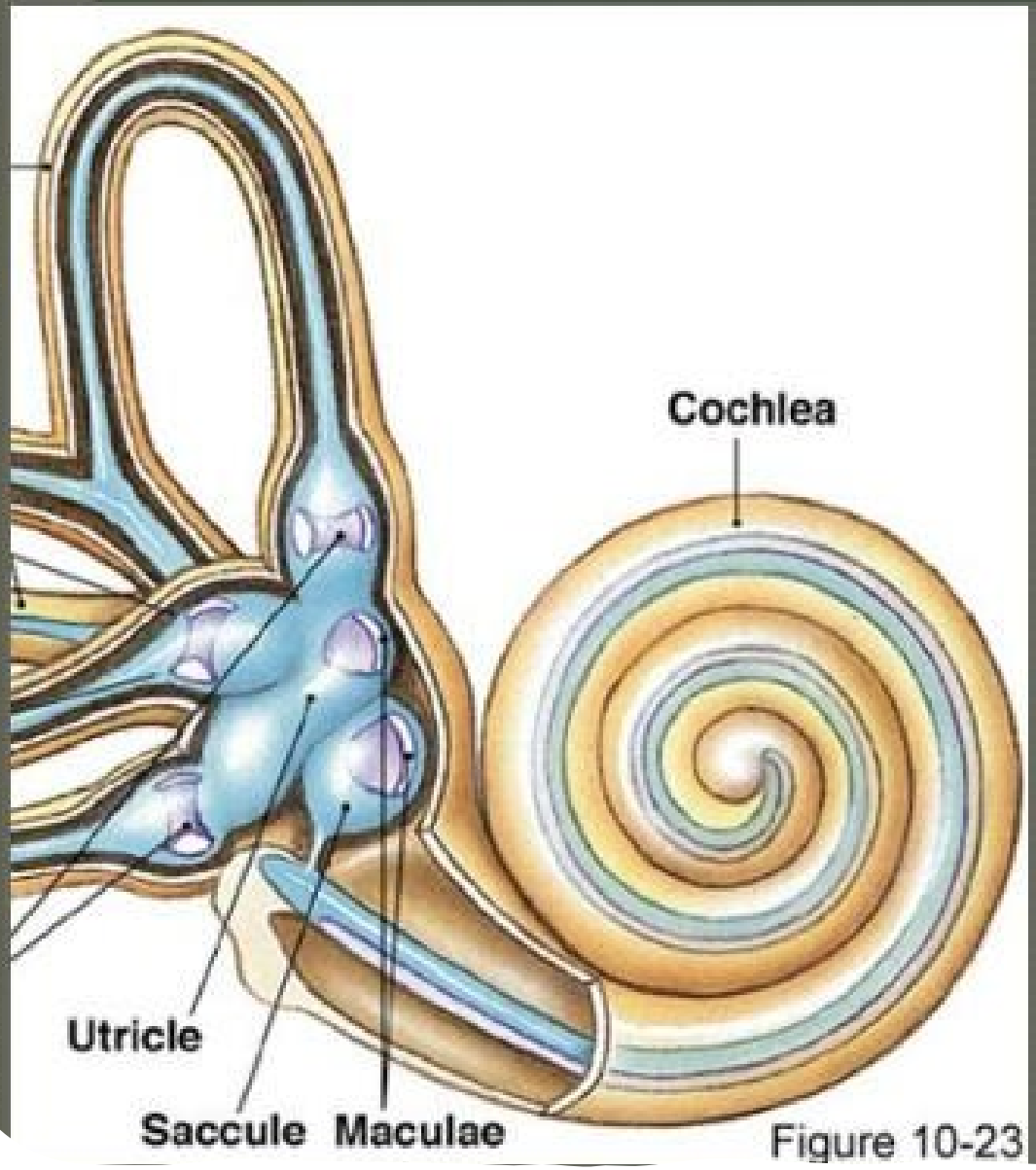


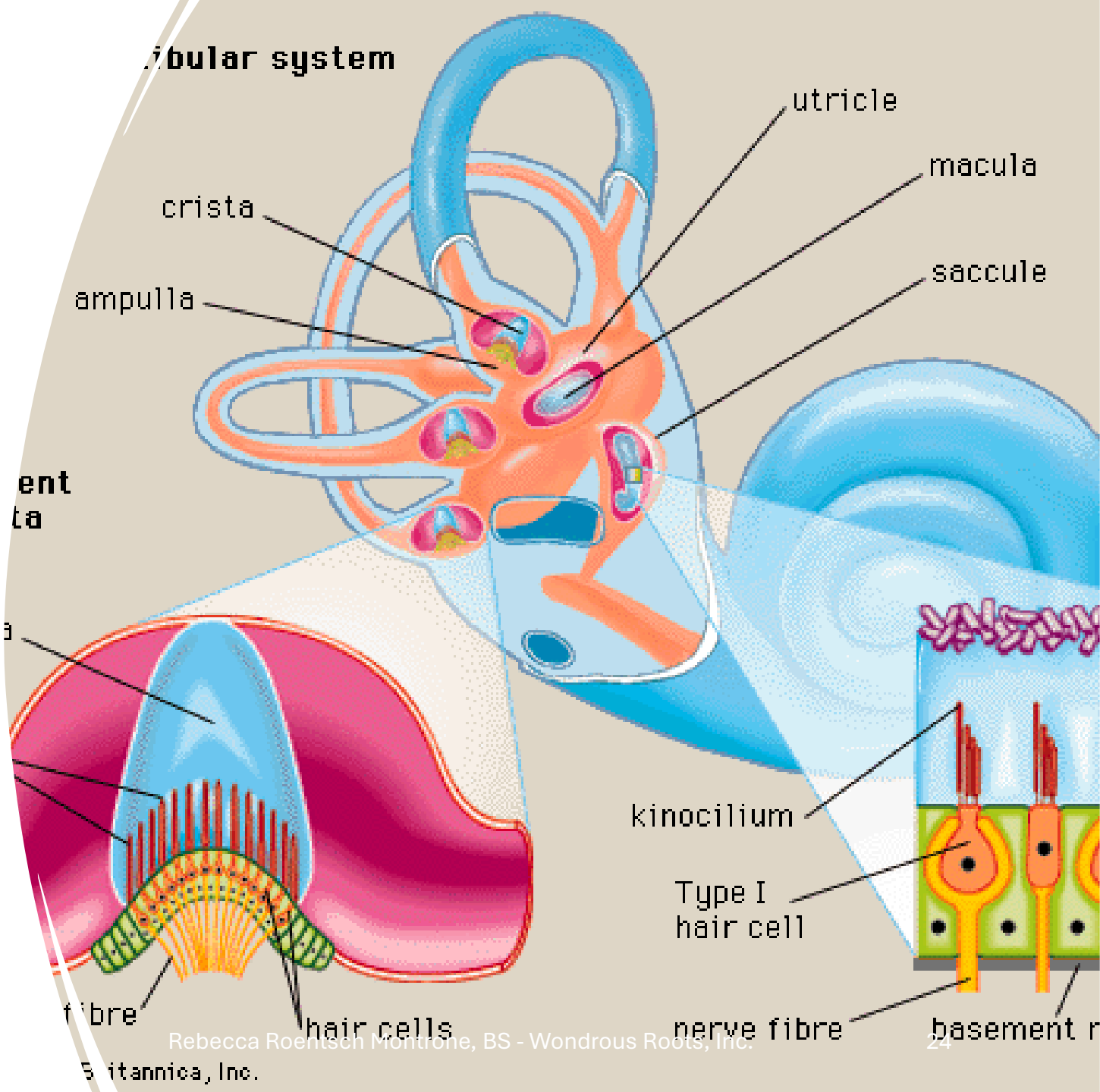
Figure 10-23

Utricle
HORIZONTAL acceleration
Saccule
VERTICAL acceleration

Mechanism:
Otoliths lags behind and pulls on the hair cells

How the Vestibular System Works - in a Nutshell 🐿️

The vestibular system detects motion and spatial orientation through a coordinated sequence of structures. The **semicircular canals** respond to rotational movements of the head, detecting changes in angular acceleration, while the **utricle** and **saccule** sense linear motion and head position relative to gravity. These signals are transmitted through the **vestibular branch of Cranial Nerve VIII** to the brainstem and cerebellum, where they are integrated with **visual input** and proprioceptive feedback from **muscles and joints**. The **brain** continuously compares information from these systems to maintain balance, stabilize gaze, and create a coherent sense of where the body is in space. When even one component misfires, the resulting sensory mismatch can produce dizziness, vertigo, or disorientation.



2. Vertigo vs. Dizziness

- **Vertigo:**

Sensation of spinning or motion

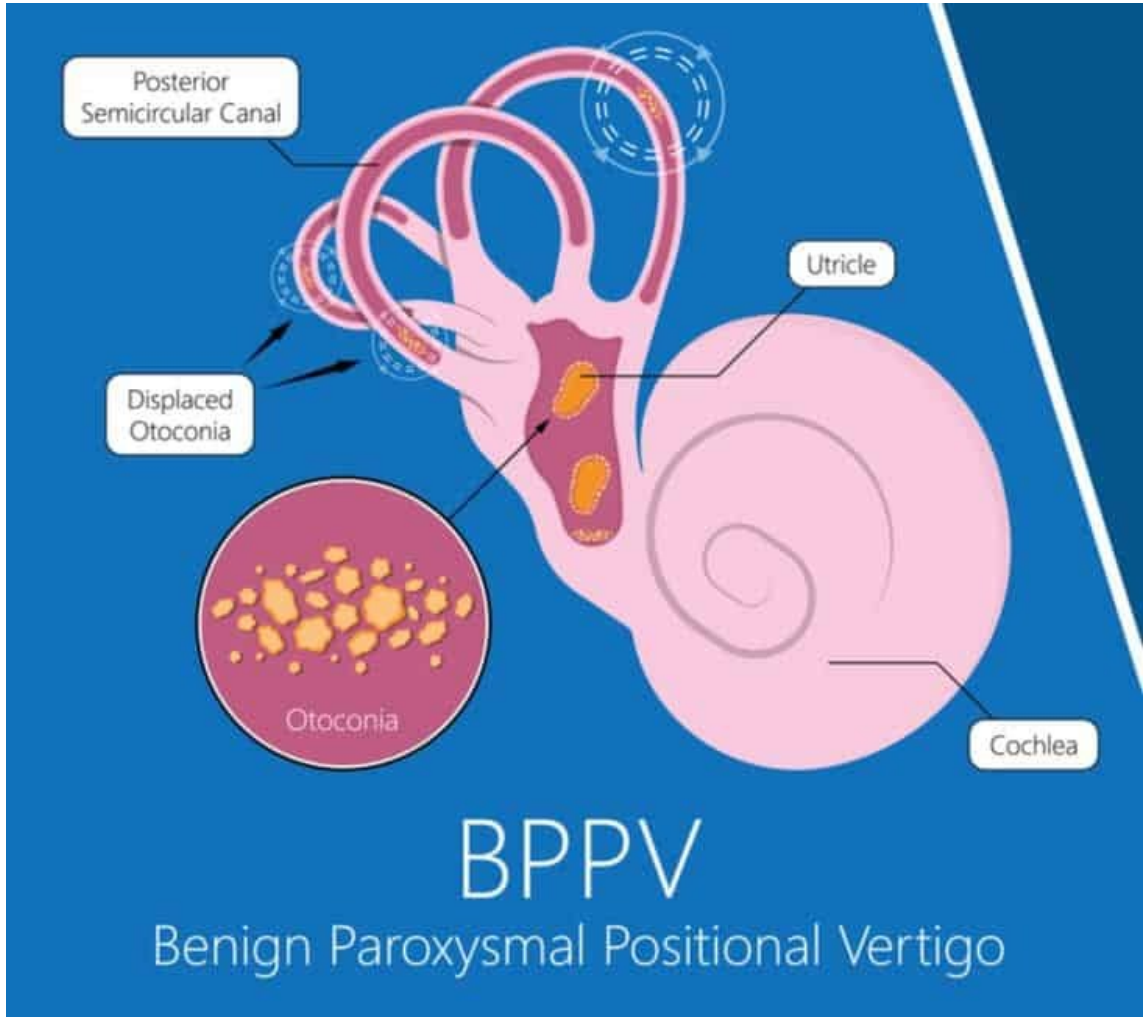
Dizziness:

- Lightheadedness or imbalance

Important distinction for evaluation.

3. Benign Paroxysmal Positional Vertigo (BPPV)

- Displaced otoconia (“crystals”)
- Positional symptoms
- Mechanical cause
- Often correctable



Benign Paroxysmal Positional Vertigo (BPPV)

- Inner ear problem that results in short lasting, but severe, room-spinning vertigo.
- ***Benign***: not a very serious or progressive condition
- ***Paroxysmal***: sudden and unpredictable in onset
- ***Positional***: comes with a change in head position
- ***Vertigo***: causing a sense of dizziness.

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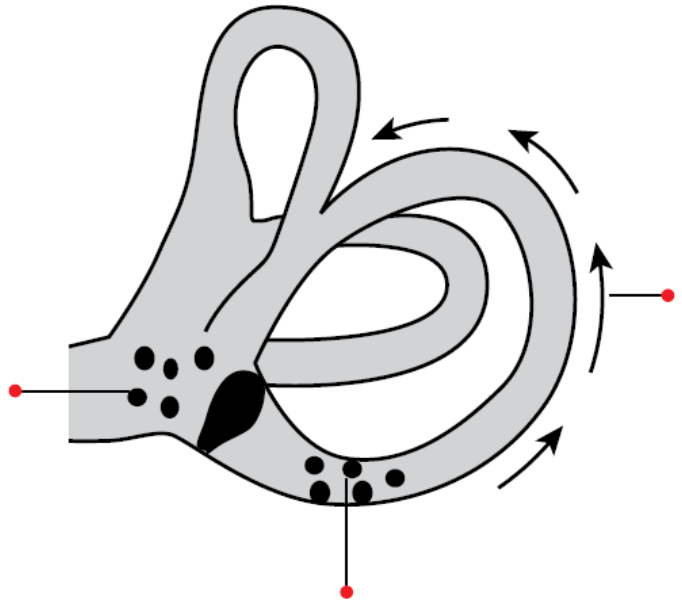
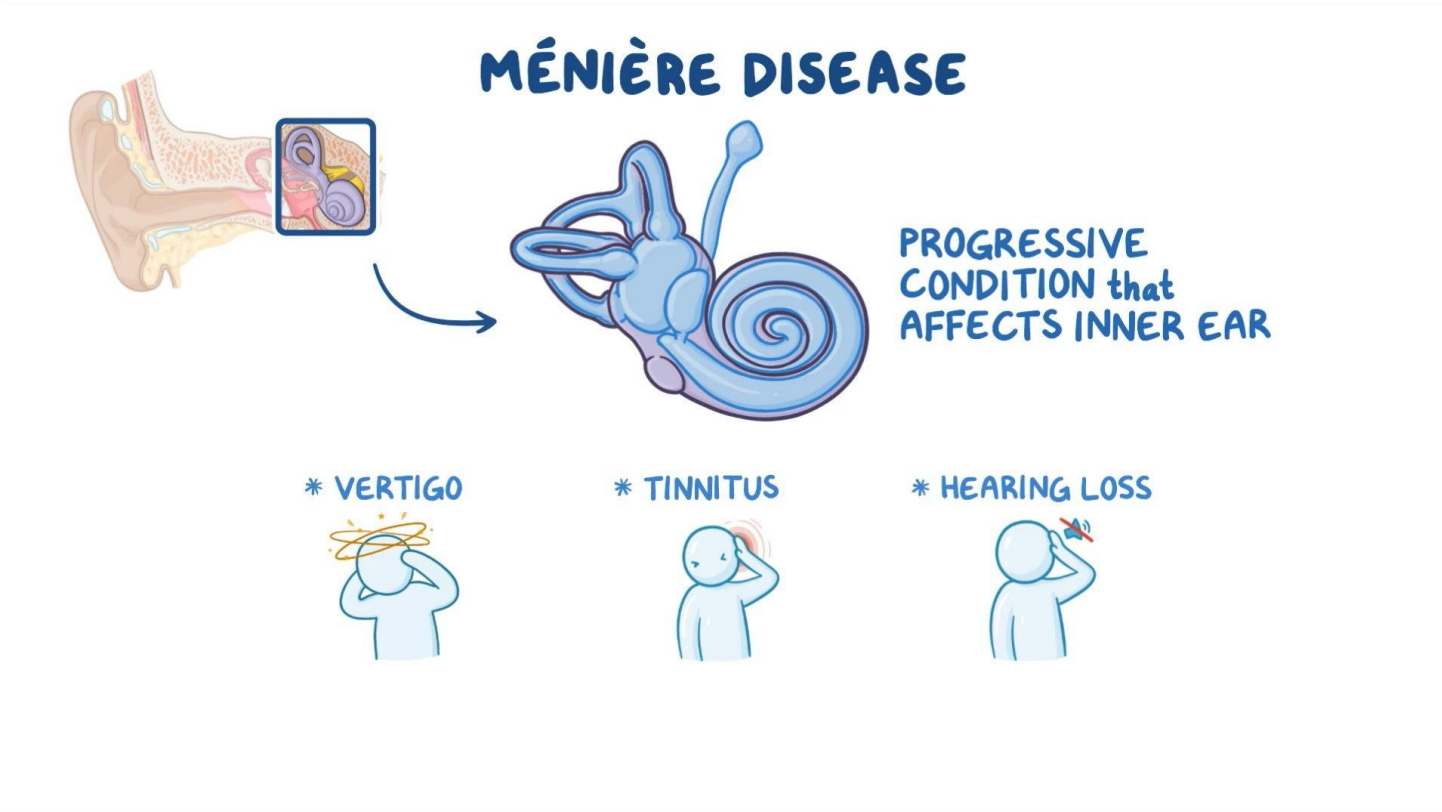


Those Mysterious “Ear Crystals”

Benign Paroxysmal Positional Vertigo (BPPV) occurs when tiny calcium carbonate crystals called **otoconia**, which normally reside in the utricle, become dislodged and migrate into one of the semicircular canals. These canals are designed to detect rotational head movement through fluid motion, not loose particles. When displaced otoconia move within the canal during position changes — such as rolling over in bed or looking upward — they abnormally stimulate the sensory hair cells, sending false signals of rotation to the brain. The result is brief but intense vertigo triggered by specific head movements. BPPV is mechanical rather than inflammatory and is often treatable with repositioning maneuvers that guide the crystals back to their proper location.

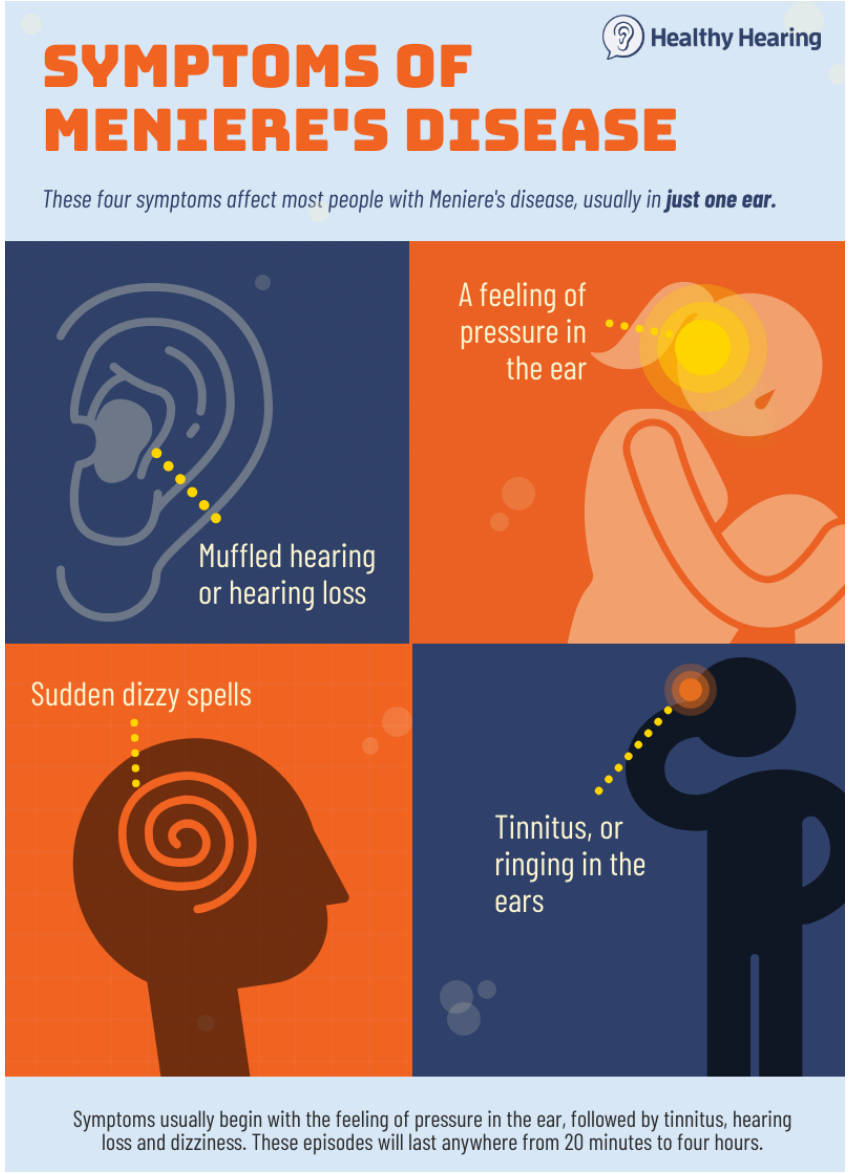
4. Ménière's Disease

- Endolymphatic fluid imbalance
- Episodic vertigo
- Hearing fluctuation
- Tinnitus and fullness



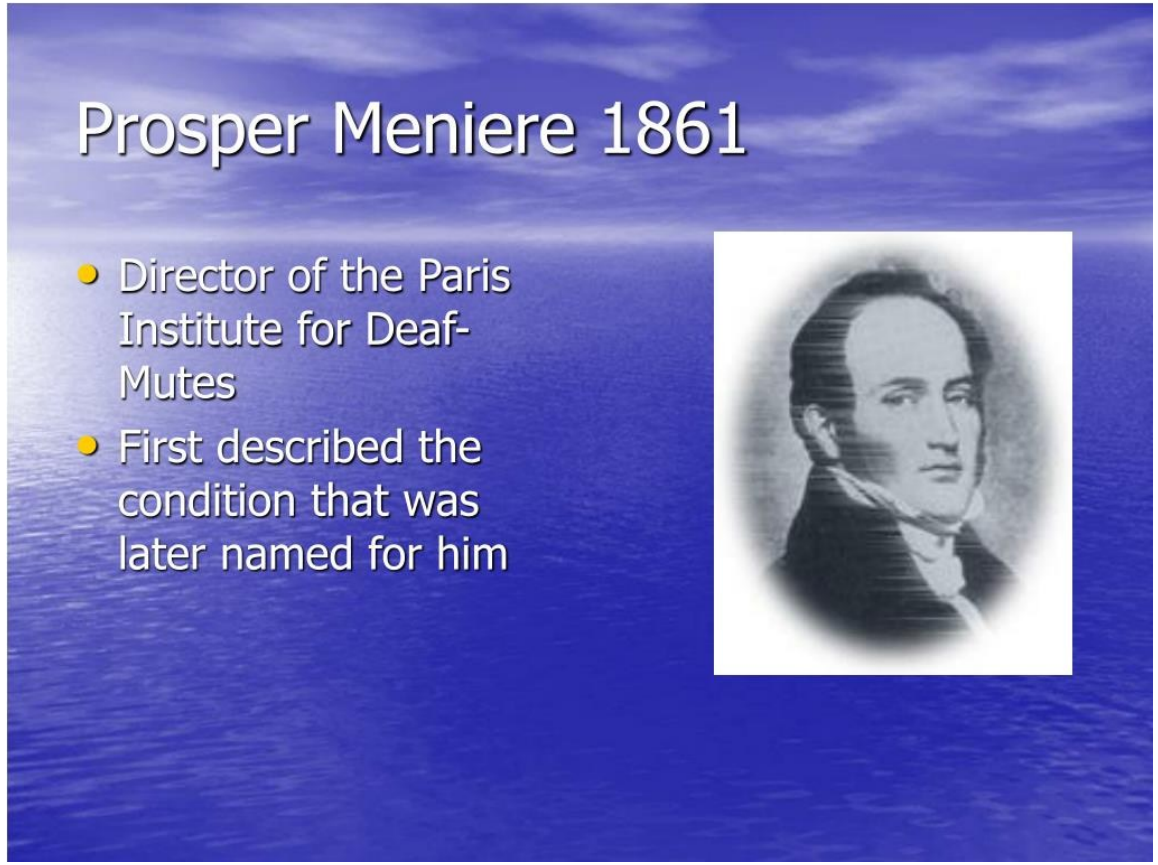
What makes Ménière's, well…*Ménière's!*

Ménière's disease is believed to result from an imbalance of endolymphatic fluid within the inner ear, leading to increased pressure in the membranous labyrinth. This fluid dysregulation disrupts both the cochlear and vestibular structures, producing episodic vertigo that can be severe and unpredictable. Because the cochlea is also affected, individuals often experience fluctuating hearing loss, typically in the lower frequencies early on, along with tinnitus and a sensation of fullness or pressure in the ear. The hallmark of Ménière's is this cluster of symptoms occurring in attacks, reflecting temporary but recurrent disturbances in inner ear fluid dynamics rather than permanent structural collapse at the outset.



Why is it called Ménière's disease?

- It is named after **Prosper Ménière**, a French physician who, in 1861, proposed that vertigo and hearing loss could originate in the inner ear rather than the brain.
- Before his work, vertigo was largely attributed to cerebral causes. Ménière's insight was groundbreaking because he localized the disorder to the labyrinth (inner ear), which was a major shift in medical thinking at the time.
- So the name honors the physician who first correctly identified the inner ear as the source of this symptom cluster.



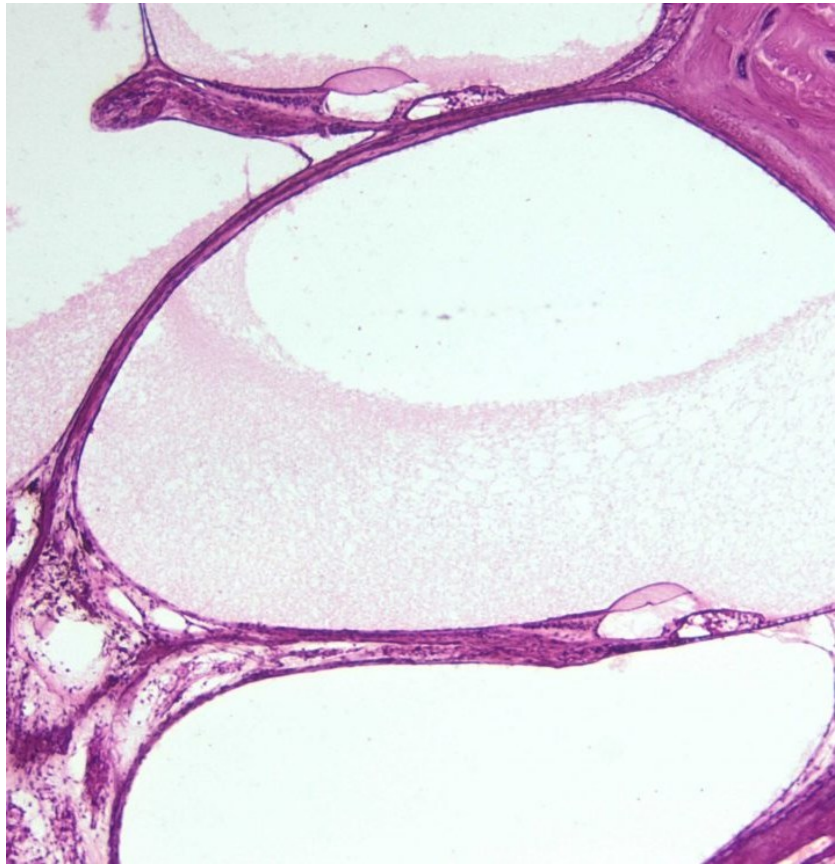
What Triggers the Onset of Ménière's?

Ménière's disease is believed to arise from **endolymphatic hydrops** — abnormal fluid buildup within the inner ear.

Possible contributing factors include:

- Impaired fluid regulation in the endolymphatic sac
- Autoimmune-mediated inner ear inflammation
- Post-viral changes affecting fluid balance
- Microvascular or blood flow dysregulation
- Migraine-related neurovascular instability
- Genetic susceptibility

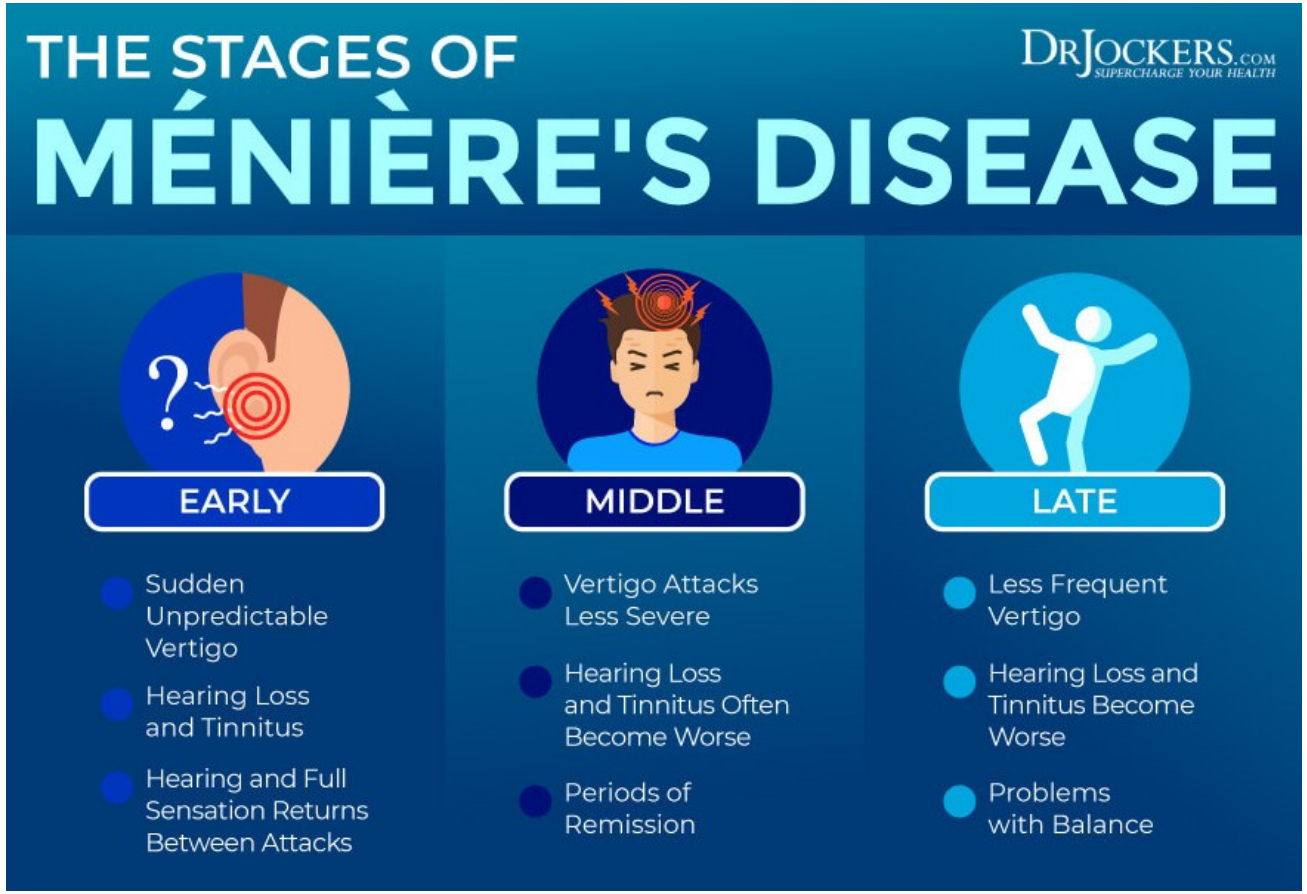
Key concept: Fluid regulation fails → pressure fluctuates → sensory structures become stressed.

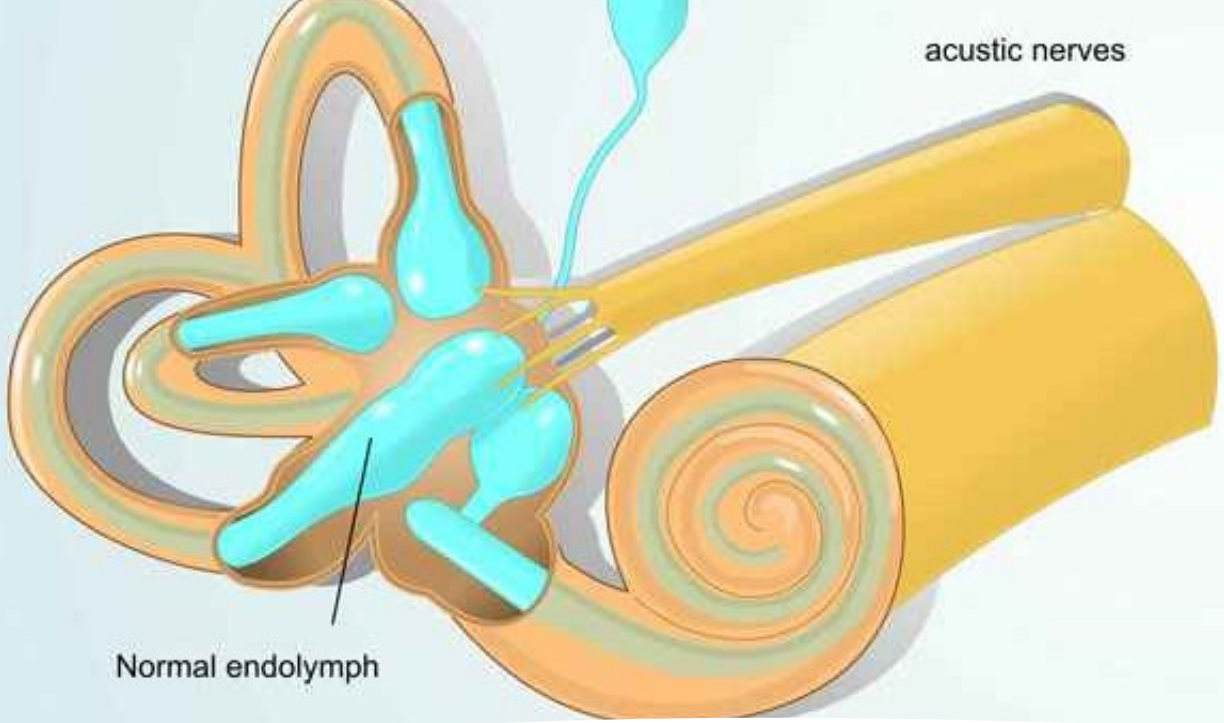


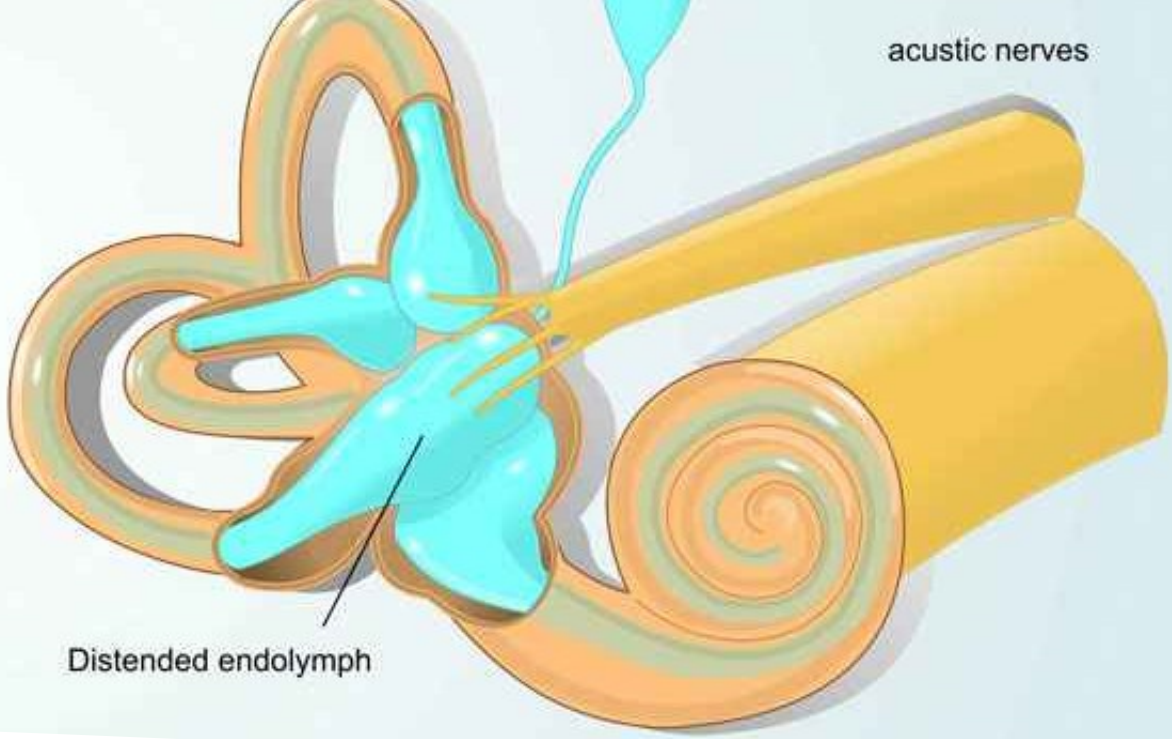
Who Is Most at Risk?

- Most commonly diagnosed between ages **40–60**
  Often begins in **one ear**
- Higher risk seen in individuals with:
  - History of migraines
  - Autoimmune conditions
  - Prior significant viral illness
  - Autonomic instability
  - Family history of inner ear disorders

Important distinction: Ménière's is not caused by noise exposure or simple aging — it is a disorder of inner ear fluid regulation.







Why Does Ménière's Progress?

Ménière's disease is believed to involve repeated episodes of **endolymphatic hydrops** — abnormal fluid buildup within the membranous labyrinth of the inner ear. Each episode of increased pressure can mechanically stress delicate hair cells and membranes, disrupting normal ionic balance and sensory signaling. Over time, these repeated cycles of fluid imbalance and mechanical strain may cause cumulative injury to both cochlear and vestibular structures. As damage accumulates, hearing recovery becomes incomplete, tinnitus stabilizes, and vestibular function gradually declines.

Key concept:
Repeated fluid stress → cumulative sensory injury → permanent loss.

To Be Continued…

As rich as today's discussion has been, we have only begun to explore the full landscape of vestibular dysfunction. In our continuation of Part 3, we will examine post-viral conditions such as vestibular neuritis and labyrinthitis, chronic disequilibrium syndromes, and the role of central compensation — including how the brain integrates hearing, balance, vision, and proprioception to create spatial stability. We will also explore why imaging is often "normal" despite persistent symptoms, and how shared mechanisms such as inflammation, microvascular compromise, autonomic dysregulation, and neuroplastic adaptation contribute to both sound and balance misfires. Understanding these central dynamics is essential, because mechanism determines management — and the inner ear and brain function as one integrated system.