Vestibular neuritis, labyrinthitis, and a few comments regarding sudden sensorineural hearing loss

Marcello Cherchi

§1: What are these diseases, how are they related, and what is their cause?

§1.1: What is vestibular neuritis?

Vestibular neuritis, also called vestibular neuronitis, was originally described by Margaret Ruth Dix and Charles Skinner Hallpike in 1952 (Dix and Hallpike 1952). It is currently suspected to be an inflammatory-mediated insult (damage) to the balance-related nerve (vestibular nerve) between the ear and the brain that manifests with abrupt-onset, severe dizziness that lasts days to weeks, and occasionally recurs.

Although vestibular neuritis is usually regarded as a process affecting the vestibular nerve itself, damage restricted to the vestibule (balance components of the inner ear) would manifest clinically in a similar way, and might be termed "vestibulitis," although that term is seldom applied (Izraeli, Rachmel et al. 1989). Thus, distinguishing between "vestibular neuritis" (inflammation of the vestibular nerve) and "vestibulitis" (inflammation of the balance-related components of the inner ear) would be difficult.

§1.2: What is labyrinthitis?

Labyrinthitis is currently suspected to be due to an inflammatory-mediated insult (damage) to both the "hearing component" (the cochlea) and the "balance component" (the semicircular canals and otolith organs) of the inner ear (labyrinth) itself. Labyrinthitis is sometimes also termed "vertigo with sudden hearing loss" (Pogson, Taylor et al. 2016, Kim, Choi et al. 2018) – and we will discuss sudden hearing loss further in a moment.

Labyrinthitis usually manifests with severe dizziness (similar to vestibular neuritis) accompanied by ear symptoms on one side (typically hearing loss and tinnitus). It is less clear whether labyrinthitis recurs, because frequent recurrence is usually clinically more suggestive of Ménière's disease.

Although labyrinthitis is usually regarded as a process affecting the labyrinth (inner ear) itself, damage to the vestibulocochlear nerve (which transmits hearing and balance signals from the inner ear to the brain) would manifest in a similar fashion clinically; this would be termed "vestibulocochlear neuritis." Thus, distinguishing "labyrinthitis" (inflammation of the entire inner ear) from "vestibulocochlear neuritis" (inflammation of the entire vestibulocochlear nerve) would be difficult.

§1.3: What is sudden sensorineural hearing loss?

Idiopathic sudden sensorineural hearing loss (SSNHL) is currently suspected to be due to an inflammatory-mediated insult (damage) to the "hearing component" (cochlea) of the inner ear, which technically should be termed "cochleitis," though that term is seldom used (Sataloff and Vassallo 1968,

Fitzgerald and Mark 1999, Luigetti, Cianfoni et al. 2011). However, damage to the auditory component of the vestibulocochlear nerve could manifest in a similar fashion clinically; this would be termed "cochlear neuritis." Thus, distinguishing between sudden sensorineural hearing loss from "cochleitis" (inflammatory damage of the hearing-related components of the inner ear) and "cochlear neuritis" (inflammation of the hearing-related nerve) would be difficult (Goodale, Golub et al. 2016).

§1.4: What is the relationship between vestibular neuritis, labyrinthitis and sudden sensorineural hearing loss?

Most textbooks depict vestibular neuritis, labyrinthitis and sudden sensorineural hearing loss as distinct clinical entities, which may be didactically useful, but is probably an oversimplification. The reality is likely more complex since damage can occur to varying proportions of the vestibulocochlear nerve, varying proportions of the labyrinth, and varying combinations thereof; in other words, the underlying pathobiology probably spans a continuum, with a corresponding range of clinical presentations – with different combinations/degrees of vestibular symptoms (dizziness/imbalance) and ear symptoms (hearing loss, tinnitus).

This "continuum" of disease is likely underappreciated because it is under-studied for practical reasons. Specifically, if a patient complains exclusively of hearing symptoms, then she will probably not undergo any balance testing; conversely if a patient complains exclusively of imbalance, then he will probably not undergo any hearing testing. However, if these patients *do* undergo more comprehensive evaluation, then some surprising results may emerge. Some examples follow.

The literature usually describes vestibular neuritis as presenting purely with vestibular symptoms (dizziness/imbalance), and no auditory symptoms. However, when such patients are studied carefully, it turns out that some of them have detectable hearing deficits on the affected side (Yao, Xu et al. 2018), or abnormalities in other auditory functions such as an elevated stapedius reflex threshold (Bergenius and Borg 1983, Bagger-Sjoback, Perols et al. 1993). In other words, "pure" vestibular neuritis may not actually be so pure.

The literature usually describes sudden sensorineural hearing loss as presenting purely with auditory symptoms (hearing loss and sometimes tinnitus), and no vestibular (dizziness/imbalance) symptoms. However, when such patients are studied carefully, it turns out that some of them have detectable vestibular deficits (Hong, Byun et al. 2008, Korres, Stamatiou et al. 2011, Zhang, Hu et al. 2013, Fujimoto, Egami et al. 2015) on the same side as the hearing loss. In other words, the "pure" sudden sensorineural hearing loss may not actually be so pure.

Thus, these terms ("vestibular neuritis"/"vestibulitis", versus "sudden sensorineural hearing loss"/"cochleitis", versus "labyrinthitis"/"vestibulocochlear neuritis") assume distinct clinical presentations (purely vestibular symptoms, purely auditory symptoms, or a combination thereof, respectively), while other terms ("vestibular neuritis" versus "vestibulitis"; "sudden sensorineural hearing loss" versus "cochleitis"; "cochleovestibular neuritis" versus "labyrinthitis") assume that the underlying disease process is restricted to a discrete anatomical area (the neural pathways versus the inner ear, respectively). But these distinctions, while perhaps terminologically convenient, are flawed,

missing what is likely a pathobiological continuum with a corresponding spectrum of clinical presentations. This is illustrated in the **Table** below.

		Spectrum of clinical presentation		
		Exclusively vestibular function affected	**Both vestibular and auditory function affected**	**Exclusively auditory function affected**
Spectrum of anatomy	**Exclusively neural structure involved**	"Vestibular neuritis"	"Vestibulocochlear neuritis"	"Cochlear neuritis"
	Both neural structure and inner ear structure involved	"Vestibular neuritis" + "vestibulitis"	"Vestibulocochlear neuritis" + "labyrinthitis"	"Cochlear neuritis" + "cochleitis"
	Exclusively inner ear structure involved	"Vestibulitis"	"Labyrinthitis"	"Cochleitis"

Table: The anatomical and clinical continuum of this group of diseases.

§1.5: What is the cause of these diseases?

The short answer is that the underlying cause of these diseases is unknown. Viral and vascular causes have been proposed, but reviews of this literature reveal the supporting evidence to be circumstantial at best (Nadol 1995, Strupp and Brandt 2009). Placebo-controlled trials attempting to treat for a presumed viral cause (herpes simplex 1 infection) showed no difference in outcomes (Strupp, Zingler et al. 2004).

§2: How do vestibular neuritis and labyrinthitis present?

Most cases of vestibular neuritis present as a spinning sensation that develops quite rapidly, usually over minutes (less commonly over hours), and is usually associated with nausea and vomiting. Some cases have more of a "sputtering" onset, with brief bursts of dizziness over several days before the continuous symptoms begin.

In some cases of vestibular neuritis patients experience pressure, pain or other discomfort in the affected ear. The mechanism for this sensation is unclear. One possibility is that there are somatosensory fibers (mediating sensations of touch, pain, etc.) in the vestibular nerve, though this has not been described. Another possibility is that the nervus intermedius of Wrisberg, which provides somatosensory innervation to portions of the ear and is neuroanatomically in very close proximity to the vestibular nerve (Tubbs, Steck et al. 2013), is simultaneously affected by whatever inflammatory process causes vestibular neuritis. Another example of multiple adjacent neural structures being affected is

Ramsay-Hunt syndrome (Hunt 1907, Kuhweide, Van de Steene et al. 2002), which affects both the vestibulocochlear nerve and the facial nerve.

Epidemiologic studies (Sekitani, Imate et al. 1993) suggest that the annual incidence of vestibular neuritis is 3.5 per 100,000 people (Strupp and Brandt 2009). Vestibular neuritis is among the most frequently diagnosed conditions in clinics specializing in dizziness. Vestibular neuritis can occur at any age, but peak incidence is in the 3rd – 6th decades, as shown in the histogram below from Sekitani et al (Sekitani, Imate et al. 1993).

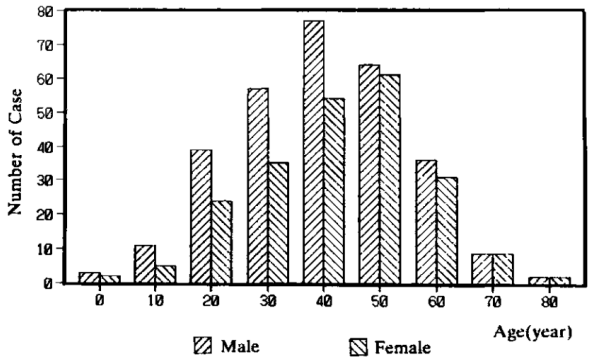

Figure: Histogram by age of the incidence of vestibular neuritis (Sekitani, Imate et al. 1993).

Labyrinthitis is similar to vestibular neuritis in terms of the dizziness/imbalance it causes, but it is different in that labyrinthitis additionally involves auditory symptoms (hearing loss and sometimes tinnitus or ear fullness) on one side; as such, it can be difficult to distinguish labyrinthitis from an initial bout of Ménière's disease.

§3: Diagnosis

§3.1: How are vestibular neuritis and labyrinthitis diagnosed?

These diseases are suspected based on a compatible clinical history.

Suspicion for vestibular neuritis or labyrinthitis increases when otovestibular testing (see below) provides evidence of a vestibular deficit on one side, and there is no evidence for a better explanation of the symptoms.

Suspicion for labyrinthitis increases when audiologic testing shows hearing loss on the same side as the vestibular deficits, and there is no evidence for a better explanation of the symptoms.

§3.2: What tests help diagnose vestibular neuritis?

In order to detect the vestibular (balance-related) deficits of vestibular neuritis and labyrinthitis, several tests may be needed. The reason is that there are multiple components of the inner ear that detect movement, each of which sends signals through a different bundle of fibers in the vestibular nerve;

there does not currently exist a test to assess these components "all at once," rather, there are individual tests for each component.

Briefly, each labyrinth includes five "organelles" that detect motion – specifically, three semicircular canals (the anterior/superior canal, the lateral/horizontal canal, and the posterior/inferior canal) that detect angular acceleration, and two otolith organs (the saccule and utricle) that detect linear acceleration.

Signals from each of these organelles travel along discrete fibers within the vestibular nerve to reach several vestibular nuclei in the brainstem. The microstructural anatomy of the vestibular nerve is difficult to analyze due to a variety of factors – including neuronal anastomoses with adjacent structures (Ozdogmus, Sezen et al. 2004), the gradual rotation in the disposition of the fibers along the course of the nerve (Silverstein, Norrell et al. 1986), the variable orientation between individuals (Rasmussen 1940), variability between genders (Moriyama, Itoh et al. 2007) and the attrition of nerve fibers with age (Bergström 1973, Nagai, Goto et al. 1999) – but a number of painstaking studies on human specimens have been conducted, such as Lee and Suarez' study of 3 temporal bones (Lee, Suarez et al. 1990), and Lopez et al's study of 13 temporal bones (Lopez, Ishiyama et al. 2005), and Bergstrom's study of 40 human temporal bones (Bergström 1973). Representative results are displayed in the Table below.

Source	Superior division (SD) of the vestibular nerve				Inferior division (ID) of the vestibular nerve			Total fibers in entire vestibular nerve
	Anterior canal	Lateral canal	Utricle	Total in superior division	Posterior canal	Saccule	Total in inferior division	
Lee and Suarez (Lee, Suarez et al. 1990)	1811 fibers = **12.8% of total**, and 21.7% of SD.	1692 fibers = **12.0% of total**, and 20.3% of SD.	4839 fibers = **34.2% of total**, and 58.0% of SD.	8342 fibers, 59.0% of total	2053 fibers = **14.5% of total**, and 35.4% of ID.	3744 fibers = **26.5% of total**, and 64.6% of ID.	5797 fibers, 41.0% of total	14,139 fibers
Bergstrom (Bergström 1973)	Anterior + lateral had 5.899 fibers = **32.15% of total**, and 49.77% of SD.		5.952 fibers = **32.44% of total**, and 50.22% of SD.	11.851 fibers, 64.60% of total	2.449 fibers = **13.35% of total**, and 37.71% of ID.	4.046 fibers = **22.05% of total**, and 62.29% of ID.	6.495 fibers = 35.40% of total	18.346 fibers
Lopez and Ishiyama (Lopez, Ishiyama et al. 2005), group 1	1338 fibers = 24.4% of SD.	1480 fibers = 27.0% of SD.	3023 fibers = 55.2% of SD.	5481 fibers	1370 fibers	—	—	—
Lopez and Ishiyama (Lopez, Ishiyama et al. 2005), group 2	1786 fibers = 33.3% of SD.	1824 fibers = 34.1% of SD.	1746 fibers = 32.6% of SD.	5356 fibers	3715 fibers	—	—	—

Table: Representative figures from several human temporal bone studies.

In vestibular neuritis, various subsets of the afferent fibers within the vestibular nerve suffer damage. Some studies (Jeong, Kim et al. 2013, Buki, Hanschek et al. 2017) oversimplify this (perhaps for didactic purposes) by describing vestibular neuritis that involves either the "superior division" of the vestibular nerve, or the "inferior division" of the vestibular nerve, or both, and conclude with observations that most cases involve either the superior division, or both the superior and inferior divisions (Taylor, McGarvie et al. 2016), while it is rare (2.3% (Taylor, McGarvie et al. 2016) to ≈5% (Kim and Kim 2012, Magliulo, Gagliardi et al. 2014, Magliulo, Iannella et al. 2015)) for only the inferior division to be involved. However, careful studies show that this pathology can affect nearly any combination of the subsets of fibers (Taylor,

McGarvie et al. 2016). Taylor and McGarvie studied 43 patients with vestibular neuritis who had a fairly complete set of vestibular tests and found the data in the **Figure** below.

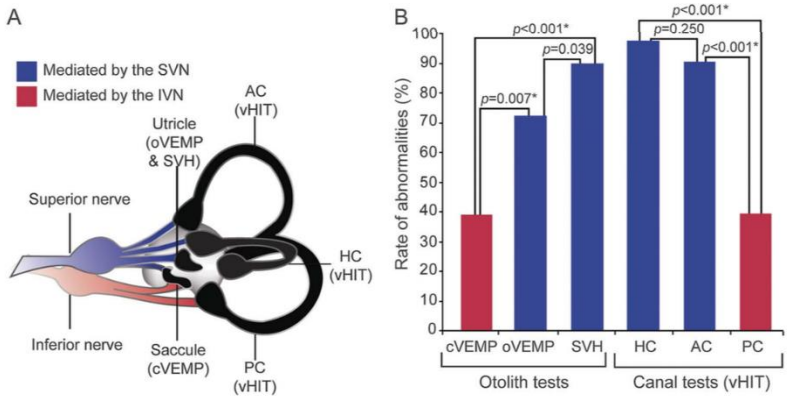

Figure: Data from Taylor and McGarvie (Taylor, McGarvie et al. 2016).

A slightly larger stud (59 patients) by Navari and Casani (Navari and Casani 2020) described fairly similar results, as shown in the **Figure** below.

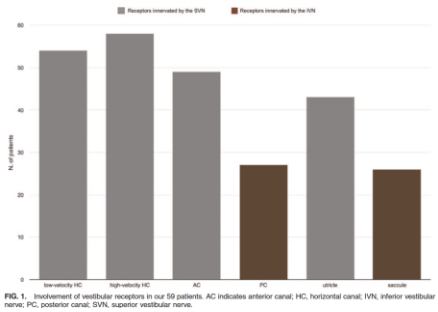

Figure: Data from Navari and Casani (Navari and Casani 2020).

The data from these studies are compared in the **Table** below.

	Horizontal canal, high velocity	**Horizontal canal, low velocity**	**Anterior canal**	**Utricle**	**Posterior canal**	**Saccule**
Test	vHIT	Caloric	vHIT	Ocular VEMP	vHIT	Cervical VEMP
Taylor and McGarvie	97.7%		90.7%	72.1%	39.5%	39.0%
Navari and Casani	97%	90%	83%	73%	46%	44%

Table: Comparison of two studies describing labyrinthine deficits in vestibular neuritis.

These data suggest that in cases of vestibular neuritis, the horizontal canal (or its afferent pathways) is most commonly affected (90% - 97.7%), the saccule (or its afferent pathways) are least commonly affected (39.0% - 44%), whereas the other structures (anterior canal, utricle, posterior canal) exhibit an intermediate level of affectation.

Given this variability, and given that all tests are fallible (meaning that any test can have false positives and false negatives), we generally try not to base the diagnosis on a single test result in isolation; confidence in the diagnosis increases when multiple independent data points (from separate tests) support the same diagnosis. Tests commonly used in diagnosing vestibular neuritis include cervical vestibular evoked myogenic potentials (cVEMPs), ocular vestibular evoked myogenic potentials (oVEMPs), videonystagmography (VNG) and video head impulse testing (vHIT); this battery is a reasonable selection that assesses many of the inner ear's "balance functions," but it still does not provide a complete picture thereof. Ongoing research is exploring other methods of assessing vestibular neuritis (Cherchi 2019).

Occasionally, MRI of the brain and internal auditory canals without and with contrast shows enhancement of the vestibular nerve (Karlberg, Annertz et al. 2004, Fundakowski, Anderson et al. 2012, Venkatasamy, Huynh et al. 2019), though larger studies did not find this (Hasuike, Sekitani et al. 1995, Strupp, Jager et al. 1998), suggesting that this finding is uncommon. When enhancement of the vestibular nerve is observed (without any increased "bulk" of the nerve), it can be viewed as compatible with vestibular neuritis, though is not required for the diagnosis.

§3.3: What tests help diagnose labyrinthitis?

For the vestibular (dizziness/imbalance) component of labyrinthitis, the same tests described for vestibular neuritis are used.

For the auditory part of labyrinthitis (hearing loss, tinnitus), hearing tests are used, including standard audiometry, and otoacoustic emissions. Almost any pattern of sensorineural hearing loss can be seen in labyrinthitis, but the most common pattern seen is one of predominantly high frequency sensorineural hearing loss.

We do not usually assess brainstem auditory evoked responses in these patients, as the degree of hearing loss may make the results uninterpretable.

§4: Treatment, prognosis and recurrence

§4.1: How do you treat the dizziness/imbalance symptoms resulting from vestibular neuritis and labyrinthitis?

During the first few days of vestibular neuritis, management is supportive, sometimes including anti-emetics (such as ondansetron, metoclopramide or promethazine) and vestibular suppressants (such as meclizine or clonazepam), but these medications play no role beyond the first few days.

Meta-analyses of treatment trials (McDonnell and Hillier 2015) conclude that there is moderate to strong evidence that appropriately targeted vestibular rehabilitation therapy is safe and effective for vestibular neuritis, thus this treatment modality is generally regarded as the standard of care.

Physical therapy is thought to accelerate recovery by promoting sensory substitution and central compensation for the vestibular deficit (Strupp and Brandt 2009). Physical therapy usually is usually initiated with gaze stabilization exercises (Meldrum and Jahn 2019), but evolves depending on an individual's progress.

There is some evidence that the earlier the vestibular rehabilitation therapy is started, the greater the likelihood that it will improve specific physical examination findings in vestibular neuritis (Michel, Laurent et al. 2019).

Note that even with appropriately-targeted vestibular rehabilitation therapy, recovery may be experienced as "uneven," in the sense that from day to day there may be fluctuation in the intensity of baseline symptoms, though when observing symptoms over a longer time frame (typically weeks) the overall trajectory is one of improvement.

§4.2: Are medications used in the treatment of vestibular neuritis?

Aside from providing supportive care during the first few days of symptoms (see above), medications play no role in the long-term management of vestibular neuritis.

An early trial (Strupp, Zingler et al. 2004) concluded that starting steroids (specifically, methylprednisolone) within 3 days of symptom onset improved outcomes at 12 months. However, subsequent trials (Yoo, Yang et al. 2017) were unable to replicate this finding, and meta-analyses of such trials (Fishman, Burgess et al. 2011) concluded that on the whole the evidence does *not* support treatment of vestibular neuritis with steroids.

Studies of vestibular rehabilitation therapy PLUS steroids found that the adjunctive steroids conferred *no* additional long-term benefit (Goudakos, Markou et al. 2014, Ismail, Morgan et al. 2019).

§4.3: What is the prognosis of vestibular neuritis?

Most patients diagnosed with vestibular neuritis understandably want to know, "When will I get better?" Unfortunately, this is a difficult question to answer because the *symptom* of dizziness is hard to measure and therefore hard to study. In fact, most research of the "prognosis" of vestibular neuritis study an objective (numeric) measure such as a test result, rather than a patient's complaint of a greater or lesser degree of severity of symptoms. For instance, the carefully conducted study by Strupp et al (Strupp, Zingler et al. 2004) measured the results of caloric testing (a subset of videonystagmography) rather than patient symptoms.

Many studies of vestibular neuritis comment that symptoms "usually" resolve within days to weeks (Kim, Kim et al. 2011). However, there are also studies documenting a less favorable prognosis. One study suggested that up to 30 – 40% of patients experience persistent dizziness after vestibular neuritis (Neuhauser 2007). Another study concluded that up to 43.3% of patients remain at least somewhat

symptomatic at 5 – 10 years (Okinaka, Sekitani et al. 1993). The underlying reason for this variability in outcomes is unknown.

Vestibular neuritis does not usually recur (see below). However, an ear affected by vestibular neuritis is at higher risk for developing other vestibular disorders, particularly benign paroxysmal positional vertigo (BPPV). Studies conclude that after a bout of vestibular neuritis, 9.8% (Mandala, Santoro et al. 2010) to 15.3% (Kim, Kim et al. 2011) developed benign paroxysmal positional vertigo within about 6 years.

§4.4: Does vestibular neuritis recur?

Vestibular neuritis can recur, but is thought to be uncommon. Most studies report recurrence rates of 1.9% - 2% over 10 years (Huppert, Strupp et al. 2006, Mandala, Santoro et al. 2010). Other studies cite rates of recurrence as high as 10.7% (Kim, Kim et al. 2011). In some cases of recurrent vestibular neuritis, the recurrence affects "the other ear" (i.e., the originally unaffected ear); this is called "bilateral sequential vestibular neuritis" (Schuknecht and Witt 1985, Ogata, Sekitani et al. 1993, Yacovino, Finlay et al. 2018).

§4.5: How is labyrinthitis treated?

The dizziness/imbalance symptoms from labyrinthitis are treated the same as for vestibular neuritis (described above), which is to say with appropriately targeted vestibular rehabilitation therapy.

The auditory symptoms (hearing loss and tinnitus) may necessitate other treatment. Some practitioners, based on extrapolation from treatment trials for sudden sensorineural hearing loss, attempt a course of oral steroids, even though the data supporting that approach are weak (Cinamon, Bendet et al. 2001, Nosrati-Zarenoe and Hultcrantz 2012). If that fails, referral to otolaryngology to be evaluated for a transtympanic injection of steroids (Garavello, Galluzzi et al. 2012) is sometimes considered, even though the evidence supporting that approach are also weak (Crane, Camilon et al. 2015, El Sabbagh, Sewitch et al. 2017).

§4.6: What is the prognosis of labyrinthitis?

The prognosis of the vestibular (dizziness/imbalance) component of labyrinthitis is similarly unpredictable as vestibular neuritis (discussed earlier).

The hearing loss from labyrinthitis may improve spontaneously to some degree over about 4 – 6 months. A common clinical practice is to perform a hearing test as close as possible to the beginning of the disease, and then repeat a hearing test at 4 – 6 months after symptom onset. If the hearing loss appears to have "plateaued," then consultation with audiology is appropriate in order to discuss amplification options (such as a hearing aid) and perhaps masking strategies (if tinnitus is present).

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