GDR Vertige

THE NEWSLETTER

Happy New Year 2022

Unit GDR 2074 N°4 – January-March 2022

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EDITO

Dear clinician friends
THANK YOU!

➢ Michel Lacour
Research Director CNRS

Christian Chabbert, Director of GDR "Vertiges", entrusted me with the responsibility of Editor-in-Chief of the Newsletter N°4, and I thank him for it. This gives me the opportunity to give the floor to my clinician friends, ENT and vestibular therapists, who honoured me in participating in the tribute that was paid to me during the 2021 GDR Symposium organized on the Marseille St Charles campus on September 24th and 25th. They underlined with great kindness, eloquence, and sometimes with a good dose of humour, how much the research of my ex-laboratory had contributed to deepen their knowledge and to improve their professional practice, for a better quality of life of the patients suffering from vestibular deficits. This recognition is worth more than medals! But do not think, Dear Friends, that we have come to the end of the road and that everything is solved. To paraphrase an old Lebanese saying, "if you think you've understood vestibular pathology, it has been poorly explained to you. »

This 4th issue of the GDRV Newsletter focuses on the acute unilateral peripheral vestibular deficit, formerly called “vestibular neuritis”, and its rehabilitation, of which I am giving a historical overview, underlines the modern neurophysiological bases and current ignorance. Shedding light on the clinical picture and the pathophysiology of this vestibular impairment (Michel Toupet), the evaluation and intervention methods (Susan L. Whitney), the practice of rehabilitation by unidirectional rotary stimulation (Alain Thiry), the active dynamics movements (Pierre Miniconi), and gaze stabilization exercises (Michael Schubert) are provided. There is also an interesting explanation for unexplained instabilities (Richard T. Ibitoye and Adolfo Bronstein). The ambition of this 4th issue is that, this time, "if you think you have not understood vestibular rehabilitation, it has been well explained to you".

I hope that this issue will remain digestible, undisturbed by the end-of-year feasts that I wish you festive and happy, and I again thank all contributors for the time they have devoted to it and the immense pleasure that they did to me by accepting this job and, for some, this challenge.

Michel Lacour

AT THE HEADLINE

A year 2022 rich in events

2022 will be rich in events for the GDRV! First of all with the return of the VERTINNOVATION summer school which will be held as usual, the first week of July at the Laboratory of Cognitive Neurosciences UMR7291 CNRS-AMU in Marseille. The “GDRV 2022 Award” is renewed for a second year. Don't hesitate to participate as soon as the Call for Projects opens mid-March. The annual GDRV conference will be devoted this year to “motion sickness”. Information on its organization will be available when registration opens in mid-May. Finally, a new initiative is emerging this year: the "Vertigo Awareness Week" through which our community in its plurality will "make noise" throughout France, echoing the "Balance Awareness Week" organized worldwide by VEDA. You will find all details about these events, as well as the registration procedures on the site http://gdrvertige.com

VESTIBULAR REHABILITATION

What we know and what we don't yet know

Michel Lacour
Research Director
CNRS / Aix-Marseille University

In its narrow sense, "vestibular rehabilitation" covers a set of tools and methods aimed at compensating for deficits resulting from damage to the vestibular system. Its broader meaning encompasses a set of reflexes and behaviors that do not only concern the vestibular system. The rehabilitation of a vestibular patient can be qualified as "vestibular" if one uses methods aimed, for example, at restoring a reflex stabilizing the gaze, such as the vestibulo-ocular reflex (VOR). It departs from stricto sensu vestibular terminology when it comes to re-educating one's postural deficits and one's balance. This is referred to as "postural instability" rehabilitation using methods shared with other pathologies or related to age.

There is no longer any doubt today either about the need for rehabilitative therapy in patients with vestibular syndrome, whether unilateral or bilateral, nor about its effectiveness. The analysis of systematic reviews and meta-analyzes (Cochran data base [1-2]) indicates very clearly that vestibular rehabilitation constitutes an effective and safe therapy improving the compensation of postural, oculomotor and perceptual-cognitive deficits of patients suffering from vestibular pathology, as well as their quality of life.

1. History

The concept of "vestibular rehabilitation" dates back to the period of the Second World War. It is linked to the meeting, in 1941, of Sir Terence Edward Cawthorne (1902-1970), a former student at the "Medical School of King's College Hospital" in London, and of Dr FS Cooksey who, in collaboration with the "Head Injury Center" at Hurstwood Park Hospital had offered rehabilitation exercises for patients with concussions. Cawthorne asked him to develop a rehabilitation program adapted to his vestibular patients.

They established a program to rehabilitate the balance of English soldiers wounded during the Second World War, who presented a concussion syndrome (headache, dizziness, hearing loss, difficulty concentrating) quite similar to vestibular syndrome. In many cases this symptomatology persisted for a year or more with little or no spontaneous recovery.

Cawthorne and Cooksey presented their program on March 1, 1946 to the Royal Society of Medicine [3-4], based on physical exercise, mental exercise, and occupational therapy. The physical exercises began in bed, then in a sitting position, then standing, then walking on a flat surface, and finally on rougher floors as the patients progressed, slowly and then faster.

The initial head and shoulder relaxation movements were followed by head movements with fixation of near or far targets, then with closed eyes to re-educate the sense of position. The progression introduced climbing and descending stairs and catching and throwing ball games in different positions. Figure 1 illustrates the original exercises offered to patients at that time.

Cawthorne and Cooksey's consideration of contextual, emotional, and psychological factors constitutes a very modern view of present-day vestibular rehabilitation. All or almost all of the current conceptions are embedded in their recommendations, which nothing, however, supported. Including their intuition that a program applied early seemed more effective.

2. Recent advances

The major advances in Neuroscience in the second half of the 20th century brought to light the great plasticity of the adult brain. The work of Michael Merzenich's group demonstrated that from the day it was born until the day it dies, the brain continually remodels, improves or deteriorates depending on how it is used. These observations are based on the concept of Hebbian plasticity which gives the environment and subject-environment interactions a capital role in the reorganization of nerve connections, experience-dependent in essence. Their recent article is an ode to a rehabilitation therapy based on brain plasticity [6].

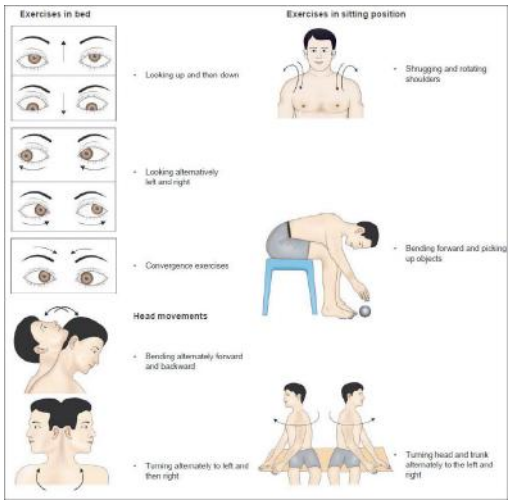

Figure 1: Exercises in the training program rehabilitation of Cawthorne and Cooksey. *Progression of exercises (eye, head and body movements) (from [5]).*

CONCEPT 1

Neuronal plasticity after vestibular deficit is dependent on post-injury time

« The earlier the better »

Critical period

The molecular and cellular plasticity mechanisms promoting synaptic reorganizations at the peripheral and / or central levels are re-expressed in the very 1st days / weeks after vestibular loss.

CONCEPT 2

Synaptic reorganizations after vestibular loss depend on neuronal activity

« Use or lose neuronal plasticity»

Hebian plasticity

Neuronal reorganizations are guided by the post-injury experience. They are context-dependent and favored by sensorimotor activity.

Figure 2: Two key concepts for Vestibular Rehabilitation. *There is an early critical period during which rehabilitation orients neuronal plasticity.*

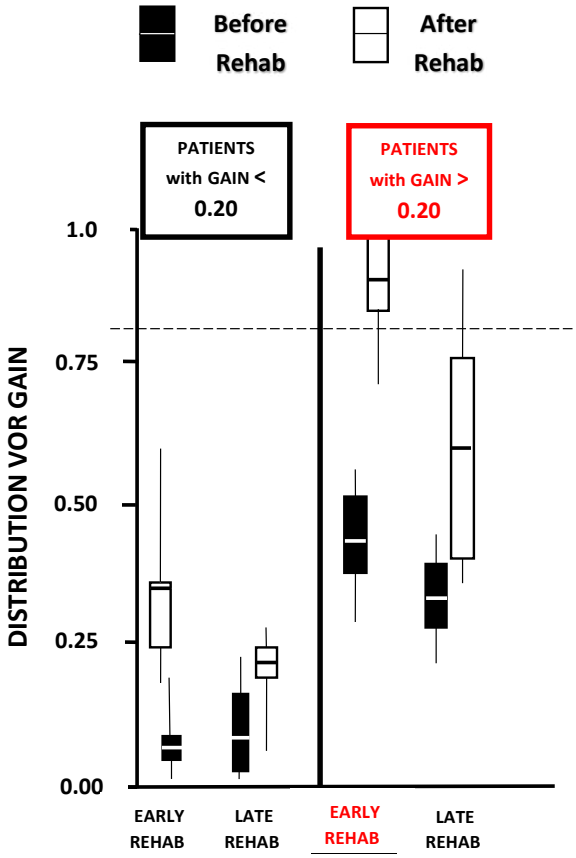

Figure 3: Two conditions to restore normal VOR
Only patients who have undergone early vestibular rehabilitation (first two weeks after the vertigo attack) and having a VOR gain greater than 0.20 on the hypofunctional side recover normal canal dynamics. Saccadic substitution is the behavioral strategy involved in those who do not meet these two conditions [10].

Compensation for static deficits (spontaneous nystagmus, head/trunk tilt and subjective visual vertical deviation on the injured side) results from the restoration of electrophysiological homeostasis of homologous vestibular nuclei. Vestibular rehabilitation accelerates this natural process of compensation [7]. The same is not true of the restoration of dynamic vestibular functions, which is generally poor, on which vestibular rehabilitation has a major effect.

An illustration of the capital role of rehabilitation is provided by the restoration of the gaze stabilization. The vestibulo-ocular reflex (VOR) is the main actor for rapid movements of the head (> 100 ° / s). Few of the patients with unilateral vestibular hypofunction recover a normal VOR, the majority using eye saccades as a backup strategy [8-9].

Our recent work carried out jointly with Dr Laurent Tardivet (ENT) and Alain Thiry (physiotherapist) demonstrated that the VOR could be completely restored under two conditions: 1) that rehabilitation is undertaken very soon after the onset of the vertigo crisis (critical period) and 2) that the initial gain of the VOR is greater than 0.20, leaving a sufficient number of nerve fibers intact for synaptic reorganizations to take place and become more refined with rehabilitation (Hebbian plasticity: cf [10]).

These data constitute the first demonstration in the vestibular patient of a critical period and of the role of the degree of peripheral vestibular loss in the restoration of dynamic vestibular functions.

Message to clinicians

Have a correct and precise etiological diagnosis very early on to undertake very early rehabilitation capable of restoring the dynamic functions of gaze stabilization and balance. Close coordination between general practitioner, ENT and physiotherapist is essential for optimal patient care…

3. What we do not know

3.1 Which rehabilitation protocol?

Among the latest recommendations of the American Association of Physiotherapists published in 2017, prominently is the one on the need to compare different programs, different protocols and different types of exercise in order to determine the optimal approach to treat vestibular patients.

Our own work on the recovery of normal canal dynamics has shown that the paradigm of passive unidirectional rotation in the horizontal plane (chair protocol of French vestibular physiotherapists) is only effective for the lateral semicircular canal. This type of rehabilitation exercise has no effect on the dynamics of the vertical canals. In contrast, active gaze stabilization exercises in the horizontal, LARP and LARP planes are effective for all 3 pairs of canals [11]. In order to restore the dynamics of the vertical channels, rotatory chair rehabilitation would therefore require placing the patients' heads in their stimulation plane, which is not always very comfortable for a patient in the acute phase.

Protocols vary depending on the equipment of practitioners, countries and schools of thought. It is becoming urgent to set up large clinical trials aimed at comparing these different approaches. The introduction of virtual reality in rehabilitation is one example, among many, of what needs to be carefully validated or not to avoid the trends and the silly rush on everything new technology.

3.2 For which vestibular patient?

Compensation mechanisms and their kinetics vary according to the type of vestibular lesion [12]. The sudden and rapid deficit of vestibular neuritis causes a cascade of molecular and cellular events in the vestibular nuclei, in no way commensurate with the very progressive deficit of a neuroma that progresses so slowly that it sometimes remains asymptomatic. Recovery after partial damage has a better prognosis than complete damage (see the crucial role of the degree of hypofunction on the recovery of VOR). The recurring crises of Ménière influence the pattern of recovery and the treatment of a BPPV has nothing in common with other vestibular pathologies. The protocols must be adapted to the pathology, and research remains to be undertaken to determine the best protocol/patient suitability.

Vestibular compensation is also patient-dependent. The existence of different frames of reference depending on the patient [13], allocentric (vision) versus egocentric (proprioception), suggests the use of rehabilitation protocols adapted to these idiosyncratic strategic differences. The age of the patients, their psychological and emotional profile (anxiety, stress) are other factors to be considered in future research [14].

3.3 When to stop rehabilitation?

This is an important question that currently has few satisfactory answers as it depends on so many parameters. Studies must systematically analyze the nature of rehabilitation exercises, their duration and frequency, their degree of difficulty and the pattern of progression to be followed.

The concept of return to work should be investigated on the basis of qualitative and quantitative criteria. Is a change of professional activity to be considered? When to resume driving? Etc… The optimal duration of vestibular rehabilitation must be determined on the basis of favorable results. What are they ? Are these subjective scales of quality of life? Or objective measurements based on gaze or posture stabilization performance?

In summary: There is currently no established consensus either on the protocols, or on their adaptation to vestibular pathology and patient specifics, or on the criteria for successful rehabilitation. Everything has to be done.

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REHABILITATION OF AN ACUTE UNILATERAL VESTIBULAR DEFICIT (AUVD)

Vestibulo-spinal rehabilitation (VSR) by active dynamic movements (ADM)

Pierre Miniconi
ENT, Carpentras

1. Objective

It is accepted that in the event of AUVD, vestibular rehabilitation, started as early as possible by the vestibular physiotherapist, can restore the patient's balance with disappearance of symptoms and secondary signs of this deficit.

We offer an original vestibular rehabilitation technique (vestibulo-spinal rehabilitation) to be done immediately after the diagnosis of AUVD, in the practitioner's office and which can be continued by the patient himself at home allowing to wait until the appointment of the physiotherapist.

These active dynamic movements performed by the patient in the 3 planes of the canal cupulas and otolithic maculas, horizontally, in RALP and LARP, cause a postural reaction (vestibulo-spinal reflex).

This first session of VSR by ADM, after having fully explained the origin of the disorders and reassured the patient, makes it possible in ¼ h to make the nausea / vomiting disappear and to regain a dynamic postural balance allowing him to move alone.

These ADMs would stimulate the somesthetic sensors (exteroceptive and proprioceptive) and the remaining vestibular sensors (ductal and otolithic) thus allowing a rapid compensation of the AUVD.

This otolithic stimulation during ADM would be due to the centrifugal force (linear acceleration detected by the macules) present during any angular acceleration detected by the cupulas.

2. Active Dynamic Movements

2.1 First series of exercises (Fig. 1)

First exercise

1. Standing, feet together, eyes open (1),
2. Turn your head to one side, with as much amplitude as possible and as quickly as possible (2),
3. Wait 3 to 5 seconds: becoming aware of the sensation in the soles of the feet, toes and ankles until you feel stable,
4. Turn your head quickly to the other side without stopping in the middle (3),
5. Wait in the same way

Repeat the exercise 5 times,
Then do it with your eyes closed 10 times.

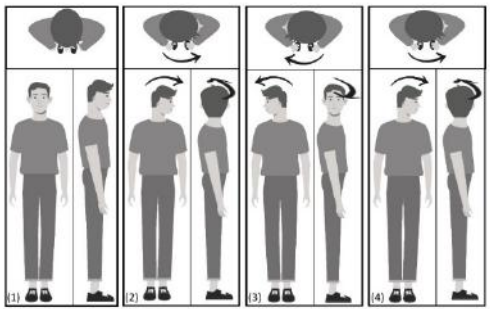


Figure 1: First exercise : *stimulation of lateral canals and utricular macules*

Second exercise (Fig. 2)

1. Standing, feet together, eyes open (1),
2. Quickly flex the hip by 90°, keeping your head in line with the trunk (2). **Watch out for the antero-pulsion,**
3. Wait 3 to 5 seconds, in the same way as described previously,
4. Straighten up quickly, keeping your head straight (3). **Beware of back-propulsion,**
5. Wait in the same way,

Repeat the exercise 5 times,
Then eyes closed (10 times).

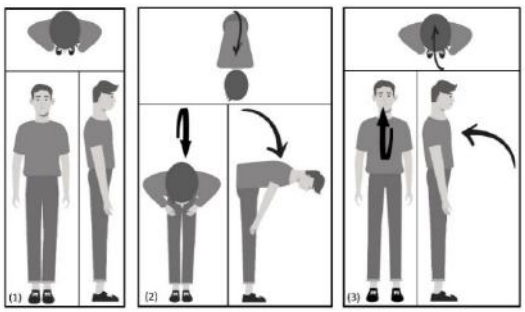


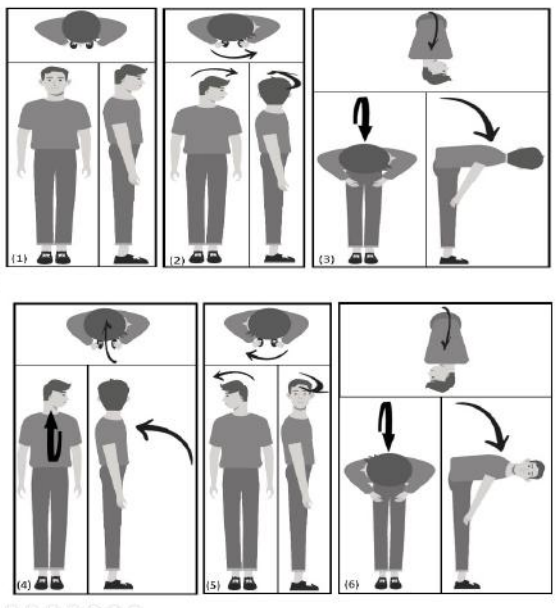
Figure 2: Second exercise : *stimulation of vertical canals, saccular macules and dorsolumbar proprioception.*

The new VNG-VCOR (Framiral, Grasse, France) has shown a very rapid participation of cervical proprioception in the case of AUVD with symmetryzation of the VCOR gain in horizontal and vertical in a few days, while the preponderance in VOR persists, thus allowing an early compensation during the natural movements of the head, with disappearance of the instability even if the spontaneous nystagmus persists.

Third exercise (Fig. 3).
To be carried out only if the first two are mastered!

1. After turning your head to one side and waiting long enough (2), keeping your head turned to the side, quickly lean forward with a hip flexion (3). **Watch out for the antepulsion,**
2. Wait as indicated above,
3. Straighten up, keeping your head turned in the same position (4). **Beware of back-propulsion,**
4. Turn your head to the other side without stopping in the middle (5), then lean forward as for the other side (6),
5. Straighten up quickly in the same way as on the other side (7).

Repeat the exercise 5 times with eyes open and then 10 times with eyes closed.



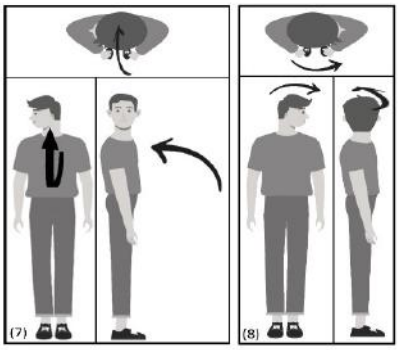


Figure 3: Third exercise

Stimulation of 6 channels, 4 macules and cervical and lumbar proprioception.

2.2. Second series of exercises

Same exercise as the first one (rapid rotation of the head in one direction, stop for 5 sec, then rapid rotation in the opposite direction but in FUKUDA with eyes open then eyes closed.

2.3. Third series of exercises

Standing with the feet a little apart, eyes open, rapid rotation in the horizontal plane of the head and the trunk with the greatest possible amplitude, stop 5 sec, then rapid rotation in the opposite direction, we repeat this movement 5 times then 10 times with the eyes closed.

Obviously, if AUVD is examined in the first few days, it will be impossible for the patient to perform these movements correctly as described above.

He should stand in the back of a chair and do them slowly at first, then increase their range and speed gradually. Usually there is vomiting at first, then vegetative complaints disappear as dynamic postural balance improves.

Usually, at the end of the session, the patient can move around alone, without support, and especially without neurovegetative symptoms.

After a few days the balance disturbances completely disappeared as well as the autonomic symptoms.

Attention :

In the event of acute unilateral vestibular deficit of the superior contingent, or total, flexion in the plane of the damaged anterior canal can cause antero-pulsion with fall.

In acute unilateral vestibular deficit of the inferior contingent, or total, extension in the plane of the injured posterior canal and saccule can cause retropulsion with fall.

Acknowledgements

I would like to thank Dr Lei Tanaka for his contribution to writing this article and making the diagrams.

The videos are available from Pierre Miniconi

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GDR Vertige

Award 2022

- As in 2021, two projects will be awarded « Annual GDRV Award" to support their fundraising efforts with national and international organizations and foundations
- Grants of €1,000 and €500 will be awarded to the two projects selected by the GDRV committee of experts

Call for Projects
March 15th 2022

- For practical information: see section "GDRV 2022 price" on http://gdrvertige.com

GAZE STABILIZATION EXERCICES

The Role of Gaze Stability Training in Vestibular Hypofunction

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1. Background

Gaze stability refers to the eyes remaining stable in space during a head motion, which is one of the primary functions of the peripheral vestibular end organ.

Gaze stability exercises are now recognized as essential treatment for patients with vestibular hypofunction, with clinical practice guidelines available to guide clinicians in their prescription [1]. In theory, the purpose of these exercises is to improve the vestibulo-ocular reflex (VOR) by exposing patients to retinal slip though it remains uncertain if such exercises change the VOR [2]. In fact, recent evidence suggests some of the exercises do not change the VOR but instead lead to recruitment of an eye movement that substitutes for the deficient VOR [3] or the exercises leads to a reduction in dizziness through the mechanism of habituation (i.e. exposure therapy [4]).

Retinal slip occurs when the image of an object moves off the fovea of the retina, resulting in visual blurring. Retinal slip is one of the primary and essential signals used to drive change in magnitude of the VOR within the brain – a form of motor learning of the VOR [5]. Because the brain can tolerate small amounts of retinal slip yet see a target clearly, the patient should be instructed to keep the target in focus during head motion [6]. Otherwise, if the retinal slip is excessive then the VOR does not retain the intended motor learning [7].

2. Exercises

The four primary exercises prescribed as part of a gaze stability program are the ×1 (times 1), ×2 (times 2), gaze shift, and imaginary target exercises. For the ×1 exercise, the patient is asked to move their head horizontally (and separately in the vertical direction) as quickly as possible while maintaining focus on a stable target (Figure 1).

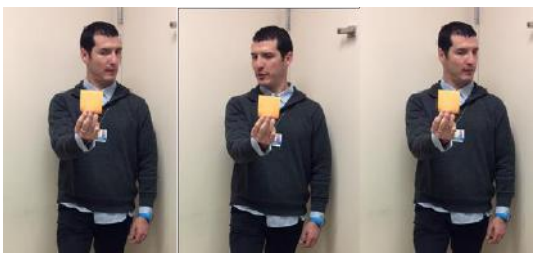

Figure 1. Times 1 exercise for horizontal head rotation

The patient must learn to slow the head movement if the target becomes blurred. A good target to use is a business card, asking the patient to focus on a word or a letter within a word. The target distance can be near or far. For the ×2 exercise, the patient is asked to move their head and target in opposite directions, while keeping eyes fixated on the target (Figure 2).

Figure 2. Times 2 exercise for vertical head rotation

The gaze shift exercise involves placing two targets on a wall in front of the patient. The patient is instructed to first make an eye rotation towards one target, followed by a head rotation towards the same target – all while keeping the eyes on that initial target. They then continue the exercise (horizontal and separately in the vertical direction) by 1st making an eye rotation followed by a head rotation towards the target (Figure 3). Finally, the imaginary target exercise teaches patients to first focus on a target placed on the wall in front of them. Next, the subject is instructed to close their eyes and imagine they are still looking at the target as they make a head rotation away from the center position. Once the head stops moving, patients are instructed to open their eyes and check if the eyes remained on target or fell away from the target (Figure 4).

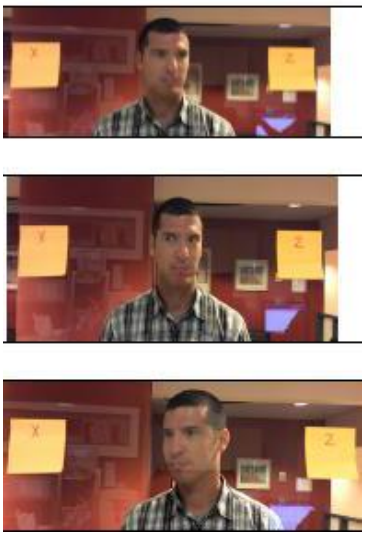

Figure 3. Gaze Shift Exercise for horizontal head rotation

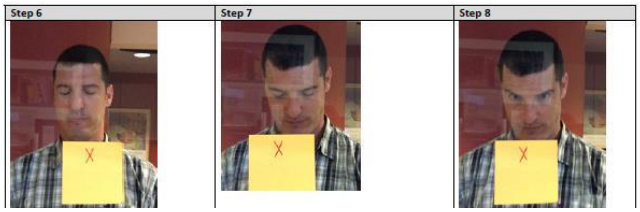

Figure 4. Imaginary Target Exercise for vertical head rotation

Each of the four exercises should be made increasingly more difficult as the patient improves. Examples of increasing difficulty include the use of a distracting background (checkerboard, venetian blinds) while the patient attempts to focus on the letter or word, varying the distance from which the patient performs the exercises, moving the head more rapidly, and performing the exercise while standing or walking.

3. Guidelines and Recommendations

As mentioned earlier, clinical practice guidelines (CPG) are now available that specify the frequency that gaze stability exercises should be prescribed. The CPG are

based in part on Cochrane Database Systematic Reviews concluding that there is moderate to strong evidence in support of vestibular rehabilitation in the management of patients with unilateral (UVH) and bilateral vestibular hypofunction (BVH), specifically for reducing symptoms and improving gaze and postural stability [8-10].

In summary, the CPG recommends supervised vestibular rehabilitation sessions for the patient to understand the goals of the program and how to manage and progress themselves independently.

As a general guide, persons without significant comorbidities and an acute UVH may need once/week supervised session for 2 to 3 weeks, while persons with chronic UVH may need once/week sessions for 4 to 6 weeks. For patients with BVH, it is recommended that rehabilitation should be once/week sessions for 8 to 12 weeks. All patients should be instructed to perform daily home exercises. For detailed information, please see [1].

Recently, we suggested a novel gaze stability exercise based on the well-known vergence effect on the VOR. When looking at a target that is ~15cm from the bridge of the nose and making a head rotation, the VOR can be increased as much as 25% {11], which occurs because the eyes must translate as well as rotate when viewing near targets. This enhancement of the VOR persists for contralesional head rotations in UVH and we believe should be prescribed as an exercise [12].

Clinicians should stay tuned to learn more about the mechanisms supporting the effectiveness of vestibular rehabilitation. For now, it is known that gaze stability training improves visual acuity during head motion as well as gait stability and subjective perception [2, 13-15].

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VESTIBULAR NEURITIS

I. Clinical presentation of the

“ Vestibular neuritis”

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1. Definition

What has been called "vestibular neuritis" is a benign condition. The initial severe phase of vertigo usually lasts between two and three days, but it can last a week or more. The average illness is six weeks, with durations of up to nine weeks or more.

The signs and symptoms of characteristic "vestibular neuritis" are sudden, intense, prolonged dizziness for several days in the absence of auditory symptoms (deafness, fullness, tinnitus) and other neurological symptoms (Headache, diplopia or dysarthria) [1, 2, 3, 4].

2. Clinical presentation

The typical presentation of the "vestibular neuritis" is that of a viral attack by shingles: zoster oticus. In addition to the great rotatory dizziness, the patient may present with a painful rash of vesicles in the territory of the VII bis typical of shingles (external auditory canal and tragus), deafness and facial paralysis on the same side. Clinical examination finds harmonious vestibular syndrome (the model of its kind): deviation of gait (Fig. 1), Romberg's test and index fingers on the affected side, and horizonto-rotatory nystagmus on the healthy side. The one-sided caloric deficit has long been the only yardstick for certainty. The Halmagyi test and its extension, the Head Impulse Test (V-HIT), confirm the unilateral vestibular deficit. The MRI shows contrast enhancement of the cochleo-vestibular nerve with a flaming image (not the rounded image of a vestibular schwannoma). It’s not that common.

Much more often there is an acute vestibular syndrome (Acute Unilateral Vestibulopathy) without facial paralysis, without deafness, without rash, without a typical picture of neuritis on MRI. But much better at the MRI 3T, four hours after gadolinium injection, a picture of labyrinthitis most often involving the horizontal semicircular canal, the superior and the utricle is observed [4, 6].

The posterior canal and saccule are not often affected, as is the cochlea. The viral background is not obvious. It is not uncommon for this clinical presentation to be seen in the aftermath of a vaccination showing the part of the post-viral inflammation to be greater than the virus itself. In half of the cases [5], these patients will present with signs of benign paroxysmal positional vertigo on the same side (Lindsay-Hemenway syndrome)

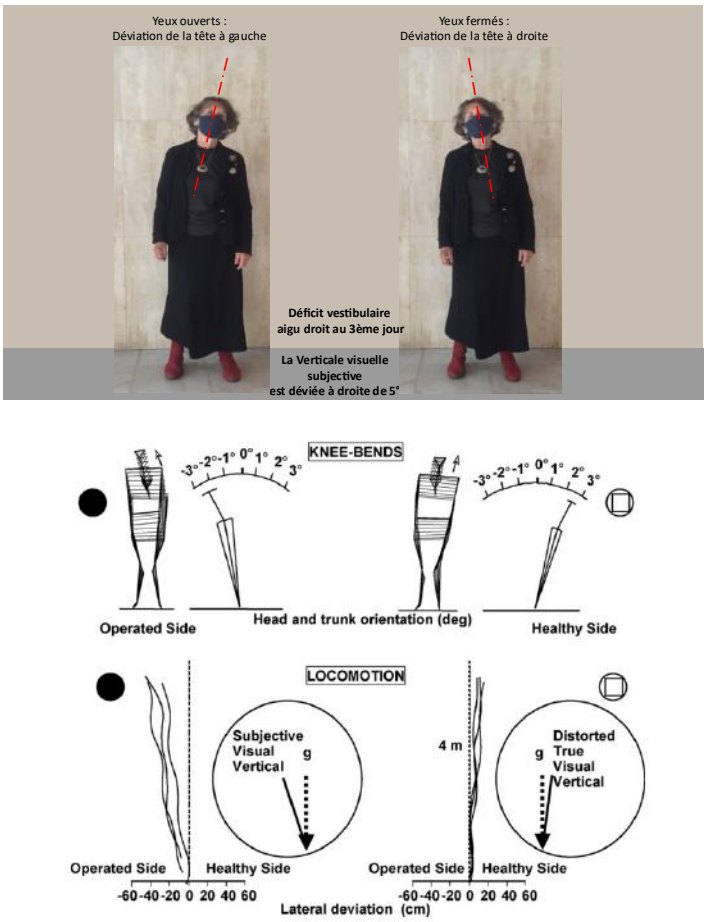

Figure 1 : Posturo-locomotor syndrome after acute unilateral vestibular deficit.

Top panel: With eyes closed, the patient raises his head, sometimes frankly deviated towards the deficit. Paradoxically, in the first days after the onset of the acute vestibular deficit, with the eyes open, the patient tilts his head to the side opposite the deficit. Probably to straighten the subjective visual vertical deviated from the side of the deficit.

Bottom panel: As early as 2008, Borel L, Lopez C, Péruch P and Lacour M. observed this reversal of deficits with or without vision just after unilateral vestibular neurotomy in patients operated on for disabling Menière's disease [11].

A third clinical tableau of acute vertigo reflects another labyrinthitis: that of the posterior semicircular canal, a little rarer than the previous one (4% of acute vestibulopathies [8]), but it undoubtedly has the same etiology. The MRI 3T four hours after injection shows contrast enhancement of the posterior canal [4]. Spontaneous nystagmus is torsional, caloric strain is normal, and body deviation as well. Clinical examination shows that movement of the head in the plane of the posterior semicircular canal on one side (up and back to the side) gives a brief dizziness (one second) and a short loss of balance (sign of Pierre Miniconni), is confirmed by Video Head Impulse Test (VHIT) and MRI 3T four hours after Gadolinium.

These two examinations reveal the same cause: the posterior semicircular canal on one side: "Inferior Vestibular Neuritis" [8]. A few years ago, peduncle involvement would have been claimed in such a clinical picture [9, 10].

The saccular and utricular otolithic evoked potentials have come to provide an essential complement today in the more precise diagnosis of acute vertigo.

The study of Subjective Visual Vertical [5], Dynamic Visual Acuity and multisensory posturography shed new light on the patient experience.

3T imaging four hours after gadolinium confirms the extent and etiology of the possibly inflammatory lesions.

WARNING !

The trap in a presentation of acute vertigo is to forget the possibility of central involvement: that of the vestibular nuclei (the historical model of which is Wallenberg syndrome), or of the hypoglossus, or the cerebellar peduncles. The clinical tableau is very close at first.

The key moment of the clinical examination is the demonstration of a gaze nystagmus, which never exists in peripheral involvement. In any case, with classical imagery, it will be necessary to look carefully at the periphery of the 4th ventricle [9,10].

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REHABILITATION OF AN ACUTE UNILATERAL VESTIBULAR DEFICIT

Rehabilitation using the rotatory chair

Alain Thiry
Physiotherapist - Nice

1. Reminder

Often described as the "French" technique, used since the beginning of Vestibular Rehabilitation in French-speaking European countries, the rotary chair rehabilitation was described by Alain Semont in the 1980s. It remains today one of the privileged tools for the rehabilitation of an acute unilateral peripheral vestibular deficit, in the same way as the exercises for stabilizing the gaze and posture, dynamic visual acuity, etc.

2. Objectives

2.1 Principle

The principle of chair rehabilitation is to produce unidirectional rotational stimulation in order to reduce vestibular asymmetries. By performing repeated and high speed rotations towards the hypofunctional side, one produces a habituation of the intact labyrinth which sees its response gradually decrease from one trial to another and from one session to another, while stimulating the hypofunctional labyrinth, to promote plasticity mechanisms involved in vestibular compensation. This results in a reduction in the slow phase velocity of spontaneous nystagmus and asymmetries such as directional preponderance [1-3].

2.1 Method

To reduce responses on the intact side and "boost" those on the hypofunctional side, the patient is subjected to high-speed unidirectional rotations (> 150°/sec) on the pathological side followed by abrupt stopping of the chair. The number of chair rotations during the same test varies depending on whether the patient tolerates the stimulation more or less well: three, five, seven or more turns depending on the autonomic feeling. The protocol must be adapted to each patient to avoid paleness, nausea and vomiting. Personally, I use rotational speeds not exceeding 250°/sec which, in the most cases, are very well tolerated.

The chair must be equipped with handles, a headrest, footrests and have a reduced impasto for high speeds. The patient's position must be strictly controlled. The peripheral vestibular system should be located in the axis of rotation of the chair, and the head tilted approximately 30° down to bring the horizontal canals into the horizontal plane (Fig. 1). Stimulation of the vertical canals (LARP or LARP planes) requires other head positions which may be uncomfortable in an acute patient.

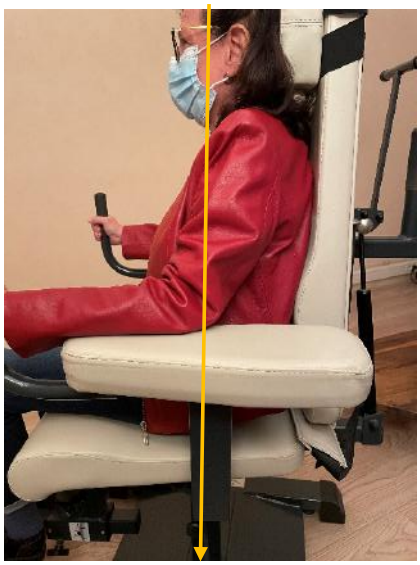

Figure 1: Patient position

Figure 2: Practitioner position

The practitioner will be positioned to the side, slightly to the front, to control the appearance of neuro-vegetative signs (Fig. 2). In a healthy subject, during a clockwise rotation, the ampullopetic endolymphatic current in the right canal, excitatory (Ewald's second law), creates the appearance of a per-rotatory physiological nystagmus, the slow phase of which follows the movement of the cupulla (left), and saccades beat on the side of the rotation (right). The opposite phenomenon is observed with a counterclockwise rotation. Suddenly stopping the chair triggers physiological left post-rotatory nystagmus (saccades) after clockwise rotatory stimulation, and right (saccades) after counterclockwise rotary stimulation.

After total unilateral vestibular loss, only post-rotatory nystagmus is observed during rotations on the pathological side.

2.3 What do we measure ?

The patient can be fitted with a wireless videonystagmography / graphing system that provides access to the slow phase speed of per- and post-rotational nystagmus and their time constant, measured by the time taken to return to the one third of the initial slow phase eye velocity value. In the fixation test, the patient has his eyes closed during the rotation, and he opens them when the chair suddenly comes to a stop. He must then stare at a target 1.5 meters in front of him, in line with his gaze. Post-rotational nystagmus gives the illusion of a moving target, and is asked to say "stop" when the illusion of the moving target ceases. The time is measured manually with the stopwatch from the patient's subjective stop.

During the vection test, the patient keeps his eyes closed when the chair is stationary. He then has an illusory sensation of body/chair movement in the opposite direction to the initial rotation performed on the hypofunctional side. We measure in the same way the time taken for this illusory sensation of displacement in the opposite direction to cease.

The repetition of unidirectional rotatory stimuli during the same session, then during successive sessions, shows a symmetrization of the vestibular responses which results in 1) a decrease in the slow phase of spontaneous nystagmus (reduction of the nystagmic preponderance and reorientation right in front), 2) habituation of the response on the intact side (reduction of the time constant of post-rotatory nystagmus), and a functional restoration of the vestibular response on the hypofunctional side (which depends on the precocity of the rehabilitation and on the degree of the initial vestibular hypofunction [3]).

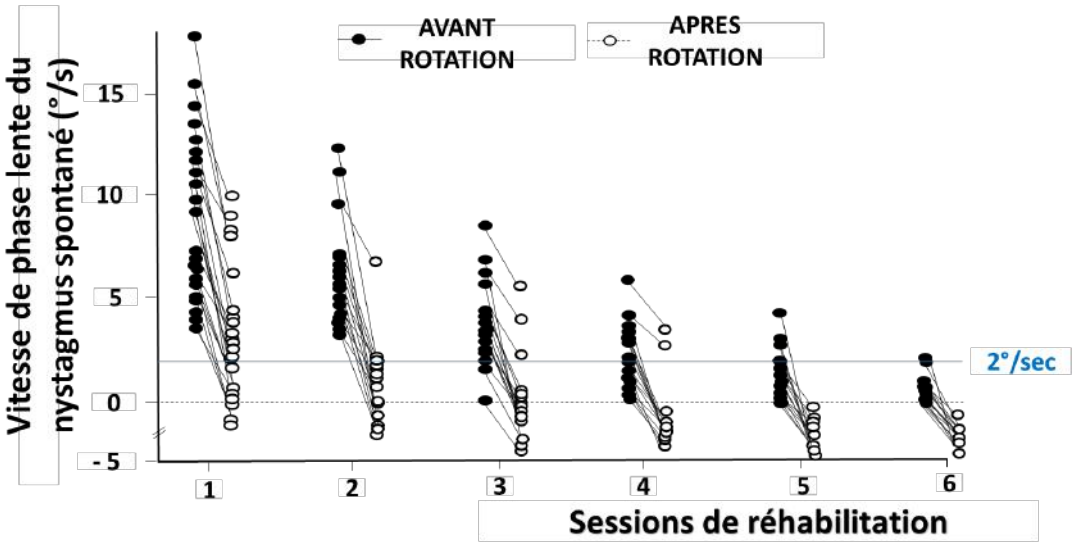

Figure 3: Slow phase eye velocity changes of spontaneous dark nystagmus by unidirectional rotational stimulation.

The slow phase speed of nystagmus is measured before and after each rehabilitation session performed in the rotatory chair (5 turns at 250°/s, hypofunctional side, repeated 5 to 10 times per session depending on patient tolerance). Average results obtained in a population of 22 patients with acute unilateral vestibular impairment, rehabilitated during the first two weeks after the onset of the vertiginous crisis. Each patient sees the slow phase eye velocity of spontaneous nystagmus reduced after rotational stimulation, which sometimes reverses, and after 6 rehabilitation sessions all patients return to non-pathological slow phase rates (<2°/s).

Figure 3 illustrates the changes in the slow phase velocity of spontaneous nystagmus measured in the dark during the first 6 sessions in a population of 22 patients. We have shown that, along with the reduction in vestibular asymmetries, unidirectional rotational stimulation reduced the directional preponderance measured by the ratio of vestibulo-ocular reflex gains on the hypofunctional and the intact side [1].

3. Conclusions

The field of predilection for rehabilitation with the rotatory chair is the acute unilateral vestibular deficit, but this method also finds its application in the treatment of motion sickness, post-traumatic or post-operative pathologies (neuroma), fluctuating (Menière), or hypervestibulia.

Direction of rotation and speed should be adjusted according to the pathology.

Rotatory chair work can be combined in the same session with eye fixation exercises, with or without active high-speed head movements, or gaze stabilization. The dynamic active exercises recommended by Pierre Miniconi will be presented to the patient as soon as possible and can be performed at home.

While it is not the exclusive tool for rehabilitation, the rotatory chair remains an essential method for accelerating and optimizing vestibular compensation. On condition of respecting the rules of its use.

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VESTIBULAR NEURITIS

II. Pathophysiology of " Vestibular neuritis"

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1. Suspected etiopathogenesis

Since the first description of a case of vestibular neuritis by Ruttin in 1909 [1], then the specification of the diagnosis by Hallpike in 1949 [2], Schuknecht [3] in 1985, then Nadol [4] in 1995, from post-mortem histological sections, especially showing degeneration of the superior component of the vestibular nerve, vestibular neuroepithelium and vestibular ganglion of Scarpa [5] with depopulation of nerve fibers [6], and with optical and electron microscopes a degeneration of myelin, the concept of neuritis seemed established.

However, recently Michaël Eliezer [7] shaked things up through confirming the already suspected labyrinthitis. Spectacular turn of events, but what about the etiology?

Vestibular neuritis is not a neuritis, and it probably is not viral

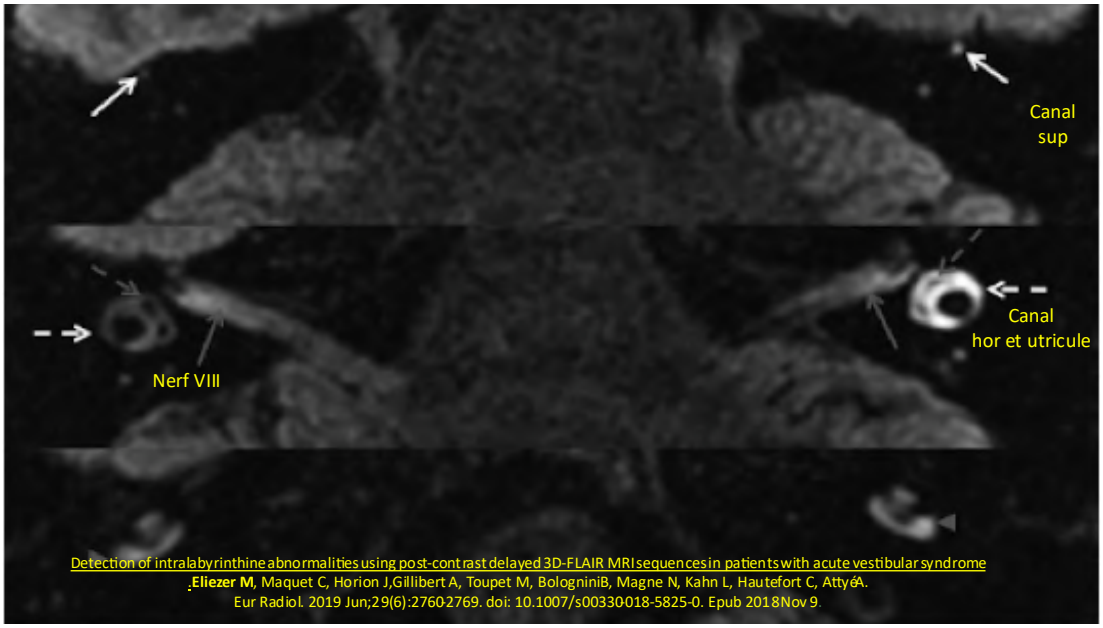
1.1 Internal auditory artery occlusion is not confirmed

Only the famous and early study (1958) by Lindsay and Hemenway showed thrombosis of a large vessel: the internal auditory artery [8], but the vascular occlusion was not - found in the four other cases studied and published by Schuknecht and Kitamura [9].

1.2 Antiviral agents did not improve results

Several studies have shown serological evidence of recent viral infections of the upper respiratory tract, particularly those caused by influenza A viruses, influenza type B viruses, and adenoviruses, as well as infections caused by the herpes simplex virus, cytomegalovirus, Epstein-Barr virus, rubella virus and parainfluenza virus [10, 11].

Despite clear serological evidence of a recent viral infection, no virus has been isolated from the blood, saliva, respiratory tract or cerebrospinal fluid of patients, despite repeated attempts [11]. There is growing evidence for an inflammatory component; this could be the result of viral infection in the progression and etiology of this disease [12]. Patients treated with corticosteroids had better clinical outcomes compared to placebo patients. However, in isolation, virostatic drugs did not improve symptoms [13, 14].



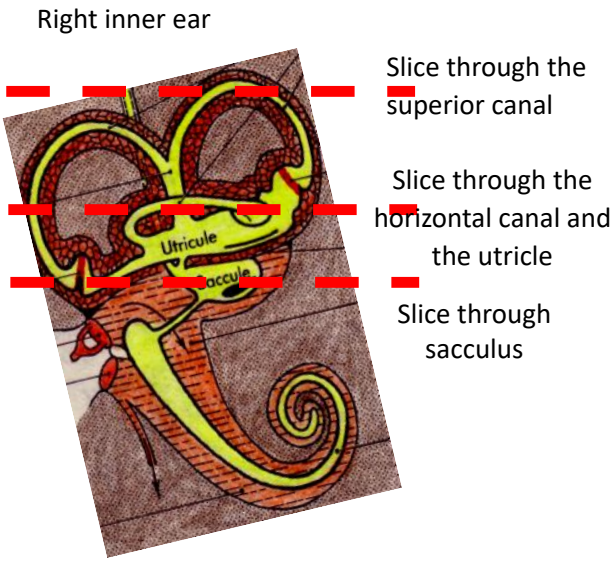


Figure 1 : MRI imaging of a "vestibular neuritis" patient

Top panel: 3T Flair MRI 4 h after intravenous injection of gadolinium. Contrast enhancement of the superior semicircular canal, horizontal, and utricle is the most common signature of acute vestibular deficits (Eliezer et al, 2018). The two vestibular nerves have the same contrast.

The name neuritis is no longer justified.

1.3 The immune-mediated mechanisms seem to be the most current etiology [15].

Biological dosages vary around the world according to the teams. Micro-thrombosis and vasculitis are under study.

1.4 Surprise: reappearance of the forgotten vestibular atelectasis

Merchant and Schuknecht in 1998 [16], then Nadol in 1995 [4] in Boston, thanks to their immense collection of post-mortem histological sections of vertigo patients were able to show that acute vertigo (often followed by other vertiginous episodes) could be vestibular atelectasis (a collapse of the endolymph and presumably of the sensory epithelium). Today Michaël Eliezer and his team confirm in MRI3T, four hours after gadolinium, this image of atelectasis. This is another intriguing etiological lead. Should we link it to one of the three viral, vascular or immunological hypotheses? Or is it the same? Is it still something else?

All these considerations do not change our early management of vertigo patients diagnosed with neuritis or labyrinthitis. Treatment with corticosteroids and vestibular rehabilitation [17] are currently our best therapeutic solutions.

Conclusion

Although the etiology remains unclear, it appears that vestibular "neuritis" could be an autoimmune disease, but the other possibilities: viral infections and vascular disorders are still debatable

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VESTIBULAR REHABILITATION EXERCISES

Evaluation and interventions for persons with vestibular hypofunction

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1. Evaluation

Persons referred with the diagnosis of vestibular hypofunction generally have a good prognosis [1]. Vestibular hypofunction includes diagnoses such as vestibular neuritis, labyrinthitis, and Meniere's disease. Bilateral vestibular hypofunction results in poorer outcomes compared to persons living with unilateral vestibular loss.

Recent evidence suggests that early rehabilitation promotes optimal outcomes, with better resultant dynamic visual acuity scores and higher (better) VOR gains [2-3]. The physiotherapy assessment should be comprehensive to eliminate the chance that the patient has a comorbid central vestibular disorder. ***History taking is key*** as persons with unilateral vestibular hypofunction may or may not have hearing loss, usually have vertigo plus nausea, report visual blurring or oscillopsia, and have direction fixed nystagmus that if seen acutely will be evident with eyes open in the light. After a few days, one may only see the direction fixed nystagmus in the dark with infrared goggles. Assessing the ***integrity of cranial nerves*** 3, 4 and 6 is essential in the examination to ensure that the extra-ocular muscles are intact. ***The head impulse test*** will be either unilaterally or bilaterally positive, they often lose more than 2 lines with the clinical ***dynamic visual acuity*** test, have a positive ***vibration*** induced nystagmus test, will often have nystagmus with the ***head shake test*** and will have normal smooth pursuits, saccades and VOR cancellation. Persons with bilateral loss will lose many lines with dynamic visual acuity testing, with less severe loss of dynamic visual acuity in persons with unilateral loss.

If ***abnormalities*** are noted with ***smooth pursuits*** (saccadic eye movements are noted during the task), ***saccades*** (over or undershooting the target), or ***VOR cancellation*** (an inability to keep the eye on the target as the head, eyes and target move at the same time about the axis of the head), a **central vestibular disorder is suspected**. During the extraocular eye movement testing, one needs to assess for direction changing nystagmus with the eyes focusing on the target 30 degrees to the right and left of the persons nose while holding gaze. The gaze at the fixed target should be maintained for up to 30 seconds to determine the direction of the nystagmus plus to determine if the fast phase of the nystagmus is in the same or opposite direction. If there is a change in the direction

of the nystagmus and the person was referred with a peripheral diagnosis, the physician should be contacted and imaging most likely will be ordered to determine if there is any central involvement.

Gait should be assessed as many patients will have disruptions in ambulation and occasionally impairments in static balance. Ataxia is NOT expected in persons with unilateral or bilateral vestibular loss but drifting to the right or left, especially with active head movement, is often noted [4]. It is rare that anyone falls during the Romberg test. If this is noted, it is essential to assess distal sensation. Peripheral neuropathy or diabetic neuropathy may be the reason for their disrupted postural control in standing on a firm surface. It is not unusual for persons with peripheral vestibular loss to have disrupted postural control acutely while standing on a foam pad with eyes open or closed. One expects improvement with rehabilitative efforts with standing on the foam pad with eyes open but standing on the foam (complaint) pad may continue to be challenging for persons with vestibular loss,

Reports of ***visual blurring*** are common early after vestibular loss. With bilateral vestibular loss oscillopsia (visual blurring associated with movement of the head) continues to be one of their primary complaints plus gait instability. Early after vestibular loss, patients will report an inability to read if they are moving their head, which may not change with severe bilateral loss. With severe bilateral loss, persons are at an increased risk of falling [5]. Persons with bilateral loss have great difficulty walking on uneven or complaint surfaces and walking in the dark (low light conditions).

2. Rehabilitation Techniques

Rehabilitation techniques include adaptation, aquatic exercises, habituation, vergence with VOR training [6], substitution, vibrotactile feedback [7], virtual reality [8-9], strengthening, cognitive behavioral techniques, motivational interviewing, patient education, and walking programs [4].

The updated clinical practice guideline for vestibular hypofunction provides additional support for the use of rehabilitation for persons living with unilateral and bilateral loss, although there has been less improvement noted in persons with bilateral versus unilateral vestibular loss. The clinical practice guideline provides additional information with tables of outcome measures commonly utilized plus specific exercises that have been used in rehabilitation across studies [1].

A newer technique to consider for persons with unilateral loss is performing VOR x1 with vergence [6]. The technique results in better increases in VOR gain that the traditional VOR x 1 exercise [6]. The VOR x 1 exercise is very helpful for habituation and may result in changes in VOR gain, but the VOR x1 exercise is less robust in its ability to change the gain of the VOR.

New technologies are under development in Australia and the US that attempt to increase the VOR gain only on the involved side to attempt to decrease the asymmetry that occurs with traditional exercises [10]. The unilateral VOR gain adaptation device is being tested in a randomized trial and it not commercially available yet. There is evidence that there are VOR gain changes even within one treatment session [10].

TEST	FINDING
Cranial nerve function of 3, 4 nd 6	Normal
Extraocular eye movements	Normal
Head impulse test	Abnormal on one or both sides
Head shake test	Abnormal – nystagmus will be noted
Dynamic visual acuity	Abnormal – lose > 2 lines
Vibration induced nystagmus	Nystagmus is noted as an abnormal finding
VOR cancellation	Normal
Modified test of clinical test of sensory organization	Often they can not stand on a foam pad with eyes open or
Saccadic testing	Normal
Smooth pursuits	Normal
Finger rub to assess for hearing loss	Could be normal, decreased or absent in one or both ear depending on whether it is unilateral or bilateral
Dynamic Gait Index	Early may have impairments in walking with head turns, head pitch movements, changes in speed during gait, and they may have difficulty with turning and stopping quickly
Functional Gait Assessment	Early may have impairments and difficulty with the tandem walking and waking with eyes closed in addition to the items listed above included in the Dynamic Assessment
Subjective visual vertical	Abnormal (off > 2 degrees) with the « bucked » test

Table. Common vestibular test findings in persons with vestibular hypofunction that can be done with low-cost equipment including an eye chart, a vibrator, a specially configured bucket, and a foam pad.

Figure 1. The patient is demonstrating the VOR x 1 exercise while doing vergence concurrently. The patient is instructed to move their head to the right and left (A) while moving the viewed target closer (B) and closer (C) from towards her face while keeping the target in focus.

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EXPLAINING « UNEXPLAINED DIZZINESS »

Dizziness and Cerebral Small Vessel Disease

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1. Background

Three in ten older people (> 65 years) complain of significant dizziness,[1,2] and despite investigation this remains largely unexplained.[3,4]

Impaired balance control,[4] or poor vestibular perception[5,6] have been suggested as mechanisms underpinning such "idiopathic dizziness".

Epidemiological studies have shown consistent association with vascular risk[1,2,7], and age[2,7] - the prevalence increasing to 50% in people over 80 years old.[2,7] Vascular risk factors have been proposed to produce dizziness through their association with cerebral small vessel disease.[4–6] Studies of cerebral small vessel disease and dizziness have however been largely inconclusive.[8–10] One retrospective investigation found an association between qualitatively severe cerebral small vessel disease and "unexplained dizziness" in specialist neuro-otology clinics, in addition to excess vascular risk and subclinical impairment of balance.[4]

Whether small vessel disease effects in white matter tracts and networks relevant to balance, or vestibular perception may account for idiopathic dizziness remains unclear (Figure 1).

We prospectively recruited patients with idiopathic dizziness (see Box 1 for a typical case description) and age-matched controls to characterise this syndrome and clarify the relationship with small vessel disease. Patients had standardised clinical assessments, tests of vestibular function, cognition, balance and gait, and structural and diffusion brain MRI (*manuscript in preparation*).[11] Separately, we investigated electroencephalographic correlates of balance control to understand the implications of idiopathic dizziness for balance-relevant brain activity, and networks involved in balance control.[12] As patients with advanced small vessel white matter disease are known to suffer from an obvious gait disorder and falls,[13] the patients recruited for our studies did not have clinical gait disorder.

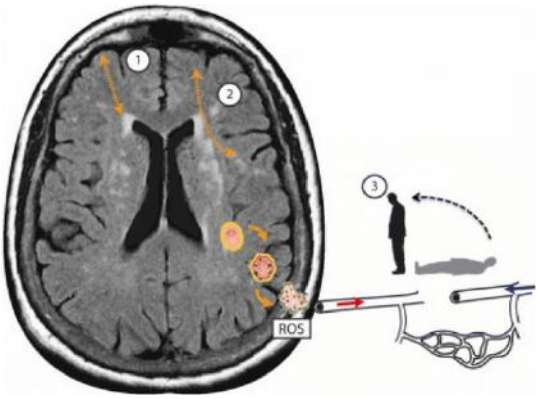

Figure 1 - Overview of proposed mechanisms for dizziness in cerebral small vessel disease. *Frontal cortico-subcortical (1), and cortico-cortical (2) disconnectivity are illustrated. Localized oxidative stress processes damage the cerebral vasculature, leading to endothelial dysfunction and promoting neurodegenerative alterations in the brain tissue through reactive oxidative species (ROS); disrupted cerebrovascular autoregulation may also putatively account for postural symptoms reported by these patients (3). Reproduced from Kaski et al.[6]*

2. Results

Patients with idiopathic dizziness had more vascular risk factors, sub-clinically poorer balance and worse cognitive function than controls. This occurred in association with greater frontal white matter hyperintensity on MRI, and lower fractional anisotropy in the genu of the corpus callosum and the right inferior longitudinal fasciculus. A large bi-hemispheric white matter network had less structural connectivity in patients. Vestibular function and vestibular perceptual function were normal.[11]

EEG power correlates of postural control differed in patients with idiopathic dizziness and controls.[12] Theta and alpha frequency oscillations reduced more on standing in patients in association with spontaneous sway.

Subjective instability correlated with low-frequency (delta) oscillatory power. Low-frequency (delta) oscillations (which have been linked to top-down executive control)[14] associated with less stability during quiet standing in both groups. More whole brain white matter hyperintensity volume correlated with more sway-related low-frequency oscillations in controls (Figure 2); the opposite relationship (negative correlation) was however seen in patients. Only patients engaged a low-frequency (delta) sway-influenced connectivity network.[12]

Box 1 - A typical patient with idiopathic dizziness

A 75 years old man complains of persistent dizziness progressing gradually over the last 6 years. Symptoms are present when standing or walking but are absent in bed. Past medical history is of treated hypertension.

General neurological examination is unremarkable for age. Gait is within normal limits for age and Romberg's test is normal. He is slow on heel-toe walking. On retropulsion he takes 2 steps to maintain balance. Eye movements are normal. Hallpike manoeuvres produce neither nystagmus nor symptoms, but he reports feeling briefly dizzy on the way up. Lying and standing blood pressures show no postural drop.

Audiometry shows moderate high-frequency loss consistent with age. Caloric vestibular testing produces normal responses. Brain MRI shows periventricular hyperintensities on T2-weighted images (see Figure 1), consistent with a moderate degree of cerebral small vessel disease; there is no other structural abnormality.

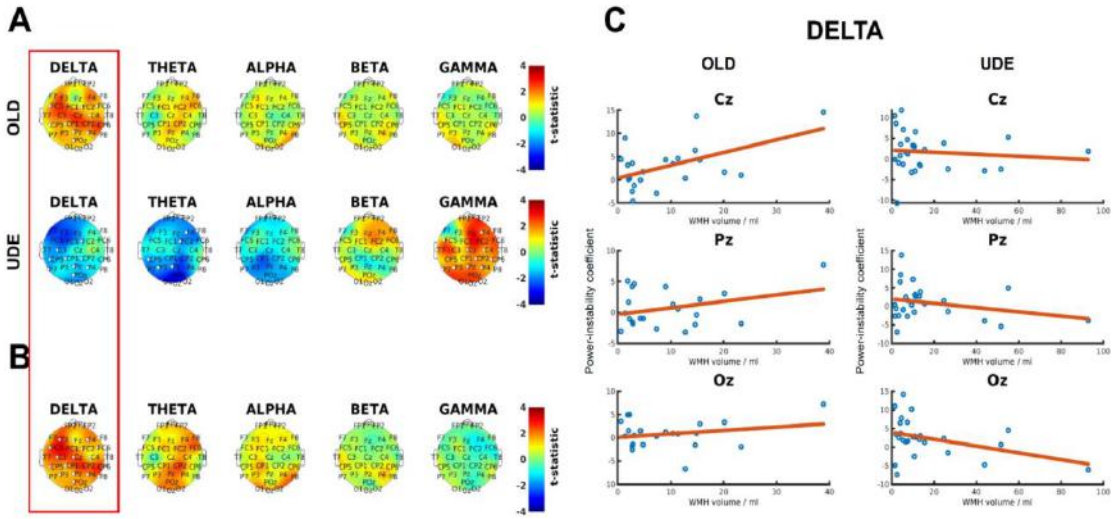

Figure 2: White matter hyperintensities degrade the delta frequency oscillatory response to instability in dizzy patients (UDE).

(A) Effect of increasing whole brain white matter hyperintensity volume on the power-instability coefficient across frequency bands for older adult controls (OLD) and patients with idiopathic dizziness (unexplained dizziness in the elderly, UDE); delta is highlighted. Positive values reflect a positive correlation - more delta power increases with sway as white matter hyperintensity volume increases. (B) Interaction effect between white matter hyperintensity volume and group (OLD, UDE) in predicting the linear relationship between delta band EEG power and sway. Positive values reflect more delta power increases with sway in older controls as white matter hyperintensity volume increases, and less delta power increases with sway in UDE. (C) Plots of linear relationship between delta EEG power and sway (t-statistic of the power-instability co-efficient) vs. white matter hyperintensity volume. These illustrate the interaction effect: a positive linear relationship is seen in older controls, whereas a negative linear relationship is seen in UDE. White dots (in A and B) indicate channel significance using threshold-free cluster enhancement. This significance takes into account the spatial evidence of clustering of correlations by threshold-free cluster enhancement.[15] Reproduced from Ibitoye et al. 2021[12]

3. Conclusion

The results suggest cerebral small vessel disease may impair balance control through microvascular injury to frontal white matter tracts as a basis to idiopathic dizziness in older people. Low-frequency cortical activity which may be involved in the top-down control of balance, and compensation for small vessel disease effects appears to saturate in older people with idiopathic dizziness as a possible neuro-physiological basis for this syndrome.

4. Future Directions

Our work advances the proposition that small vessel disease underpins idiopathic dizziness in older people, but more work is needed. Longitudinal studies will be needed to test if advancing cerebral small vessel disease in balance-relevant tracts predicts idiopathic dizziness in support of a causal role.

Additionally, our 'vascular' hypothesis predicts an association between idiopathic dizziness, more subsequent cerebral small vessel disease progression and poorer long-term cognitive and gait outcomes. The proportion of older people in the *community* complaining of dizziness in whom their dizziness is *idiopathic* remains to be clarified (rather than dizziness accounted for by other disorders such as benign paroxysmal positional vertigo). This would help ascertain the generalisability of our findings beyond the clinic.

Further epidemiological work could also establish if populations with lower vascular risk have less dizziness as predicted by a 'vascular hypothesis'.

Ultimately, established treatments (e.g. tighter blood pressure control), and treatments in development[16] for small vessel disease should be trialled in these patients to determine the effect on dizziness symptoms and long-term cognitive and balance function.

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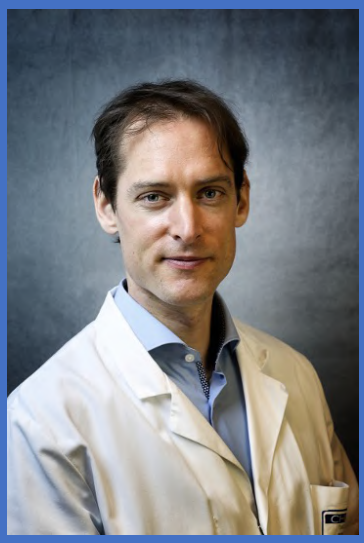

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