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Vestibular Rehabilitation Mini Series

Session One: Differential Diagnosis

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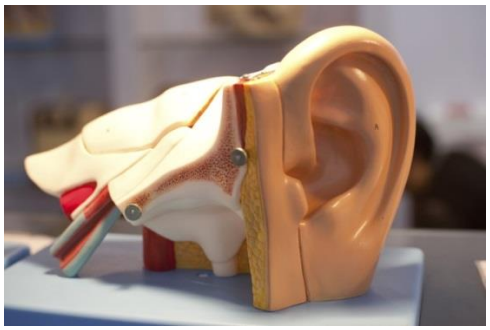

WEBINAR MINI SERIES: VESTIBULAR REHABILITATION
Dizziness: Differential Diagnosis and Management

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Background:
In general, peripheral vestibular disorders are not correctly diagnosed or managed. If we take the emergency department as an example, misdiagnosis rates are in the region of 74-81%(1, 2). However, dizziness is one of the top reasons for consulting a healthcare professional and the lifetime prevalence of dizziness is around 30% (3). Despite the poor recognition rates in primary and secondary care, evidence based treatments do exist and are usually very effective. Due to the clinician's own 'dizzyphobia' there is often an over-reliance on unnecessary and costly investigations such as MR imaging (4). In clinical practice, common disorders such as vestibular neuritis and BPPV are often confused with each other and for other more serious central causes such as strokes. In order to improve the situation clinicians can empower themselves with the basic knowledge of the most common peripheral vestibular disorders. The likelihood of a more serious central disorder increases dramatically once we are able to confidently rule out a peripheral cause and hence this is the focus of the lecture.

Terminology (symptoms):

Symptom	Description
Dizziness	Sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion. This includes sensations sometimes referred to as giddiness, lightheadedness, or nonspecific dizziness but does not include vertigo.
Presyncope (near faintness)	Sensation of impending loss of consciousness. This sensation may or may not be followed by syncope. When patients report "lightheadedness," it should be classified as presyncope, dizziness, or both.
Syncope (faint)	Transient loss of consciousness due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. Syncope usually leads to loss of postural control and falling.
Vertigo	Sensation of self-motion (of head/body) when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.
Unsteadiness	The feeling of being unstable while seated, standing, or walking without a particular directional preference. This sensation has previously been called disequilibrium or imbalance.

Acute Continuous Vertigo

Vestibular neuritis is the condition caused by acute loss of vestibular function on one side. The incidence is ~3.5 per 100,000 (5). It results in sudden onset vertigo, nausea/vomiting, and unsteadiness which typically resolve after a few days or weeks. Aetiology is thought to be viral with probable reactivation of a latent herpes simplex virus type 1 (HSV-1) infection. Other causes include autoimmune or microvascular ischaemic insults. Patient suffering the acute vertigo often prefer to lie on their non-affected side as this suppresses the nystagmus and vertigo (6). As the continuous vertigo settles, this often progresses to head movement provoked symptoms. These patients often show unsteadiness on testing, usually towards the affected side due to problems within the vestibulospinal tract, however in practice we have not found the direction of imbalance to be a very specific finding.

Although most patients may say they can't walk, in practice they should still be able to remain standing unaided when their feet are apart as they should still have adequate somatosensory and visual input.

Examination in the acute phase often reveals spontaneous nystagmus beating away from the lesioned side. This is due to the fact that the vestibular system has a resting firing rate. As you turn your head to one side, the side you are moving will increase the firing rate and the other side will be inhibited. In the absence of any other input the brain can interpret this as head movement. Due to acute loss of vestibular afferents on one side, this gives the false impression of head movement; hence the vestibular ocular reflex moves the eyes in the opposite direction. The cerebellum corrects this by moving the eyes back quickly. Hence it is the slow phase of eye movement which the vestibular system is responsible for, although typically we document nystagmus in the direction of the fast phase as this is easier to see. So a left beating nystagmus will be due to a right sided lesion in the case of a peripheral vestibular neuritis. As vestibular neuritis more commonly affects the superior division of the vestibular nerve, and hence the horizontal and anterior canals, the nystagmus is usually horizontal with a small torsional component. The acute diagnosis should be further confirmed by the presence of an abnormal head thrust test towards the involved semi-circular canals. This is due to deficit in the vestibular-ocular reflex.

This table shows the main features that can distinguish between peripheral or central causes of acute nystagmus.

Nystagmus	**Peripheral**	**Central**
Spontaneous	Can be suppressed with fixation. May only be visible with fixation removed (e.g. in the dark)	Remains the same or increases with fixation. Nystagmus may be vertical
Gaze-holding	Nystagmus will increase when looking in the direction of the fast phase (Alexander's Law). Regardless of where the eyes are looking, the nystagmus should be beating in one direction.	Nystagmus may change direction with different eye positions

Additional laboratory tests such as caloric irrigation and video head impulse have been developed to objectively quantify the degree of vestibular function. These tests are routinely carried out in our specialist balance clinic. However, if the physical findings in the acute stages are thorough then the use of these tests is not required. Unfortunately, clinicians often fail to document nystagmus properly.

With more advanced vestibular testing available, the presence of superior and inferior vestibular neuritis has now become a recognised phenomenon (7). Superior vestibular neuritis is the most common (55-100%) followed by total involvement (15-30%) and finally isolated inferior vestibular neuritis is seen rarely (3.7-15%). This may be due to the anatomical differences between the superior and inferior vestibular nerves (8).

The following table shows the main clinical tests used to differentiate between inferior and superior vestibular neuritis.

Clinical test	**Healthy Subjects**	**Superior Vestibular Neuritis**	**Inferior Vestibular Neuritis**	**Unilateral Vestibular Loss**
Horizontal Head Thrust Test	Normal	Abnormal	Normal	Abnormal
Head Thrust Test down in the plane of the anterior canal	Normal	Abnormal	Normal	Abnormal

Head Thrust Test up in the plane of the posterior canal	Normal	Normal	Abnormal	Abnormal
oVEMP (tests utricular maculae)	Normal	Abnormal	Normal	Abnormal
cVEMP (tests saccular maculae)	Normal	Normal	Abnormal	Abnormal

Labyrinthitis also presents with acute spontaneous vertigo which usually follows the same course and characteristics of vestibular neuritis except that it includes an ipsilesional change in hearing. As a result, this should be treated with more urgency to exclude any other cause of unilateral hearing loss such as autoimmune disorders or auditory neuroma's. Treatment should consist of steroid for the hearing loss and a short cause of vestibular suppressant medication for the dizziness. Long term use of vestibular suppressant medication has been associated with prolonged recovery and long term side effects so it should be avoided.

Vestibular compensation usually comprises 2 stages

Static Phase	**Dynamic Phase**
Stimulated by static asymmetry	Requires error signal (repeated over time)
Spontaneous nystagmus with fixation present should resolve	Changes in the function of the VOR and/or VSR
Followed by reduction in nystagmus without fixation	Desensitize to movements of the head
Reduction in sensation of falling and improved balance in Romberg position	Vestibular rehabilitation can improve

Long term outcome is not associated with the degree of unilateral vestibular loss. Peripheral vestibular function may or may not spontaneously recover (9). There is not necessarily a difference in outcome but interestingly we can still see signs of vestibular dysfunction on clinical testing many years after the event in asymptomatic people. The recurrence rate is between 2 and 11% (10, 11). As the recurrence rate is low, it is reasonable to treat any true recurrence with caution as it may be something else entirely.

Central causes of Acute Vestibular Syndrome

Central causes such as stroke can often mimic vestibular problems and it can be quite challenging to differentially diagnose. Fortunately simple mnemonics have been created to guide the examination. The presence of any of the following signs: Normal horizontal head impulse test; direction-changing nystagmus in eccentric gaze; a skew deviation (vertical misalignment of the eyes) was 100% sensitive and 96% specific for stroke. This outperformed MRI which was falsely negative in 12% in the first 48 hours (12).

An adaptation on the HINTS (Head Impulse, Nystagmus, Test of Skew) mnemonic is 'SEND HIM ON HOME SAFE'(13). This stands for Straight Eyes (no skew); No Deafness (no new hearing loss); Head Impulse Misses (you should see abnormal head thrust in peripheral disorders but can be normal in central disorders); One-way Nystagmus (direction fixed nystagmus, predominantly horizontal); Healthy Otic and Mastoid Exam (normal tympanic membranes); Stands Alone (able to stand without support); Face Even (no facial palsy or weakness).

Cerebellar Infarctions causing acute vestibular syndrome (14)

Features	**Posterior Inferior Cerebellar Artery**	**Anterior Inferior Cerebellar Artery**	**Superior Cerebellar Artery**
Typical origin	Vertebral artery	Proximal or mid-basilar artery	Distal basilar artery
Core cerebellar syndrome	Isolated acute vestibular syndrome without auditory symptoms (pseudo neuritis)	Isolated acute vestibular syndrome with auditory symptoms (pseudo labyrinthitis)	Acute gait or trunk instability with associated dysarthria, nausea, or vomiting

			(pseudo gastroenteritis)
	Lateral medullary syndrome: hemifacial analgesia, unilateral absent gag reflex, palatal palsy, vocal cord palsy, Horner syndrome, body hemianalgesia, limb hemiataxia, dysmetria	Lateral pontine syndrome: hemifacial sensory loss, facial palsy (lower motor neuron type), Horner syndrome, body hemianalgesia, limb hemiataxia, dysmetria	Lateral midbrain syndrome: fourth nerve palsy, hemifacial sensory loss, Horner syndrome, body hemisensory loss, limb hemiataxia, dysmetria
Neurological signs	Vertebral artery syndrome: 12th nerve palsy, body hemisensory loss, hemiplegia, or quadriplegia	Mid-basilar syndrome: impaired arousal or coma, sixth nerve palsy or internuclear ophthalmoplegia, horizontal gaze palsy, body hemisensory loss, hemiplegia, or quadriplegia	Top of the basilar syndrome: impaired memory or attention, visual field cut, ptosis, third nerve palsy, vertical gaze palsy, hemiplegia, or quadriplegia

Acoustic Neuroma/Vestibular Schwannoma

This usually causes progressive hearing loss on one side (unilateral). Vestibular symptoms such as short spells of vertigo can be associated but less often as the tumour develops slowly and hence allows time for compensation to occur. Balance problems do often develop over time. An audiogram should be arranged to confirm the presence of unilateral sensorineural hearing loss and an MRI IAMs will then confirm the diagnosis. A vestibular schwannoma is a benign tumour of glial cell origin which usually arises from the vestibular branch of the VIIIth nerve. If there is bilateral schwannoma's, a diagnosis of neurofibromatosis Type 2 should be further considered. It is rarely life threatening and in some cases no treatment is indicated. Other conservative options such as vestibular rehabilitation are usually successful as ameliorating any associated vestibular symptoms. Depending on the size and growth, surgery is considered. It is good practice to offer VR soon after surgery to help them compensate. Therefore, as a rule of thumb, asymmetrical hearing loss should be treated as a red flag and the appropriate investigation followed up on.

Episodic Vestibular Syndromes

Benign Paroxysmal Positional Vertigo (BPPV)

This presents with rotational vertigo which usually lasts <1 minute. It is triggered by specific positions such as lying down or head extension. Pitch plane movements are usually worse as the posterior canal is the most commonly affected. It is caused by loose otoconia (canalithiasis) in the semi-circular canals which make the canals sensitive to gravity. Otoconia can also adhere onto the cupula and this is called cupulolithiasis variant. It is the most common cause of episodic vertigo. It affects more females than males and is more common in older adults. It is diagnosed based on the Dix-Hallpike test. Depending on which canal is involved will cause different direction of nystagmus. Posterior canal is the most common followed by horizontal canal. Anterior canal accounts for <1%. Many central disorders present with downbeating positional nystagmus so they need further investigation before reaching a diagnosis of AC-BPPV. BPPV is common following vestibular neuritis and typically affects 10-15% of patients(15). This is thought to be due to utricle damage but relative sparing of posterior canal function.

	Posterior	**Horizontal**	**Anterior**
Nystagmus	Upbeating & torsion towards the affected side	Horizontal	Downbeating & torsion
Canalithiasis	Duration <30-40s	Geotropic nystagmus (towards the ground)	Duration <30-40s
Cupulolithiasis	Duration >60s	Ageotropic (nystagmus towards the ceiling)	Duration >60s

Vestibular Paroxysmia
Presents with spontaneous, recurrent, attacks of vertigo lasting for a few seconds or minutes. It often occurs in stereotyped fashion but is not bought on by a specific trigger. In our clinic we have had a case that presented with an irritative lesion but other times vestibular testing can be normal. In most patients MRI shows neurovascular cross-compression of the VIIIth nerve but this is not necessary to make the diagnosis and is also observed in asymptomatic patients. First treatment of choice is carbazepine, and a good response to this can be used to confirm the clinical diagnosis.

Vestibular Migraine
The presence of dizziness and headache co-occurring can be dated back hundreds of years and prevalence of these two conditions appears to be more that chance alone (16). Vestibular migraine carries a 1% lifetime risk (17) and affects more women than men at a ratio of 3:1 (18). Just like other migraine disorders it is more common is younger people and the mean age is 35 years (19). There are many different names for vestibular migraine you may come across in the literature but for now vestibular migraine is the most commonly used.

Diagnostic Classification (ICHD)(20)

Migraine without Aura	**Migraine with Aura**
A. At least 5 attacks1 fulfilling criteria B-D B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated) C. Headache has at least two of the following characteristics: 1. unilateral location 2. pulsating quality 3. moderate or severe pain intensity 4. aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs) D. During headache at least one of the following: 1. nausea and/or vomiting 2. photophobia and phonophobia E. Not attributed to another disorder	A. At least 2 attacks fulfilling criterion B B. Migraine aura for one of the subforms 1.21-1.2.6. Migraine aura manifests with fully reversible focal neurological symptoms that usually develop gradually over 5-20 minutes and last less than 60 minutes. Headache with the features of migraine without aura usually follows the aura symptoms. Less commonly, headache lacks migrainous features and is completely absent C. Not attributed to another disorder1

Diagnostic Classification of Vestibular Migraine(21)

Definite Vestibular Migraine	**Probable vestibular Migraine**
A. Recurrent episodic vestibular symptoms of at least moderate severity B. Current or previous history of migraine according to ICHD criteria C. One of the following migrainous symptoms during at least 2 vertiginous attacks: migrainous headache, photophobia, phonophobia, visual, or other auras D. Other causes ruled out by appropriate investigations	A. Recurrent episodic vestibular symptoms of at least moderate severity B. One of the following 1. Current or previous history of migraine according to ICHD criteria 2. Migrainous symptoms during ≥ 2 attacks of vertigo 3. Migraine precipitants before vertigo in >50% of attacks: e.g. food triggers, sleep irregularities, hormonal changes 4. Response to migraine medications in >50% of attacks C. Other causes ruled out by appropriate investigations
Vestibular symptoms include vertigo and unsteadiness, which can be spontaneous, positional, or provoked by head or visual motion. Moderate intensity is when symptoms interfere with but do not prohibit daily activities. Severe intensity means that they cannot continue daily activities	

Vertigo is not regularly associated with the headache in ¼ of attacks of VM and 30% of people diagnosed with VM initially present without headache(18). As a result we do not typically consider vertigo to be an aura.

Patients with vestibular migraine can present with an array of neurotological abnormalities, but they are usually subtle. In the acute phase, they can present with both central and peripheral abnormalities (22). Between attacks testing is usually normal, although mild abnormalities in vestibular function testing are common (23). It is also interesting to note that vestibular testing can trigger migraine, suggesting a bidirectional relationship(24). Hearing however is not usually affected.

There is generally limited evidence for the benefit of following strict diet for vestibular migraine. Some of this confusion may arise from the fact that many people experience food craving is the prodromal migraine phase, but this is not necessarily the cause of the migraine. Nevertheless it is useful for some patients who identify a strong relationship with diet.

The risk of stroke has been found to be far higher in people who suffer migraine with aura and smoke, particularly in females. These patients should stop smoking immediately. Other behavioural advice can be useful, including maintaining regular sleep-wake cycles, not skipping meals, regular exercise and reducing stress.

For abortive and prophylactic medications, EFNS and NICE guidelines exist(25). A greater than 50% reduction in attack frequency and severity is considered an excellent therapeutic outcome and a prospective uncontrolled study shows complete resolution or substantial control in 70% of patients (26). Ideally you should allow 6-8 weeks to see the effects, so the drug should not be withdrawn unless in the event of severe side effects. Analgesia overuse can not only lead to analgesia overuse headaches, it can impede the efficacy of prophylactics. Following 6-8 months they should go back to the Doctor to see whether treatment is still requires and consider gradual withdrawal.

Meniere's Disease

Classification (27)

Definite Meniere's Disease	**Probable Meniere's Disease**
1. 2 or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours 2. Audiometrically documented low-to-medium-frequency sensorineural hearing loss in the affected ear on at least 1 occasion before, during, or after one of the episodes of vertigo 3. Fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear 4. Not better accounted for by another vestibular diagnosis	1. 2 or more episodes of vertigo or dizziness, each lasting 20 minutes to 24 hours 2. Fluctuating aural symptoms (hearing, tinnitus, fullness) in the reported ear 3. Not better accounted for by another vestibular diagnosis

Meniere's Disease is perhaps the most overly misdiagnosed condition in neurotology. Nevertheless it is amongst the most common episodic vestibular conditions. It is a clinical disorder and the diagnosis is based on the history and audiology findings. Some patients with meniere's disease report episodes of drop attacks without loss of consciousness, usually preceded by some form of visual tilt or pulsion sensation, so called Tumarkins crisis (the mechanisms are not fully understood).

Vestibular testing findings depend on the stage and severity of the disease. In late MD, testing can show abnormalities ranging from mild to moderate unilateral canal paresis. Head thrust testing is usually positive, except occasionally during an attack. This may be accounted for by hydropic expansion of the lateral canal membranous duct rather than a loss of vestibular receptor hair cell function (28).

As the disease progresses you usually see an asymmetrical sensorineural hearing loss, most commonly in the lower frequencies. Regardless, an MRI is usually carried out in this instance to exclude retrocochlear pathologies such as acoustic neuroma.

Treatments for Meniere's disease usually start conservatively as the disease can often resolve in time and there is a large treatment effect (around 80%) amongst most trials.

The disease can progress bilaterally, with those experiencing symptoms for longer at a higher risk of bilateral involvement.

Perilymphatic Fistula/ Superior Semicircular Canal Dehiscence (SSCD)
Usually presents with attacks of vertigo, dizziness, or imbalance induced by changes of pressure with or without hearing problems. Classic sign is the reproduction of eye movements in the plane of the canal with pressure, low frequency conductive hearing loss with normal acoustic reflexes (bone better than normal), abnormally low VEMP threshold and high resolution CT of temporal bone. Due to opening of superior canal and middle cranial fossa or over communication between perilymph and other structures. Can also affect other canals, but less often. Treatment usually includes rest with sound level control or surgery. The prognosis is usually good and vestibular rehabilitation is rarely used unless in the presence of comorbidity.

Chronic Vestibular Disorders
Bilateral Vestibular Loss
Patients with bilateral vestibular disorders typically do not C/O vertigo. This is because there is vestibular damage on both sides and hence no asymmetrical neural activity to drive vertigo. Instead they suffer from unsteadiness and oscillopsia. Oscillopsia is a sensation where the environment moves when the head moves. This is usually due to dysfunction within the vestibule-ocular reflex, which would act to stabilise your vision under normal conditions. Balance is often worse when other senses are absent, such as in the dark or on uneven surfaces.

There are many different causes of bilateral vestibular loss but the one we see most commonly is due to ototoxicity. Aminoglycoside medication has been found to cause damage to the hair cells in the inner ear, which can be permanent. Gentamicin and Streptomycin are the most vestibulo-toxic. Tobramycin is often the cause of bilateral loss in children as it is commonly used in cystic fibrosis. Fortunately it does not appear to have the same damaging effects when inhaled rather than administered intravenously.

Ototoxicity does not affect everyone and there may be several reasons for this. You are more likely to experience toxicity if you have renal impairment and when used with other antibiotics such as Vancomycin. Several genes may also predispose people, although this is not clinically useful.

Bilateral hair cell loss may never recover and the patient may be left with some permanent degree of disability. Vestibular and balance exercises can improve postural control and substitute strategies for the defunct VOR, although it is unlikely that they will return to normal. Patients with bilateral vestibular loss require advice to manage their condition, such as always having the lights on if getting up in the middle of the night. They should also avoid swimming unaccompanied and should not be allowed to dive under water (because of the lack of visual references for orientation).

Chronic Subjective Dizziness
See (29) for full review.
CSD is not considered to be a primary psychiatric disorder. Whilst anxiety may be predisposing factor, it is not necessary to reach the diagnosis. CSD develops over time and often follows an acute vestibular crisis or other similar medical or psychiatric event causing dizziness. The hallmarks are the relationship to posture, hypersensitivity to motion and visual stimuli. It is also called Persistent Postural Perceptual Dizziness (PPPD). We do not use this term in our clinic due to the close resemblance to another common vestibular condition (BPPV).

Treatment for CSD is primarily vestibular rehabilitation. Additional antidepressant medication (SSRI/SNRI) has also been suggested to add some benefit. Cognitive behavioural therapy may have some use, but in practice this is used for people with additional anxiety or depression.

Many of these conditions co-exist with one another, a thorough history and clinical examination is essential in order to fully account for the patient's condition.

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