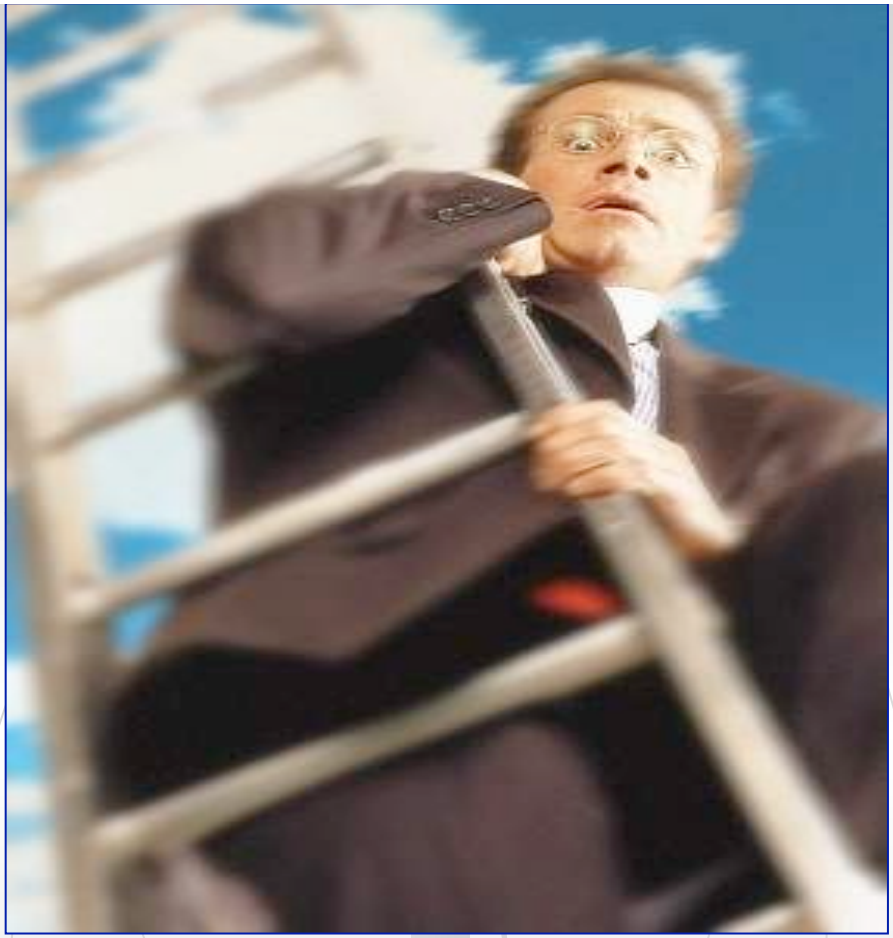
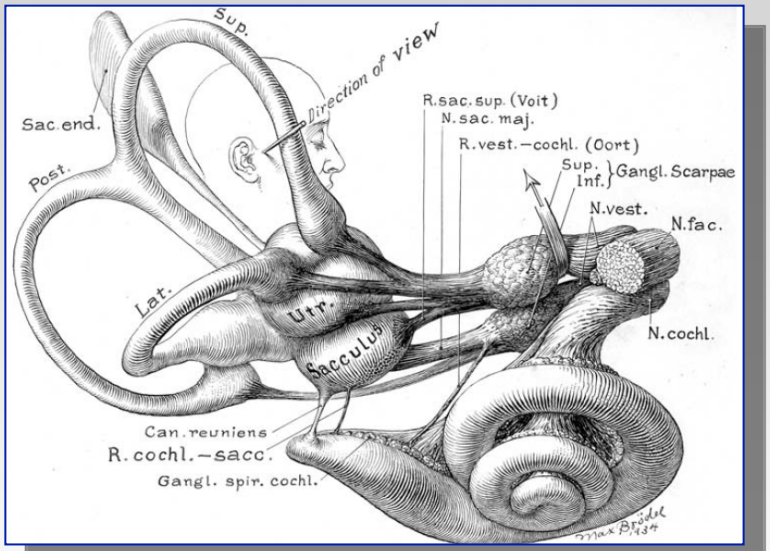


Il Deficit vestibolare acuto monolaterale:

Luigi Califano

UOSD di Audiologia e Foniatria

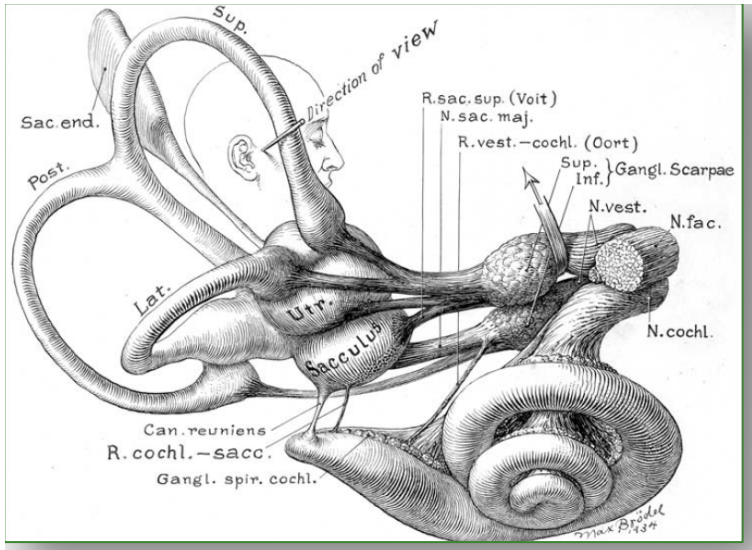
A.O. "S.Pio" Presidio ospedaliero "G.Rummo" Benevento



Malattie del labirinto

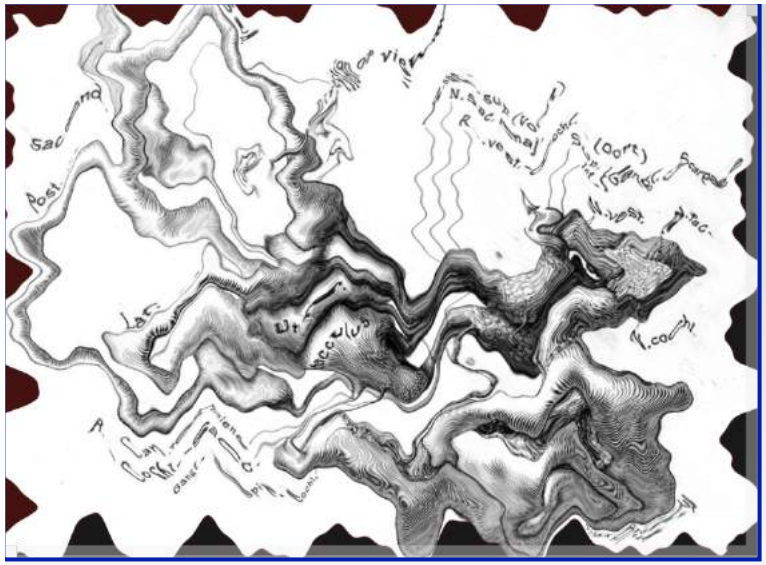
I ASSIOMA: L'ANAMNESI HA SEMPRE UN RUOLO RILEVANTE

II ASSIOMA: UNA PATOLOGIA (COCLEO)LABIRINTICA IN FASE ACUTA PRESENTA SEMPRE SEGNI OBIETTIVABILI, OLTRE CHE I SINTOMI SOGGETTIVI



Malattie del labirinto

- **Criteri diagnostici di sospetto anamnestico per la "vertigine vestibolare" sono la vertigine spontanea rotazionale, la vertigine posizionale, ma anche una vertigine/dizziness ricorrente con nausea ed oscillopsia (Neuhauser, Lempert)**
- **Possono associarsi sintomi uditivi (acufene, fullness, ipoacusia) ad insorgenza improvvisa o progressiva**



Malattie del labirinto

- BPPV (canale posteriore, canale laterale)
- **Deficit vestibolare acuto monolaterale (neurite vestibolare; labirintopatia vascolare acuta; vestibulopatia acuta monolaterale)**
- Malattia di Menière (in evoluzione; ab initio)
- Trauma labirintico
- Sindromi da ipo/areflessia bilaterale: Ototossicosi (esogena; endogena: diabete, dismetabolismi); Presbivertigo
- Migraine related vertigo


I GIOVANNITI
S.O.S. IT
AMBULANZA

Il Deficit Vestibolare acuto monolaterale

Un deficit rapido (o improvviso) della funzione vestibolare monolaterale tale da non permettere temporalmente (in modo immediato) l'intervento di meccanismi periferici o centrali di compenso determina il quadro clinico noto come

"Deficit Vestibolare acuto monolaterale"

L’ equilibrio vestibolare

L’ equilibrio vestibolare perso

NISTAGMO

Il corrispettivo neurofisiologico è costituito dalla perdita improvvisa , parziale o totale, della funzione di un emisistema vestibolare, con il successivo compenso centrale ed il possibile variabile recupero della “funcio lesa”.

Diagnosi clinica e strumentale

✓Comparsa di un attacco vertiginoso acuto, improvviso e spontaneo, con durata di oltre 24 ore , accompagnato da nausea e vomito

✓Grave difficoltà nel mantenere la posizione eretta (fase precoce) e/o andatura atassica (fase medio-tardiva)

✓Presenza di nistagmo spontaneo, orizzontale rotatorio, unidirezionale, stazionario e persistente, con geotropismo, "modulabile" mediante alcuni tests (HST, Iperventilazione, Test vibratorio, Ice test simultaneo)

✓Presenza di una disfunzione canalare monolaterale significativa, evidenziabile dal Test impulsivo sec. Halmagyi e da quello calorico. La disfunzione può essere transitoria o persistente

✓Otoscopia e valutazione audiologica negativa

✓Assenza di deficit neurologici associati.

…evoluzione clinica

- STADIO I (primi 3-5 gg): ny spontaneo I-II-III grado visibile ad occhio nudo (ny deficitario)
- STADIO II (prime 2-3 settimane): ny spontaneo soppresso dalla fissazione, visibile con occhiali di Frenzel o VNS
- STADIO III (dopo le prime 4 settimane) ny spontaneo di recupero (incostante) (Ny irritativo)
- STADIO IV (dopo circa 3 mesi) scomparsa del ny spontaneo e di posizione ma persistenza di una ipofunzione con compenso centrale evidenziabile con test ad alta frequenza (HTT, HHT, HST, test vibratorio)

Nuti, 2005

Il Deficit Vestibolare acuto monolaterale

Methylprednisolone, Valacyclovir, or the Combination for Vestibular Neuritis

Michael Strupp, M.D., Vera Carina Zingler, M.D., Viktor Arbusow, M.D., Daniel Niklas, Klaus Peter Maag, M.D., Ph.D., Marianne Dieterich, M.D., Sandra Bense, M.D., Diethilde Theil, D.V.M., Klaus Jahn, M.D., and Thomas Brandt, M.D.

VESTIBULAR NEURITIS IS THE SECOND most common cause of peripheral vestibular vertigo (the first being benign paroxysmal positional vertigo). It accounts for 7 percent of the patients who present at outpatient clinics specializing in the treatment of dizziness[1] and has an incidence of about 3.5 per 100,000 population.[2] The key signs and symptoms of vestibular neuritis are the acute onset of sustained rotatory vertigo, postural imbalance with Romberg's sign (i.e., falls, with the eyes closed, toward the affected ear), horizontal spontaneous nystagmus (toward the unaffected ear) with a rotational component, and nausea. Caloric testing (irrigation of the ear with warm or cold water) invariably shows ipsilateral hyporesponsiveness or nonresponsiveness.

In the past, either an inflammation of the vestibular nerve[3-5] or labyrinthine ischemia[6] was proposed as a cause of vestibular neuritis. Currently, a viral cause is favored. The evidence, however, remains circumstantial.[1,7,8] Postmortem studies have shown atrophy of the vestibular nerve and the vestibular sensory epithelium that is similar to the histopathological findings in known viral disorders, such as herpes zoster oticus.[9] Herpes simplex virus type 1 (HSV-1) DNA has been detected on autopsy with the use of the polymerase chain reaction in about two of three human vestibular ganglia.[10,11] This indicates that the vestibular ganglia are latently infected by HSV-1, as are other cranial-nerve ganglia.[12-14] A similar cause is also assumed for Bell's palsy and is strongly supported by the detection of HSV-1 DNA in the endoneurial fluid of affected persons.[15]

Methylprednisolone, Valacyclovir, or the Combination for Vestibular Neuritis

Michael Strupp, M.D., Vera Carina Zingler, M.D., Viktor Arbusow, M.D., Daniel Niklas, Klaus Peter Maag, M.D., Ph.D., Marianne Dieterich, M.D., Sandra Bense, M.D., Diethilde Theil, D.V.M., Klaus Jahn, M.D., and Thomas Brandt, M.D.

BACKGROUND

Vestibular neuritis is the second most common cause of peripheral vestibular vertigo. Its assumed cause is a reactivation of herpes simplex virus type 1 infection. Therefore, corticosteroids, antiviral agents, or a combination of the two might improve the outcome in patients with vestibular neuritis.

Andamento epidemico stagionale ed elevata concomitanza o antecedenza (circa 30%) con flogosi delle vie aeree superiori (Hirata 1989, Shimizu 1992, Silvoniemi 1988)

Late and sudden recovery of sudden deafness or vestibular neuronitis

Reprise fonctionnelle tardive et soudaine de cas de surdité brusque ou de neuronite vestibulaire

J.-P. Guyot[a,*], M. Toupet[b]

Vestibular neuronitis (VN) [1] and sudden deafness (SD) [2] are well-defined entities. Vestibular neuronitis corresponds to a sudden loss of vestibular function resulting in characteristic symptoms of imbalance and segmental body deviation. Viral infection affecting the sensory neuroepithelium of the vestibular endorgans [3], or the nerve [4], as well as a vascular disorder of the inner ear [5] is the most frequently cited hypothetical factors. Other factors could be implicated such as an immunological disorder or toxicity [6]. Cases of VN have also been reported following a blunt head trauma [7].

Vestibular Neuritis

Michael Strupp, M.D.,[1] and Thomas Brandt[2,3]

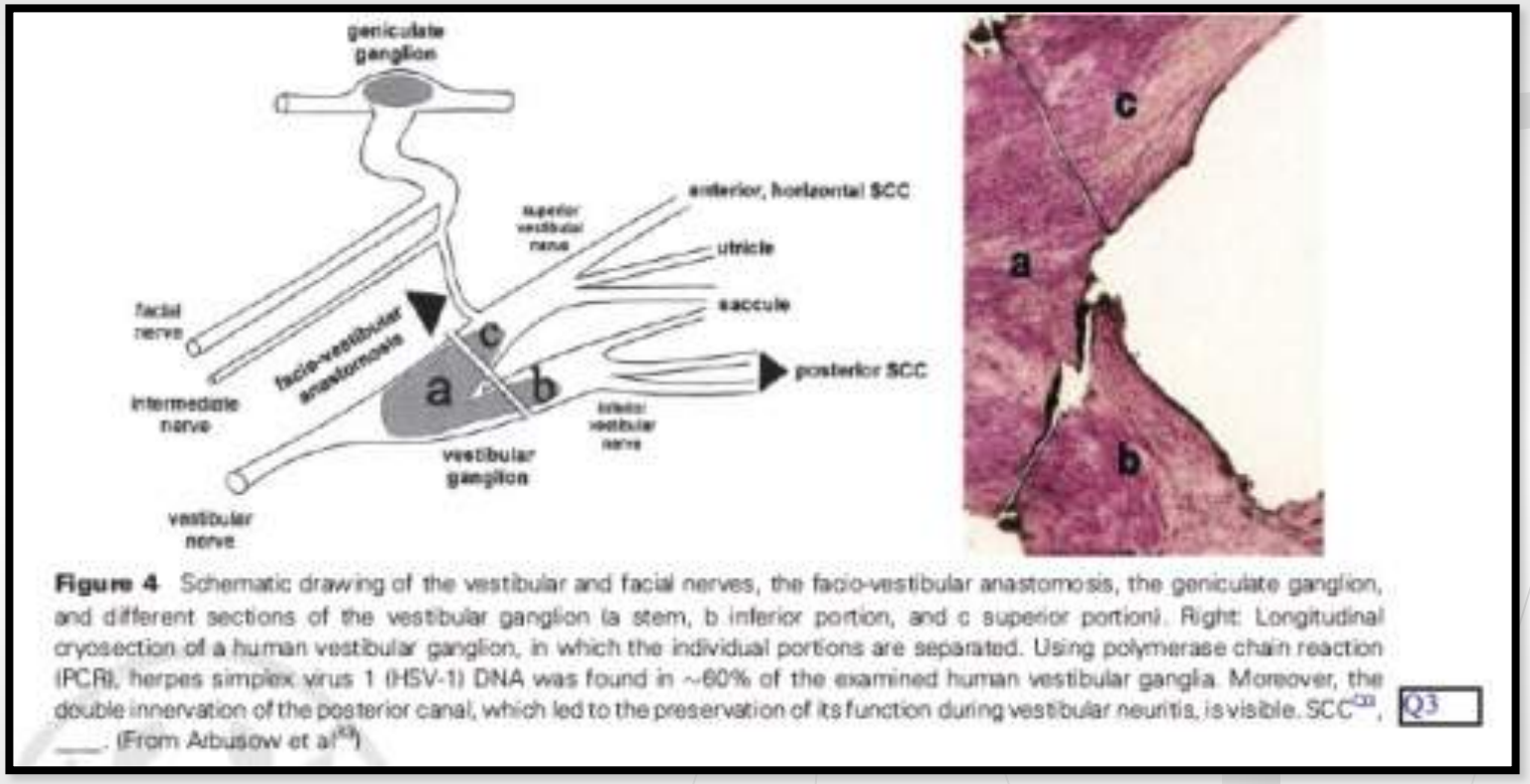


Figure 4 Schematic drawing of the vestibular and facial nerves, the facio-vestibular anastomosis, the geniculate ganglion, and different sections of the vestibular ganglion (a stem, b inferior portion, and c superior portion). Right: Longitudinal cryosection of a human vestibular ganglion, in which the individual portions are separated. Using polymerase chain reaction (PCR), herpes simplex virus 1 (HSV-1) DNA was found in ~60% of the examined human vestibular ganglia. Moreover, the double innervation of the posterior canal, which led to the preservation of its function during vestibular neuritis, is visible. SCC[33], ___. (From Arbusow et al[33])

Il danno riguarda il "nervo vestibolare superiore" : canale anteriore, canale laterale, utricolo, (sacculo): NEURITE VESTIBOLARE SUPERIORE

Anatomic Differences in the Lateral Vestibular Nerve Channels and their Implications in Vestibular Neuritis

*Gerard Gianoli, †Joel Goebel, ‡Sarah Mowry, and ‡Paul Poomipannit

Conclusion: The lateral bony channel of the superior vestibular nerve is seven times longer than the inferior vestibular and more than three times longer than the singular channel. There are a larger percentage of bony spicules occupying the superior vestibular compared with the inferior vestibular or singular channels. In addition, the superior nerve passes through a longer area of severe narrowing compared with the inferior or singular nerves. This anatomic arrangement of a longer bony channel with more interspersed bony spicules could make the superior vestibular nerve more susceptible to entrapment and ischemia.

CLINICAL NOTE

Acute Vestibular Neuritis Visualized by 3-T Magnetic Resonance Imaging With High-Dose Gadolinium

Mikael Karlberg, MD, PhD; Mårten Annertz, MD, PhD; Måns Magnusson, MD, PhD

Enhancement ad alte dosi di Gd del solo ganglio genicolato a destra

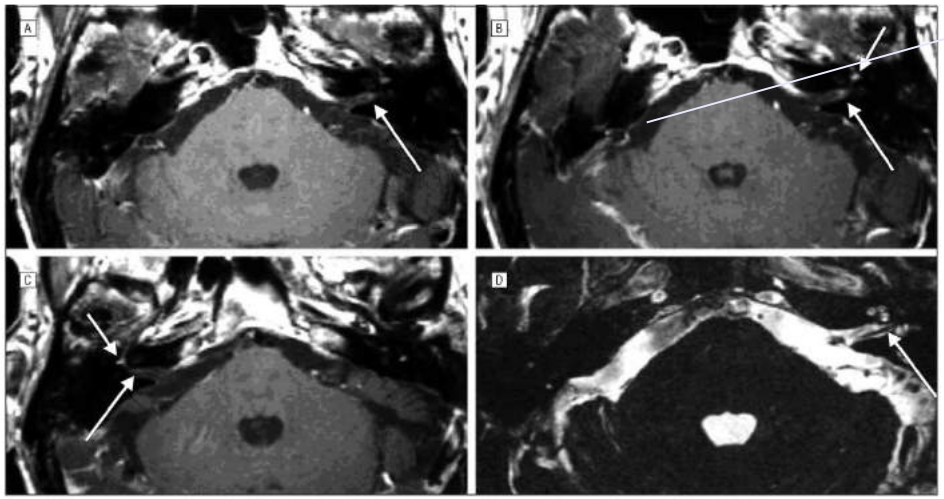
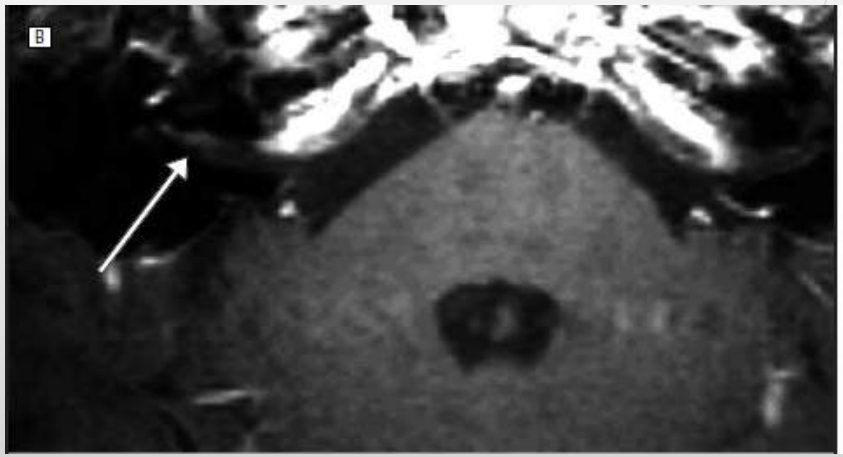


Figure 1. Magnetic resonance images (3.0 T) of a patient with left-sided acute vestibular neuritis (case 1). A, An axial T1-weighted image using 0.1 mmol/kg of body weight of gadolinium–pentetic acid (Gd-DTPA) shows no clear-cut enhancement of the left vestibular nerve (arrow). B, An axial T1-weighted image using 0.3 mmol/kg of Gd-DTPA shows contrast enhancement of the left vestibular nerve (long arrow) and the left geniculate ganglion (short arrow). C, An axial T1-weighted image using 0.3 mmol/kg of Gd-DTPA shows no contrast enhancement of the right vestibular nerve (long arrow) but does show contrast enhancement of the right geniculate ganglion (short arrow). D, An axial constructive interference in steady state image shows no abnormality of the left vestibular nerve (arrow).

Enhancement ad alte dosi di Gd del nervo e del ganglio genicolato a sinistra



The physiologic data from our 2 patients (Table) suggest that the disease process was located on the superior vestibular nerve. No coronal or sagittal images were recorded with high-dose Gd-DTPA, and it is thus not possible to tell if the MRI enhancements were localized to the superior vestibular nerve.

The Laryngoscope

Clinical Value of 4-Hour Delayed Gadolinium-Enhanced 3D FLAIR MR Images in Acute Vestibular Neuritis

Hayoung Byun, MD; Jae Ho Chung, MD, PhD; Seung Hwan Lee, MD, PhD; Chul Won Park, MD, PhD; Dong Woo Park, MD, PhD; Tae Yoon Kim, MD

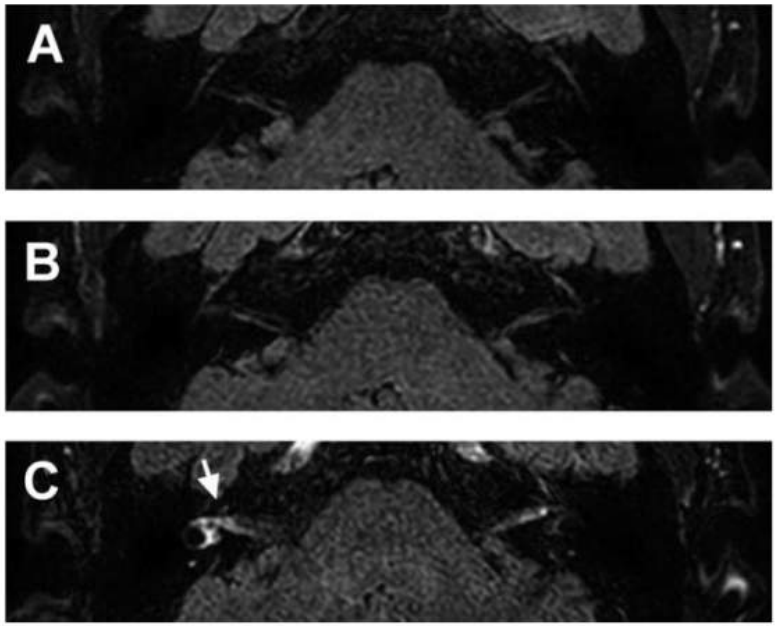


Fig. 1. A 3D-FLAIR MR image in a 47-year-old female patient with right-sided vestibular neuritis. Precontrast (A) and 10-minute delayed (B) images did not show notable enhancement. The right vestibular nerve and inner ear structures (white arrow) are clearly visible in the 4-hour delayed image (C).
3D-FLAIR = three-dimensional fluid-attenuated inversion recovery.

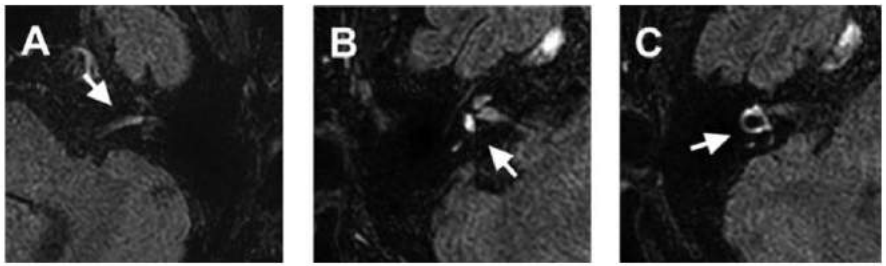


Fig. 2. Four-hour delayed 3D-FLAIR MR images showing the variable extent of enhancement: the left vestibular nerve in the internal auditory canal (A), right vestibule (B), and right lateral semicircular canal (C). Regions of interest are indicated by a white arrow.
3D-FLAIR = three-dimensional fluid-attenuated inversion recovery.

In this study, increased signal intensities of the affected vestibular nerve and inner ear fluid structures were observed in 20 of 29 patients (69.0%) in 4-hour delayed MR images (Table I). In 12 of these patients, the enhancement affected the whole inner ear, including the vestibule and SCCs. In terms of enhancement pattern, enhancement of the vestibule and IAC was always seen in cases with strong enhancement of the SCCs. There were no cases with enhancement of the SCCs only. We thus suppose that inner ear enhancement in VN occurs in sequence from IAC and fundus to SCC, and that the greater the weakening of the BLB, the more the Gd molecules enter the perilymphatic space. Such Gd diffusion may explain the high signal intensity of the cochlea in patients with normal hearing thresholds (11 patients, 37.9%, in this study). Other inner ear structures, including the vestibule and SCCs, also had high signal intensities in all cases involving cochlear enhancement. The distribution of contrast medium may be influenced by the circulation of inner ear fluids. This pattern of enhancement could provide an answer to the question whether there was active inflammation of inner ear structures in addition to neuritis. We suggest that the high signal intensity of the labyrinth, accompanied by obvious enhancement of the vestibular nerve, can be explained by diffusion of Gd molecules into the perilymph rather than by active inner ear inflammation, a view supported by the simultaneous cochlear enhancement without hearing loss. It is also still possible,

Review Article

Is Vestibular Neuritis an Immune Related Vestibular Neuropathy Inducing Vertigo?

A. Greco, G. F. Macri, A. Gallo, M. Fusconi, A. De Virgilio, G. Pagliuca, C. Marinelli, and M. de Vincentiis

Organs of Sense Department, ENT Section, Policlinico "Umberto I" University of Rome "Sapienza", Lgo Valerio Bacigalupo 32 C, 00142 Rome, Italy

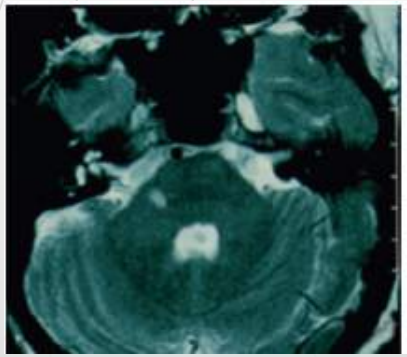
5. Immunological Hypothesis

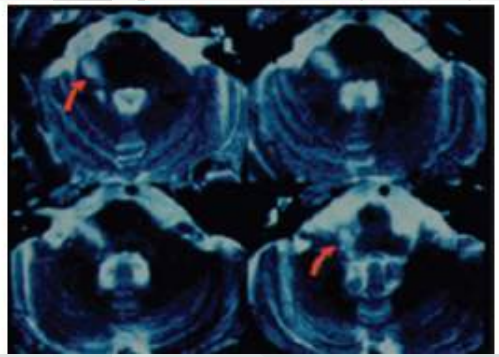
The characteristic interval that separates the onset of a respiratory tract infection and the onset of vertigo may suggest that the disease is caused by an immune mediated complication of the infection rather than direct viral infection of the nerve.

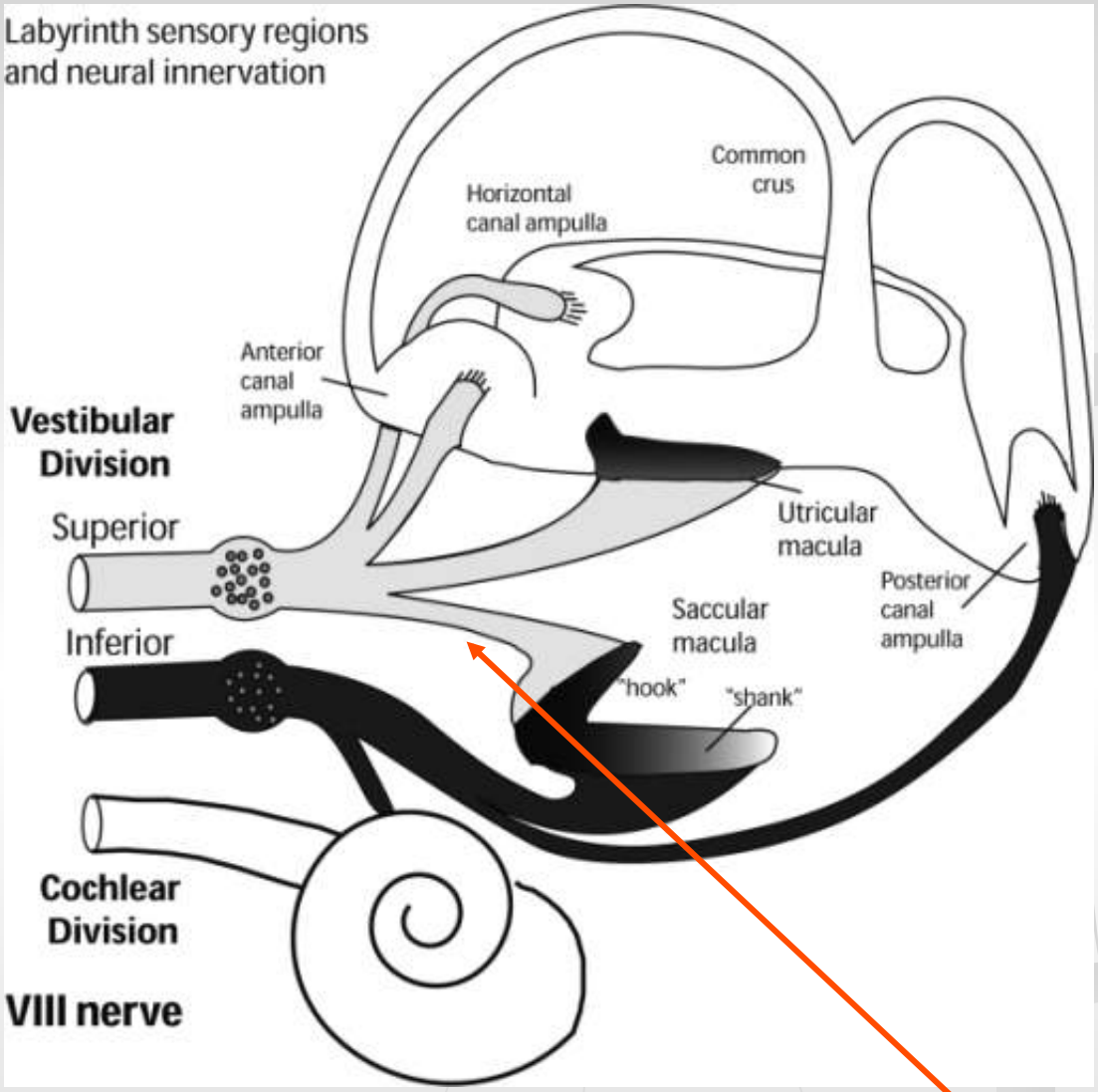
Immune mediated neurological disease is a well-recognised sequela of infectious fevers, and a parallel of localised immune mediated peripheral neuropathy is vaccine-induced brachial neuropathy that occasionally complicates deltoid immunisation.

Immunologic mechanisms have been suggested as possible causes for vestibular neuritis following influenza vaccination [58].

The view that vestibular neuronitis is immune mediated is supported by a study in which thymus lymphocytes-subpopulations (T4 T-helper and T8 T-suppressor cells) were found in inner ear diseases (e.g., sudden hearing loss, neuronitis vestibularis, Menière's disease, and Bell's Palsy) by specific monoclonal antibodies. The T-cell subset ratio (T4/T8) was elevated (less than 3) in approximately 50% of all patients. DR-typing was performed because of the well-known control of the immunoregulation through the class II HLA-DR antigens. There was a relative risk of 5.2 in peripheral vestibular lesion (neuronitis vestibularis). The relative risk of autoimmune diseases is found at this level [59].



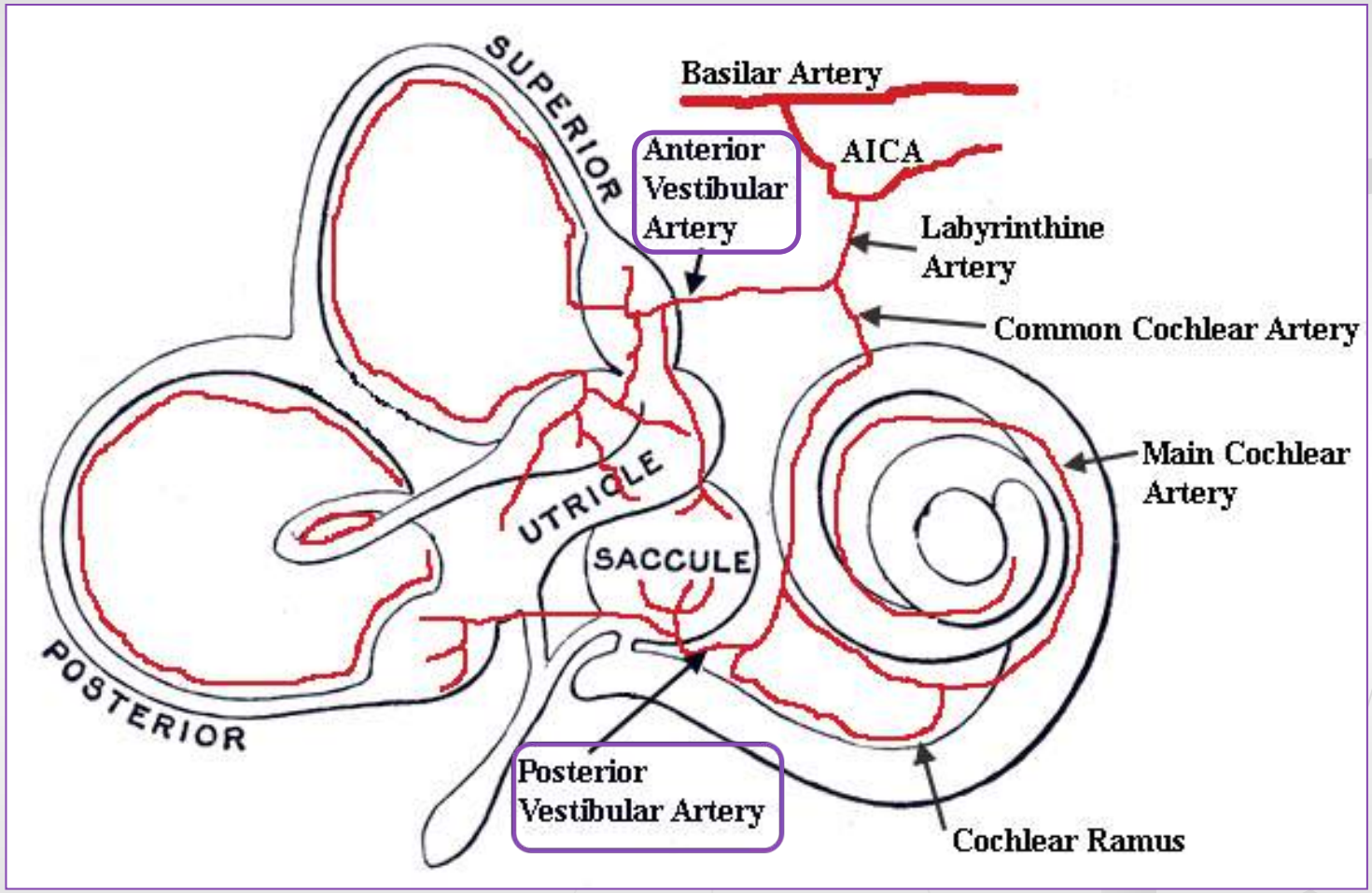




Fibre del nervo vestibolare superiore alla macula del sacculo

La branca vestibolare superiore innerva le creste ampollari dei canali semicircolari anteriore e laterale, la macula dell'utriculo e la porzione antero-superiore della macula del sacculo

La branca inferiore innerva la cresta del canale semicircolare posteriore e la maggior parte della macula del sacculo


SUPERIOR
Basilar Artery
AICA
Anterior Vestibular Artery
Labyrinthine Artery
Common Cochlear Artery
Main Cochlear Artery
UTRICLE
SACCULE
POSTERIOR
Posterior Vestibular Artery
Cochlear Ramus

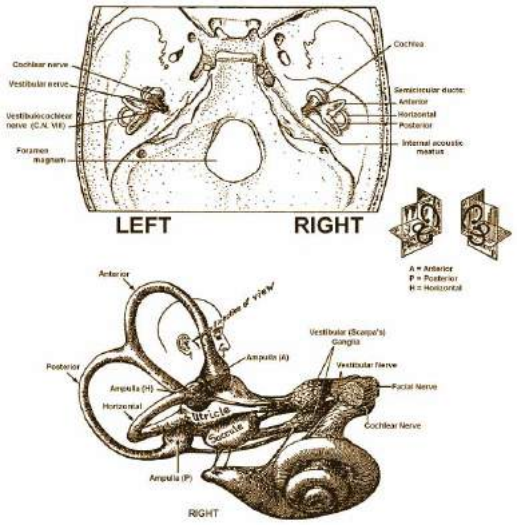
La distribuzione dei due ramuscoli arteriosi, anteriore e posteriore, rispecchia «grosso modo» la distribuzione dell'innervazione tra nervo vestibolare superiore ed inferiore.

POSTURAL VERTIGO DUE TO UNILATERAL SUDDEN PARTIAL LOSS OF VESTIBULAR FUNCTION

JOHN R. LINDSAY, M.D.

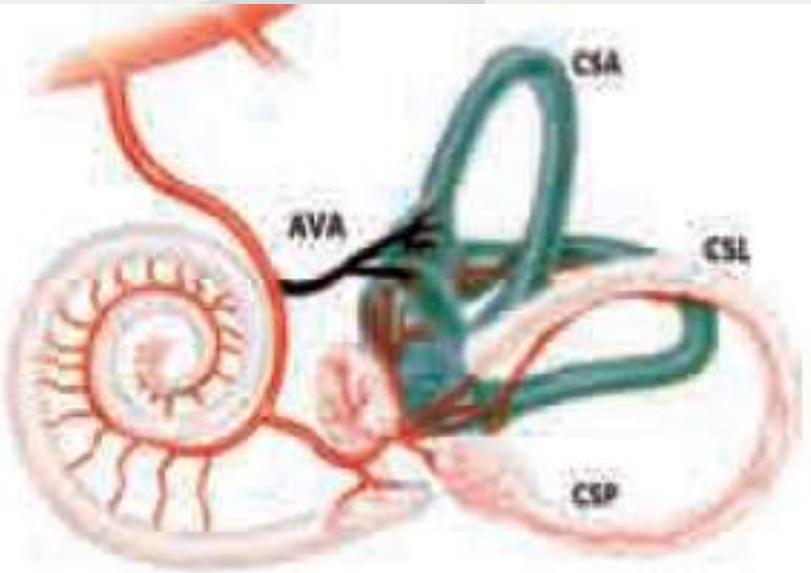
AND

WILLIAM G. HEMENWAY, M.D.



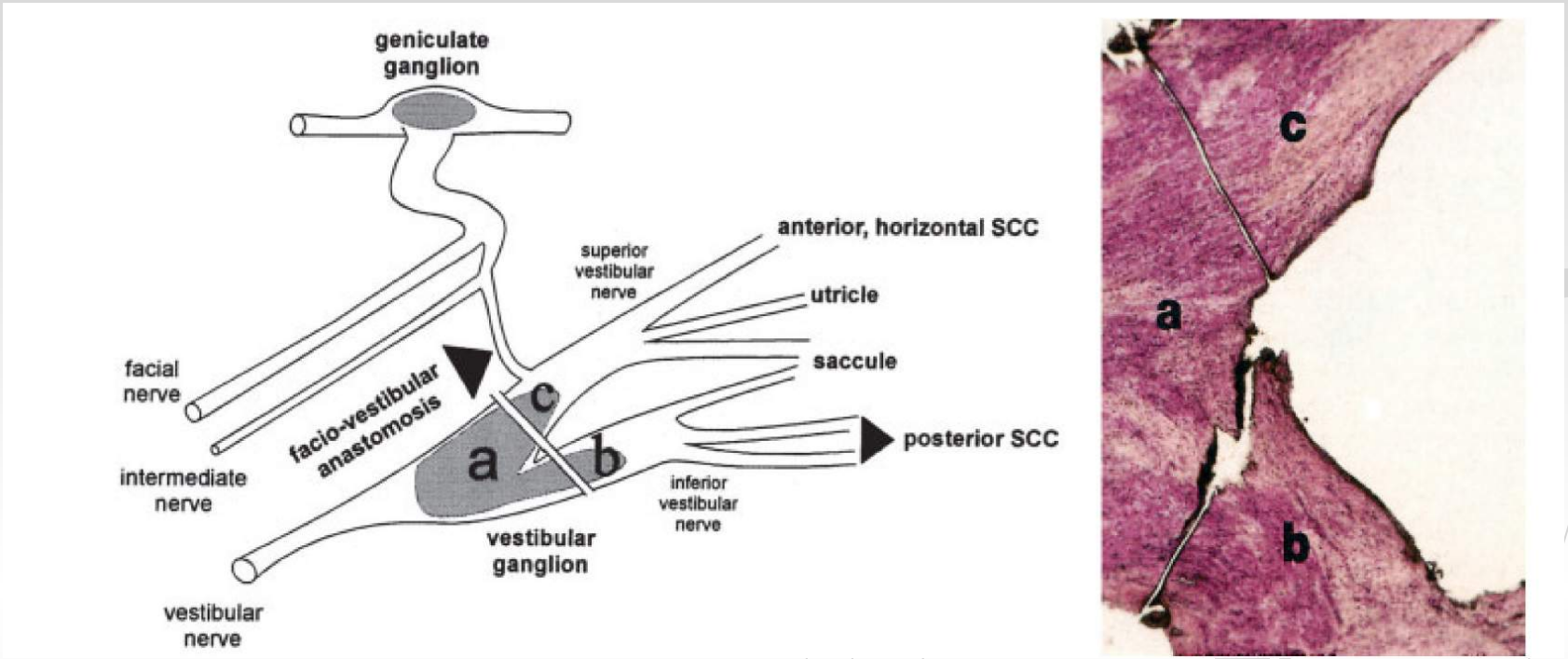
ANATOMIA DEL CIRCOLO POSTERIORE E CORRELAZIONI CON IL CIRCOLO ANTERIORE

Antonio Paparelli, Paola Lenzi, Niccolò Cerchiai, Augusto Pietro Casani

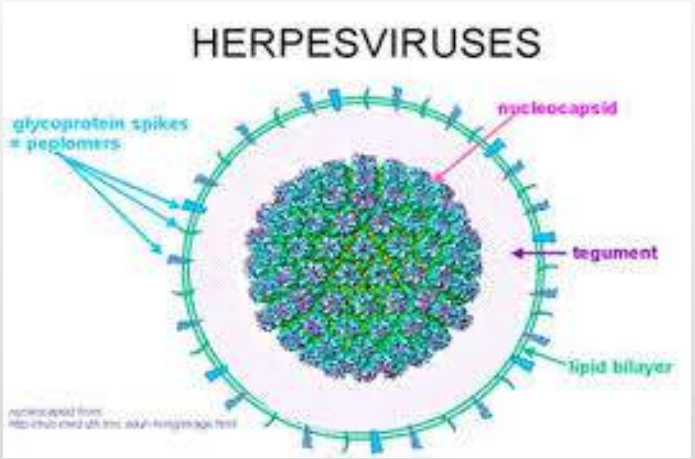


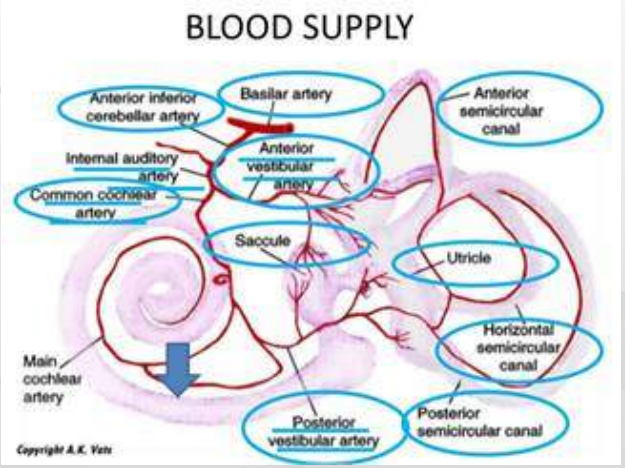
L'ostruzione dell'arteria vestibolare anteriore (AVA) causa una lesione ischemica del canale semicircolare anteriore (CSA), laterale (CSL) e dell'utricolo, mentre il canale posteriore (CSP) viene risparmiato.

Figura 10 Schema della sindrome di Lindasy-Hemenway.


geniculate ganglion
anterior, horizontal SCC
superior vestibular nerve
utricle
saccule
facial nerve
facio-vestibular anastomosis
c
a
b
posterior SCC
intermediate nerve
inferior vestibular nerve
vestibular ganglion
vestibular nerve

c
a
b


HERPESVIRUSES
glycoprotein spikes = peplomers
nucleocapsid
tegument
lipid bilayer


BLOOD SUPPLY
Anterior inferior cerebellar artery
Basilar artery
Anterior semicircular canal
Internal auditory artery
Anterior vestibular artery
Common cochlear artery
Saccule
Utricle
Horizontal semicircular canal
Main cochlear artery
Posterior vestibular artery
Posterior semicircular canal
Copyright A.K. Vats

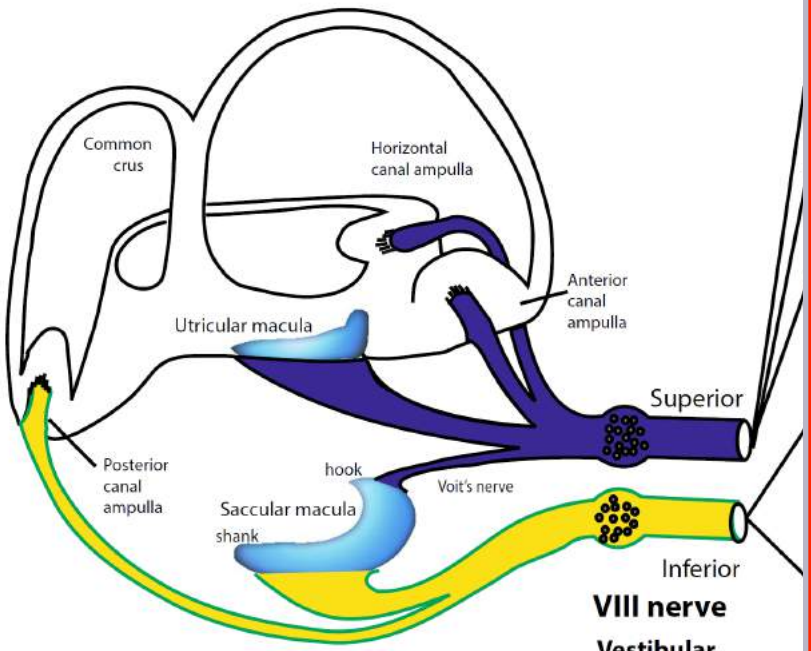
Exp Brain Res (2017) 235:957–972
DOI 10.1007/s00221-017-4874-y

REVIEW

The new vestibular stimuli: sound and vibration—anatomical, physiological and clinical evidence

Ian S. Curthoys[1]

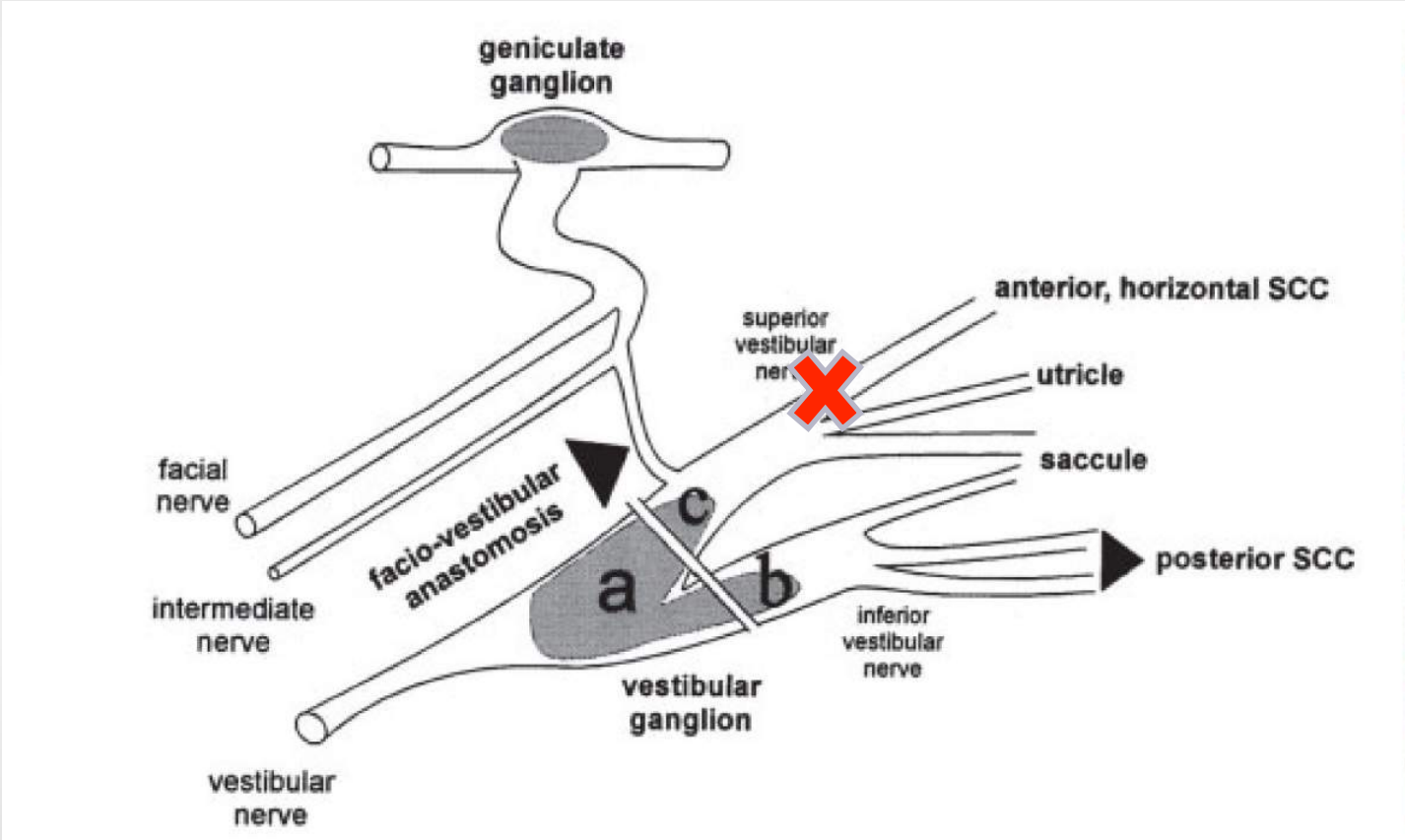
Labyrinth sensory regions and neural innervation



I modelli delle perdite parziali o totale della funzionalità vestibolare ci forniscono un buon modello di studio strumentale selettivo….

….ma non esauriscono la questione

Deficit selettivo «paradigmatico» del nervo vestibolare superiore di sinistra



Eur Arch Otorhinolaryngol
DOI 10.1007/s00405-016-4055-9

OTOLOGY

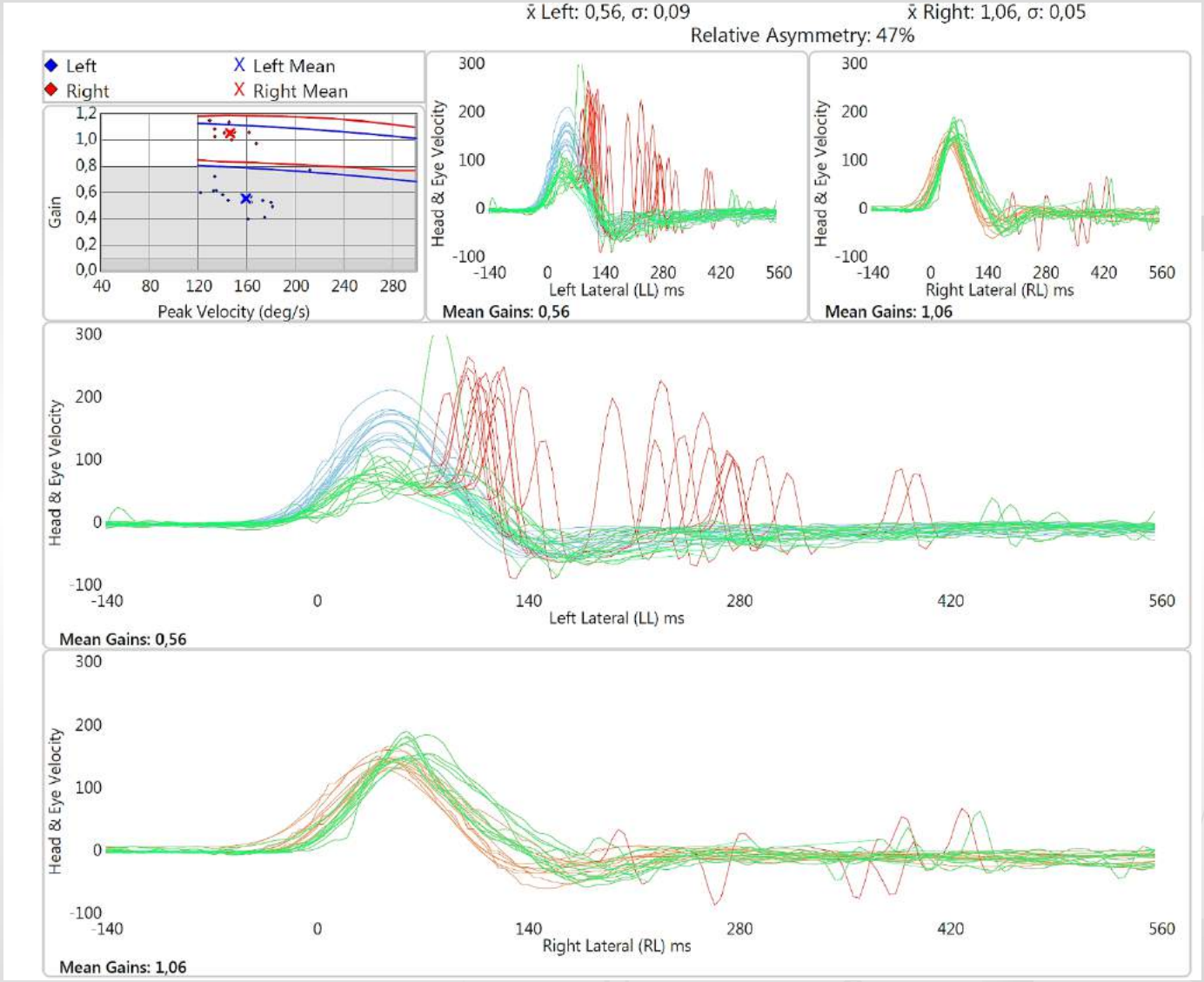
Determining vestibular hypofunction: start with the video-head impulse test

B. F. van Esch[1] · G. E. A. J. Nobel-Hoff[2] · P. P. G. van Benthem[3] · H. J. van der Zaag-Loonen[1] · Tj. D. Bruintjes[1]

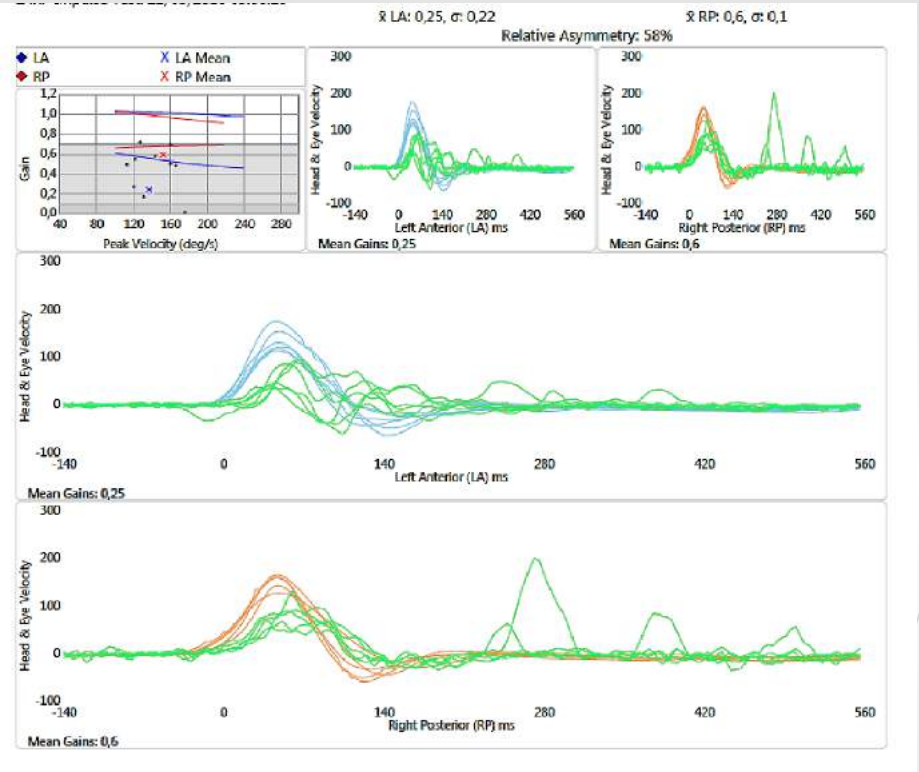
Conclusions

In conclusion, comparison with caloric testing revealed that the vHIT is a very specific rather than sensitive test for detecting vestibular hypofunction. In case of a normal vHIT, additional caloric testing remains indicated and the vHIT does not replace the caloric test. The high positive predictive value of the vHIT, especially if a gain cut-off point of 0.6 is applied, indicates that an abnormal vHIT is strongly related to an abnormal caloric test result. Therefore, in case of an abnormal vHIT, additional caloric testing is not necessary. We conclude that the vHIT is clinically useful as a first test in determining vestibular hypofunction in dizzy patients.

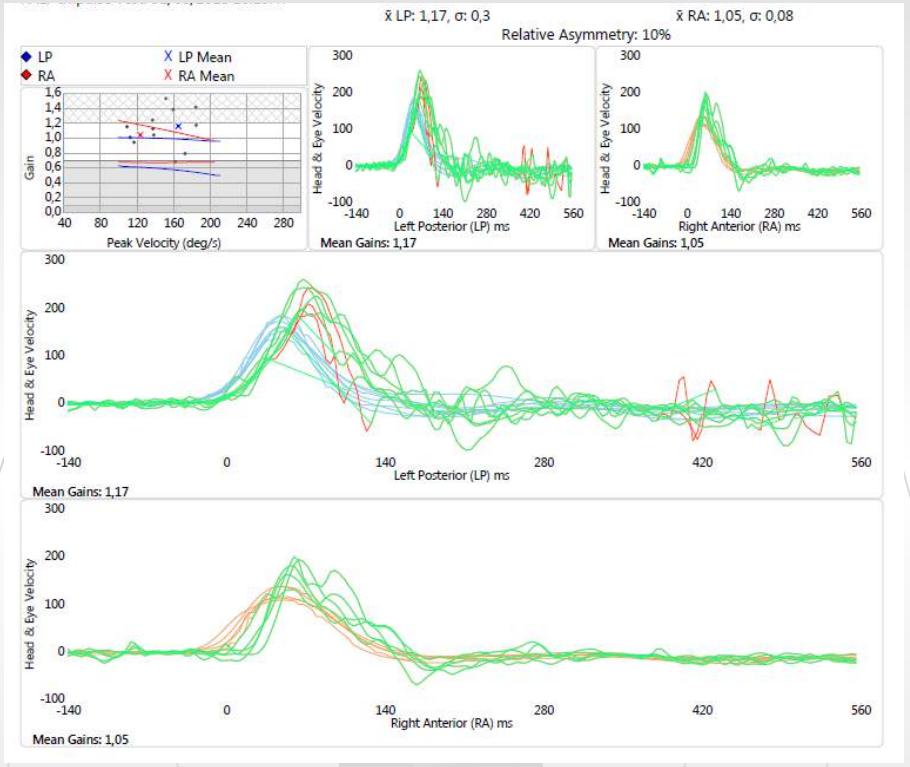
Video Head Impulse test



Sia al protocollo HIM che al Protocollo SHIM deficit del gain laterale sinistro a 0.56



LARP. Gain dell' anteriore sinistro 0.25

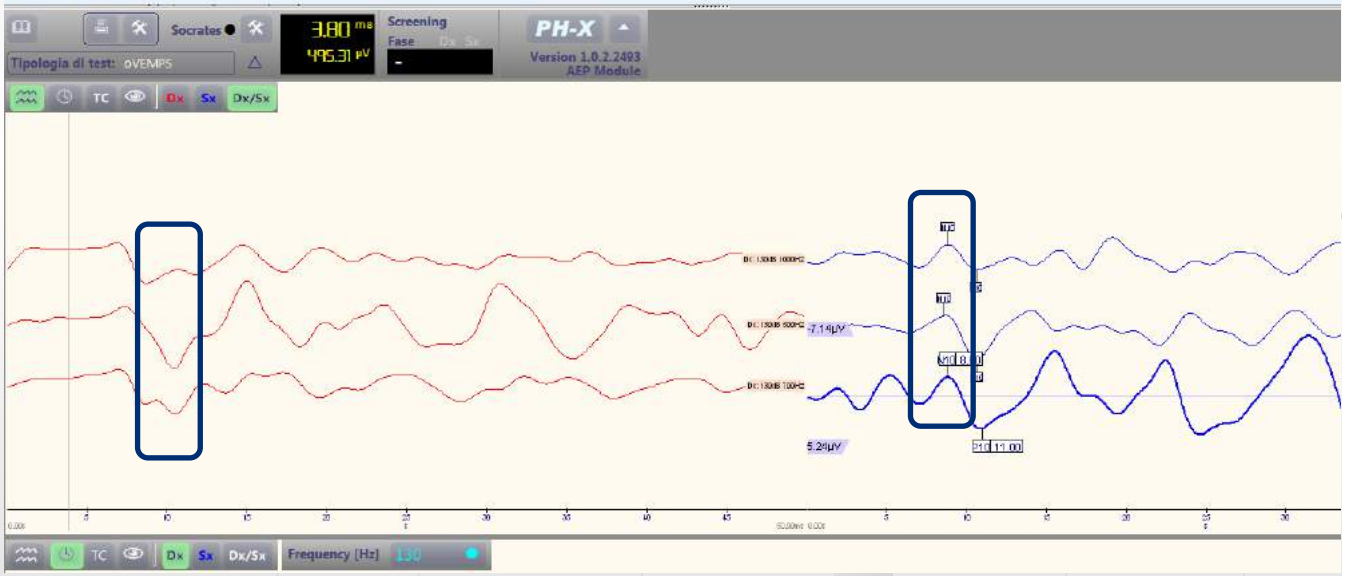


RALP. Gain del posteriore sinistro 1.1

Via sacculare conservata

A sinistra.
Cervical VEMP AC- 500 Hz, 125 dB SPL normorappresentato

Via utricolare danneggiata



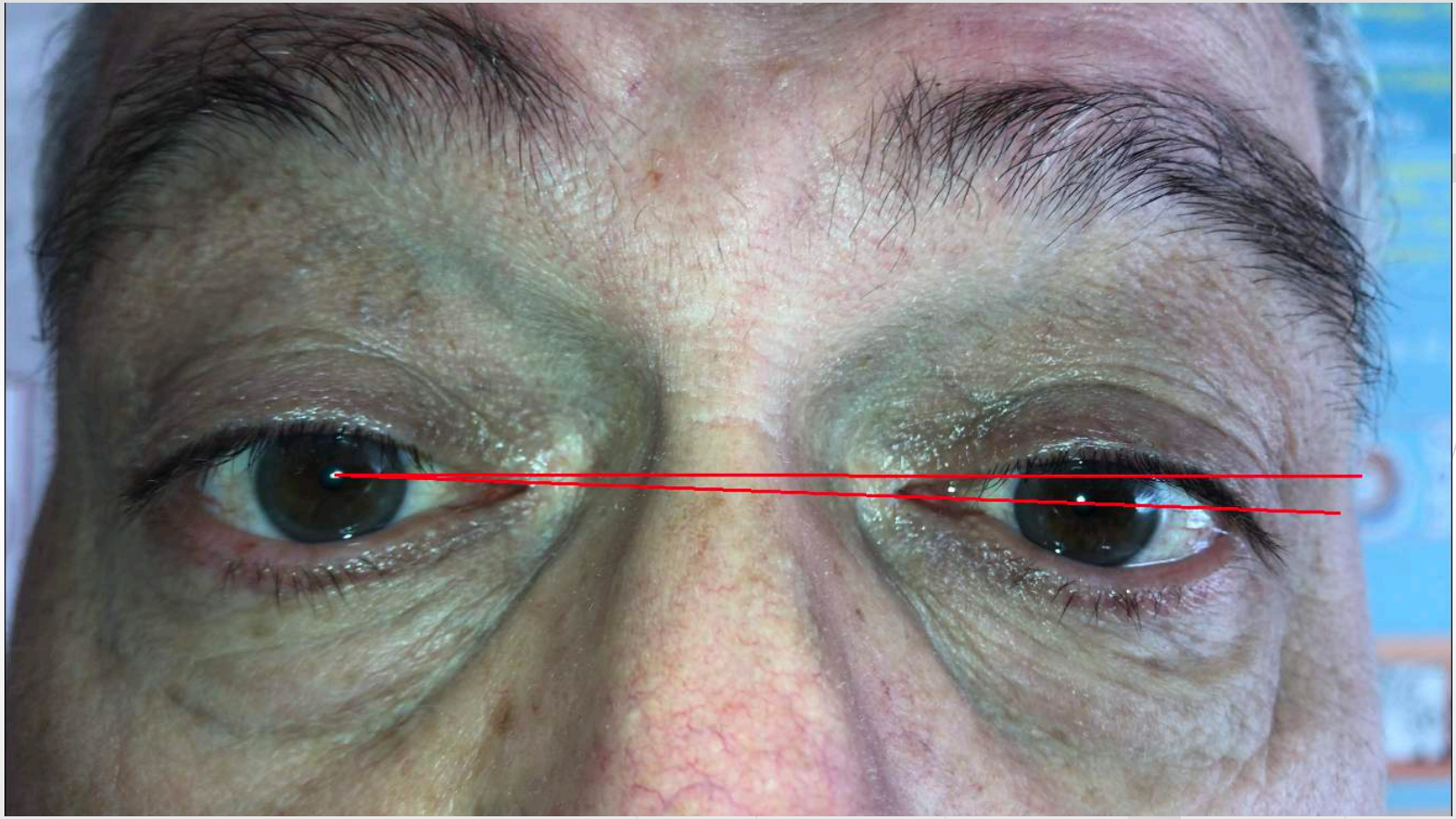
A destra (risposta crociata).
Ocular VEMP BC- 500 Hz, 130 dB SPL assente

Danno all' intero territorio del ramo vestibolare superiore di sinistra

- Canale laterale (basse ed alte frequenze opp. recettori tonici e recettori fasici)
- Canale anteriore
- Sacculo

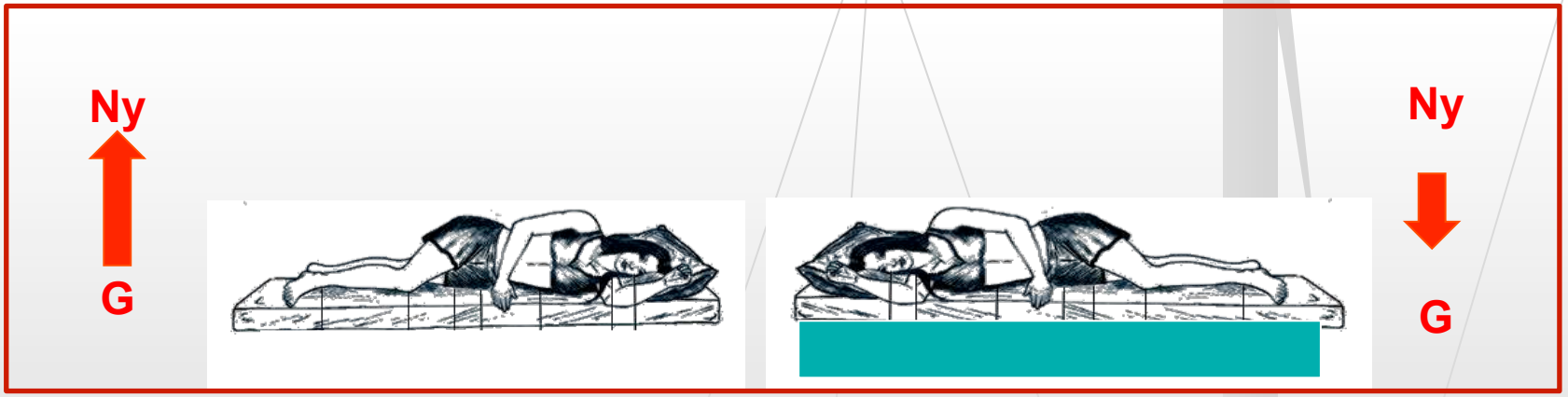
Preservato il territorio del ramo vestibolare inferiore di sinistra

- Canale posteriore
- Utricolo



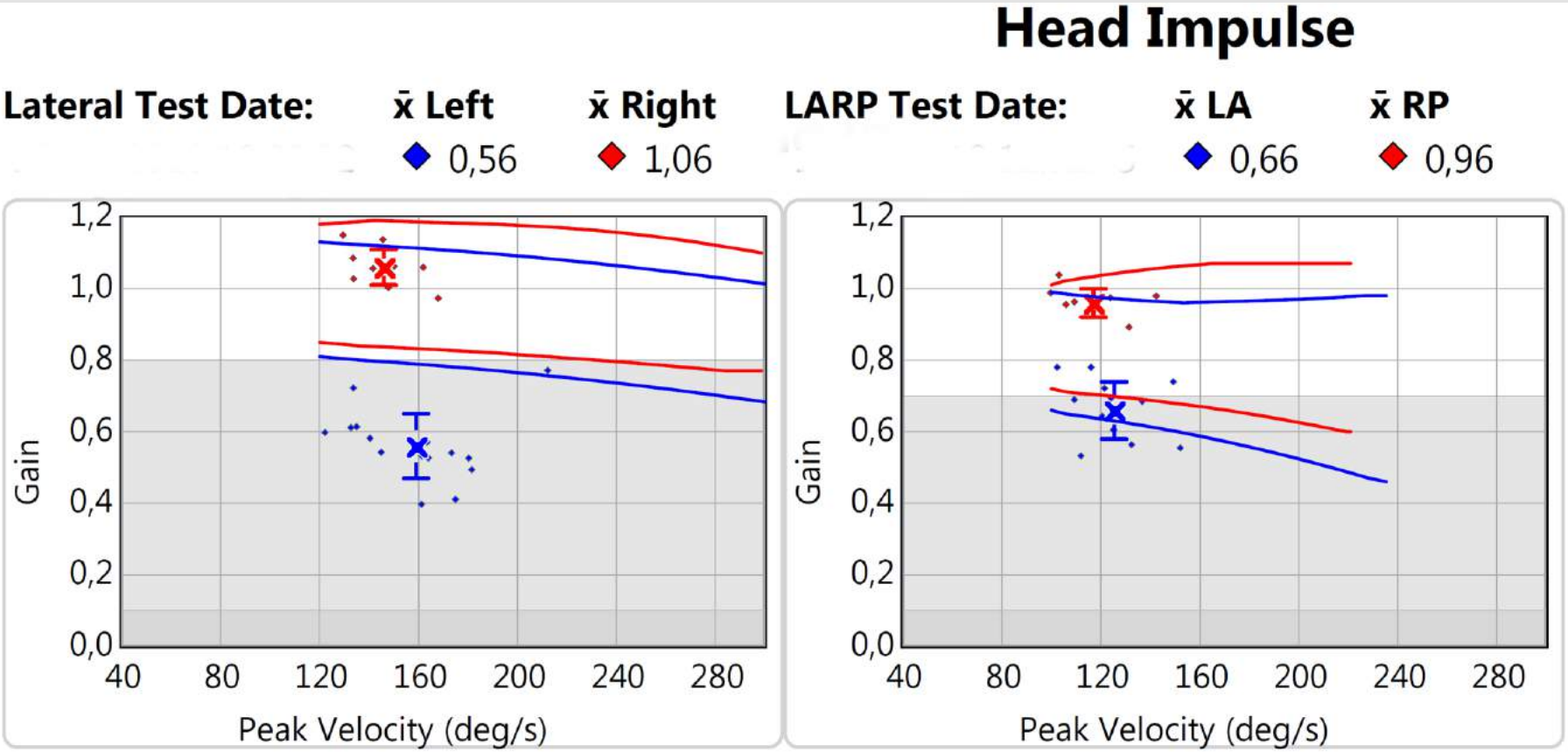
… ma la clinica bed-side ci aveva già detto che la lesione utriculare fosse presente:
Ipotropia oculare sinistra

Il rinforzo del nistagmo in fianco sinistro era indicatore di lesione utriculare sinistra per influenza del vettore gravitazionale G ed il mismatch tra le due macule nei decubiti rispettivi

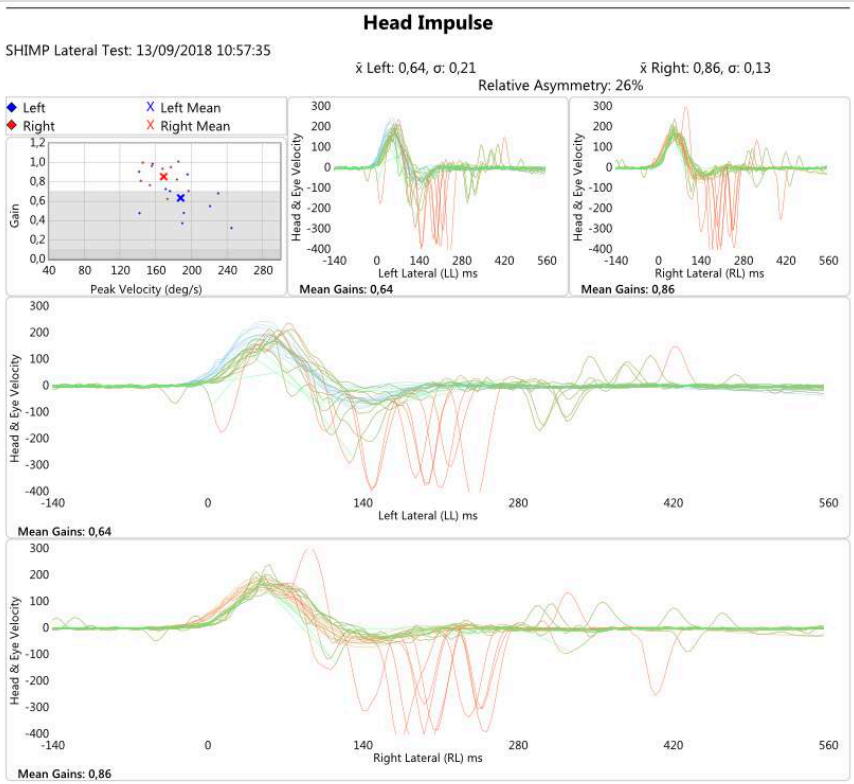


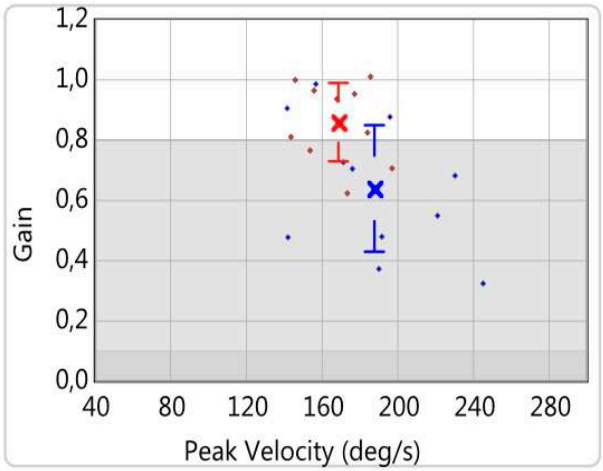
Da V. Marcelli:
Vestibologia clinica

Head Impulse

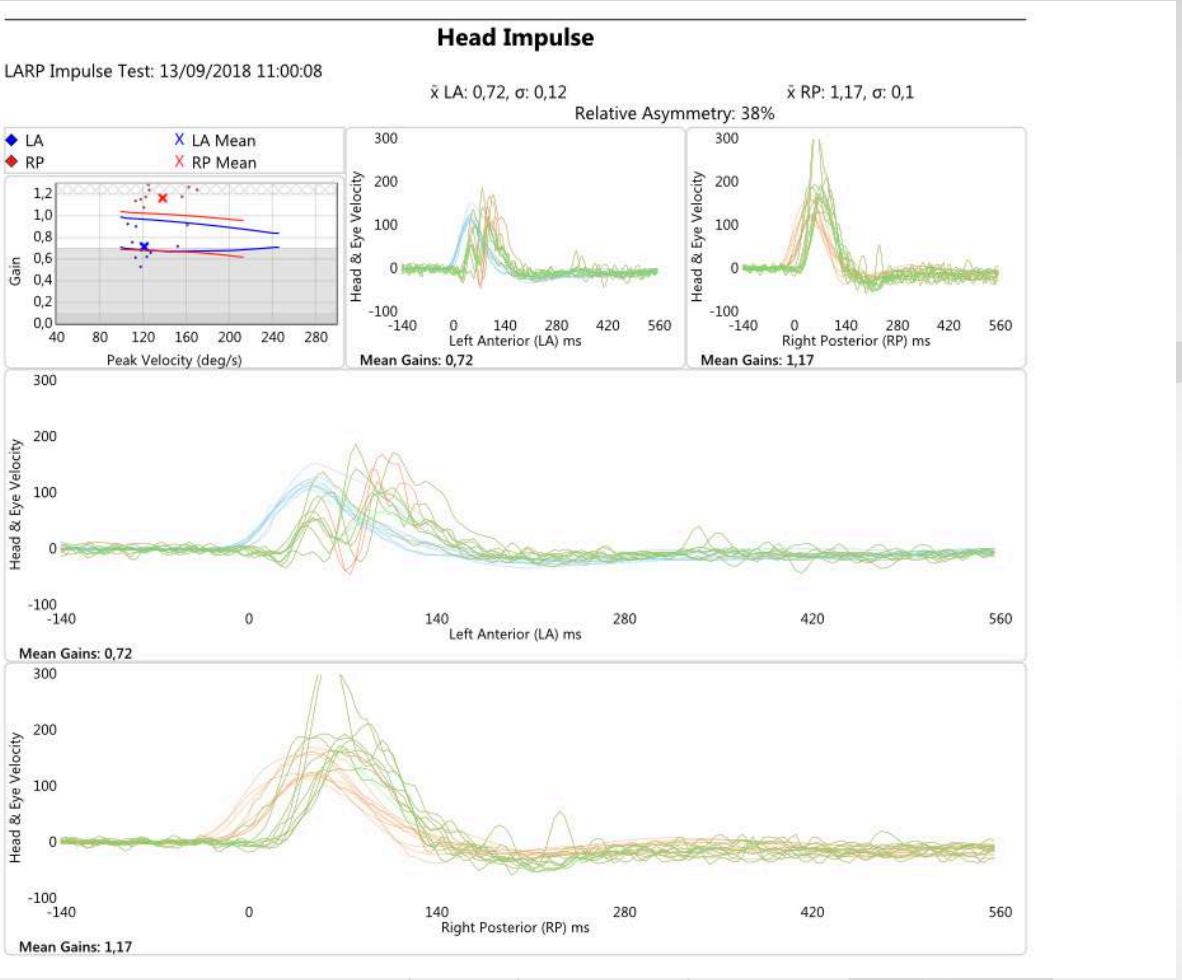


Dopo 20 giorni gain del laterale sinistro a 0.56, gain dell' anteriore sinistro a 0.67





Dopo due mesi gain del laterale sinistro a 0.64



Dopo due mesi gain dell' anteriore sinistro a 0.72

Dopo due mesi Ocular Vemps a destra –risposta crociata della via utricolare di sinistra- ancora assenti

Brain (1996), **119**, 755–763

Vestibular neuritis spares the inferior division of the vestibular nerve

M. Fetter and J. Dichgans

Department of Neurology, Eberhard-Karls University, Tübingen, Germany

Correspondence to: M. Fetter, Department of Neurology, Hoppe-Seyler Straße 3, 72076 Tübingen, Germany

Discussion

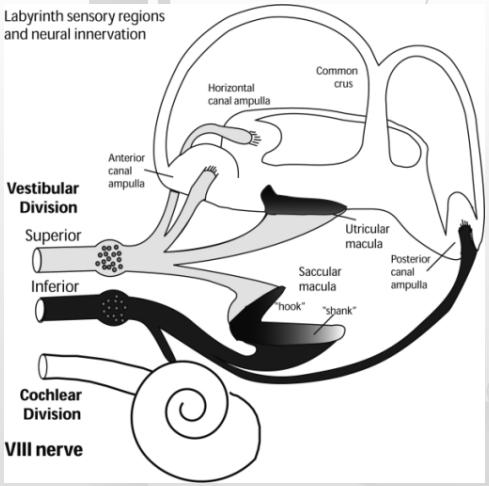
This study uses non-invasive 3D VOR analysis to determine which SCC afferents are involved in acute vestibular neuritis. The analysis of spontaneous nystagmus direction and dynamic asymmetries for stimulation in the different SCC planes suggests that in our patients, the disease involved either the horizontal SCC alone, or, more often, the horizontal plus anterior SCC on one side.

The clinical scenario of a "typical" unilateral acute vestibular neuritis presents lateral and superior canal involvement, i.e. we are talking about a superior vestibular neuritis

Vestibular-evoked myogenic potentials are preserved in two-thirds of the patients with vestibular neuritis.

M. Strupp, 2009

Therefore saccular macula is preserved in 2/3 of cases

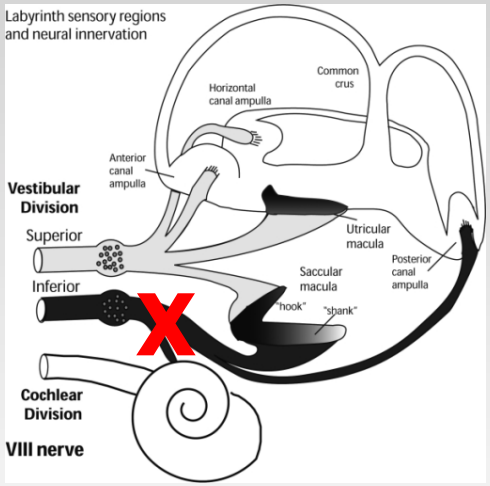


Inferior Vestibular Neuritis

G.M. HALMAGYI, S.T. AW, M. KARLBERG, I.S. CURTHOYS, AND M.J. TODD

Neurology Department, Royal Prince Alfred Hospital, Camperdown, NSW 2050, Sydney, Australia

ABSTRACT: Sudden, spontaneous, unilateral loss of vestibular function without simultaneous hearing loss or brain stem signs is generally attributed to a viral infection involving the vestibular nerve and is called acute vestibular neuritis. The clinical hallmarks of acute vestibular neuritis are vertigo, spontaneous nystagmus, and unilateral loss of lateral semicircular function as shown by impulsive and caloric testing. In some patients with vestibular neuritis the process appears to involve only anterior and lateral semicircular function, and these patients are considered to have selective *superior* vestibular neuritis. Here we report on two patients with acute vertigo, normal lateral semicircular canal function as shown by both impulsive and caloric testing, but selective loss of posterior semicircular canal function as shown by impulsive testing and of saccular function as shown by vestibular evoked myogenic potential testing. We suggest that these patients had selective *inferior* vestibular neuritis and that contrary to conventional teaching, in a patient with acute spontaneous vertigo, unilateral loss of lateral semicircular canal function is not essential for a diagnosis of acute vestibular neuritis.



Normal:

Lateral and anterior canal HTT

Caloric test

Abnormal on the same side:

C-Vemp (saccular failure)

Posterior canal HTT

BMC Neurology 2006, 6:4 BMC Neurology 2006, 6:45 doi:10.1186/1471-2377-6-45

BMC Neurology

BioMed Central

Case report

Open Access

Inferior vestibular neuritis: 3 cases with clinical features of acute vestibular neuritis, normal calorics but indications of saccular failure

Per Monstad*[1], Siri Økstad[2] and Åse Mygland[1]

Abstract

Background: Vestibular neuritis (VN) is commonly diagnosed by demonstration of unilateral vestibular failure, as unilateral loss of caloric response. As this test reflects the function of the superior part of the vestibular nerve only, cases of pure inferior nerve neuritis will be lost.

Case presentations: We describe three patients with symptoms suggestive of VN, but normal calorics. All 3 had unilateral loss of vestibular evoked myogenic potential. A slight, asymptomatic position dependent nystagmus, with the pathological ear down, was observed.

Conclusion: We believe that these patients suffer from pure inferior nerve vestibular neuritis.

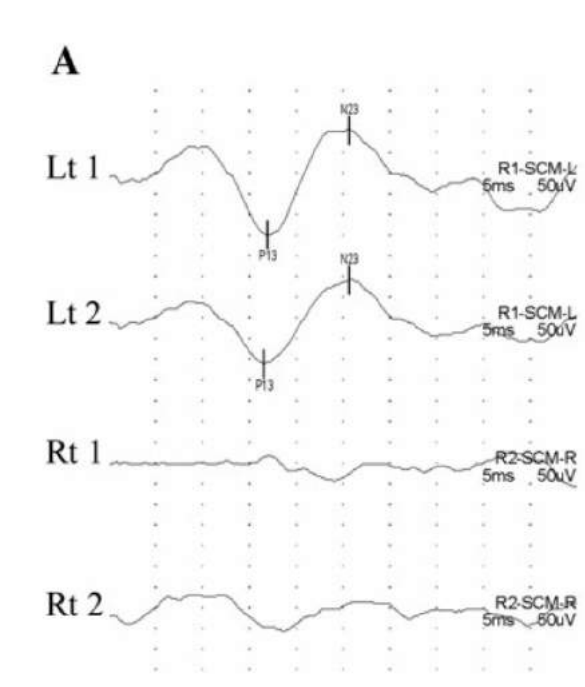
is difficult to understand, however. Saccular dysfunction causes an asymmetric failure of the velocity-storage mechanism [19]. Loss of otolithic input from the utricle, after surgical lesion of the utricular nerve in cats, has been shown to result in positional nystagmus with the lesioned ear down [20]. Our patients had probably normal utricular function, as this organ is innervated by the superior vestibular nerve. Only pat 1 was tested by subjective visual vertical, a test of utricular function, this was found to be normal. A similar experiment with lesion of the sacculus has not been done, to our knowledge.

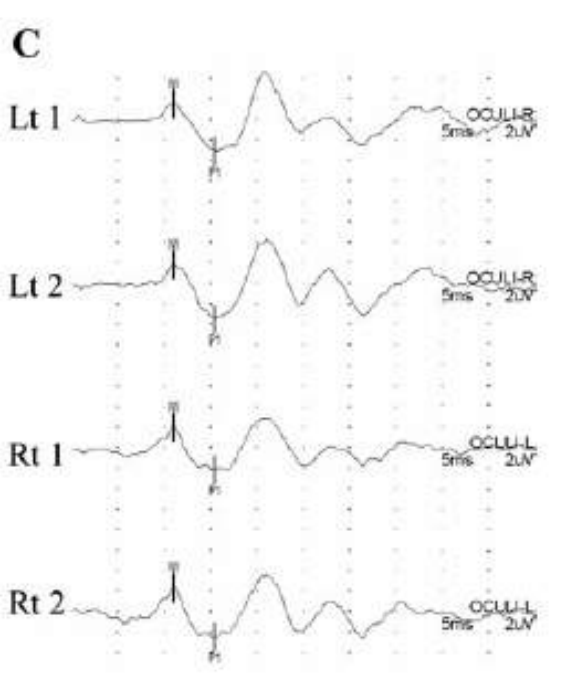
J Neurol (2012) 259:1553–1560
DOI 10.1007/s00415-011-6375-4

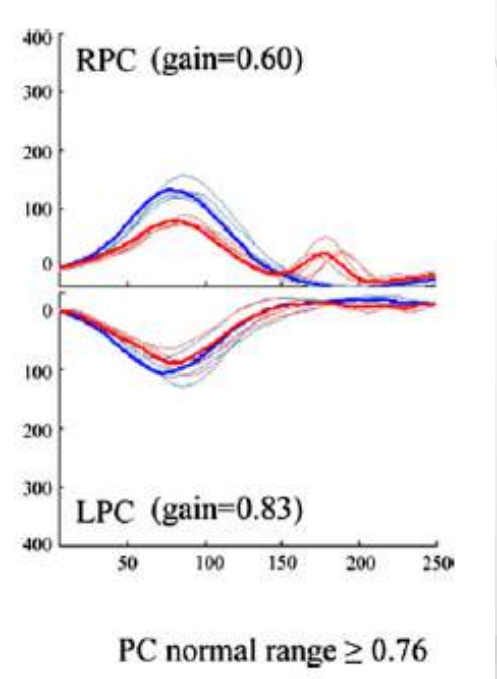
ORIGINAL COMMUNICATION

Inferior vestibular neuritis

Ji-Soo Kim · Hyo Jung Kim







J Neurol (2012) 259:1553–1560
DOI 10.1007/s00415-011-6375-4

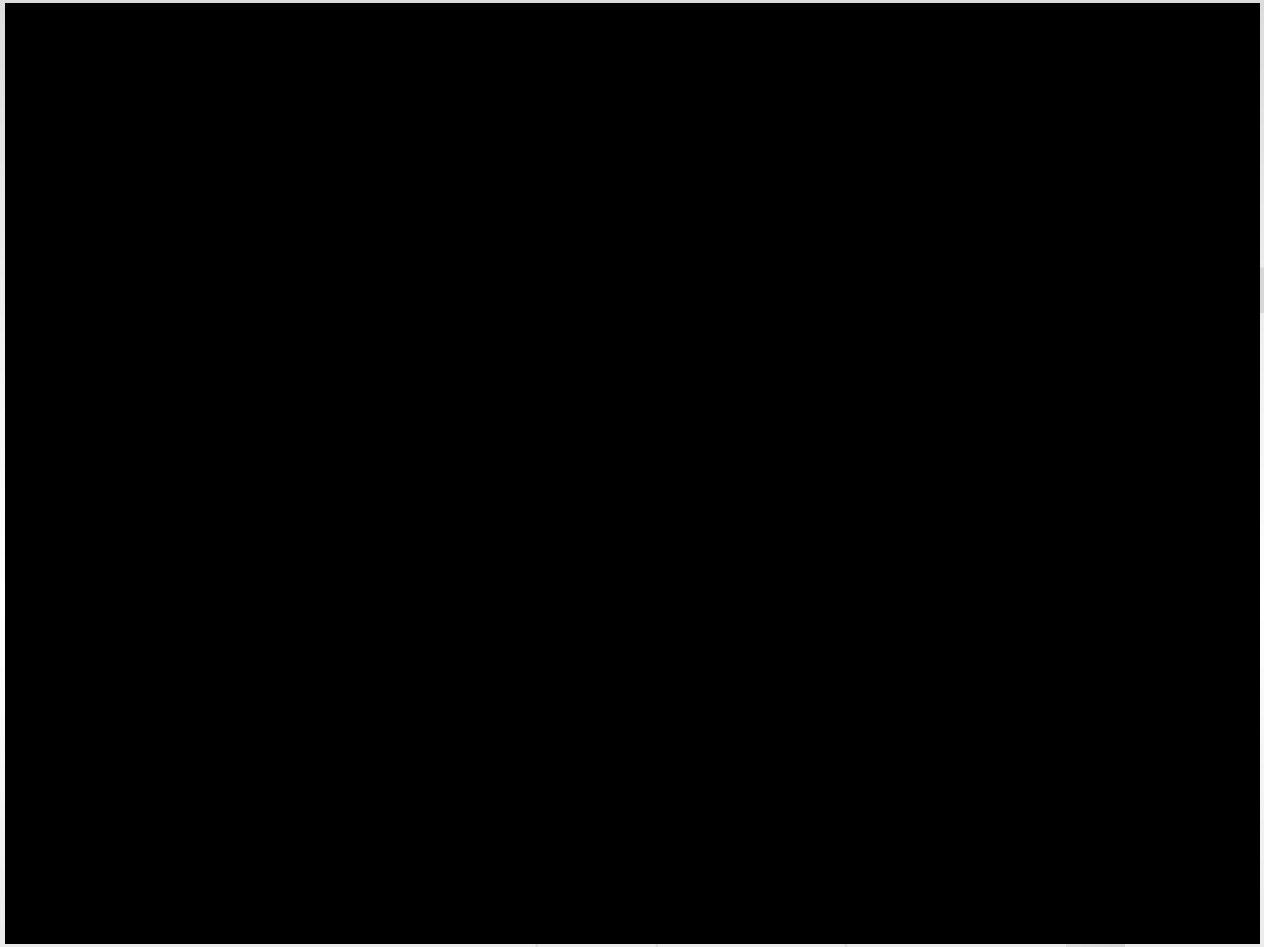
ORIGINAL COMMUNICATION

Inferior vestibular neuritis

Ji-Soo Kim · Hyo Jung Kim

Table 2 Diagnostic criteria of isolated inferior vestibular neuritis

1	Acute vertigo with nausea/vomiting and imbalance
2	Spontaneous torsional downbeat nystagmus
3	Abnormal head impulse test for the posterior semicircular canal
4	Abnormal cervical vestibular-evoked myogenic potential
5	Normal head impulse tests for the anterior and horizontal semicircular canals
6	Normal calorics
7	Exclusion of central pathologies using neurological examination and brain MRI

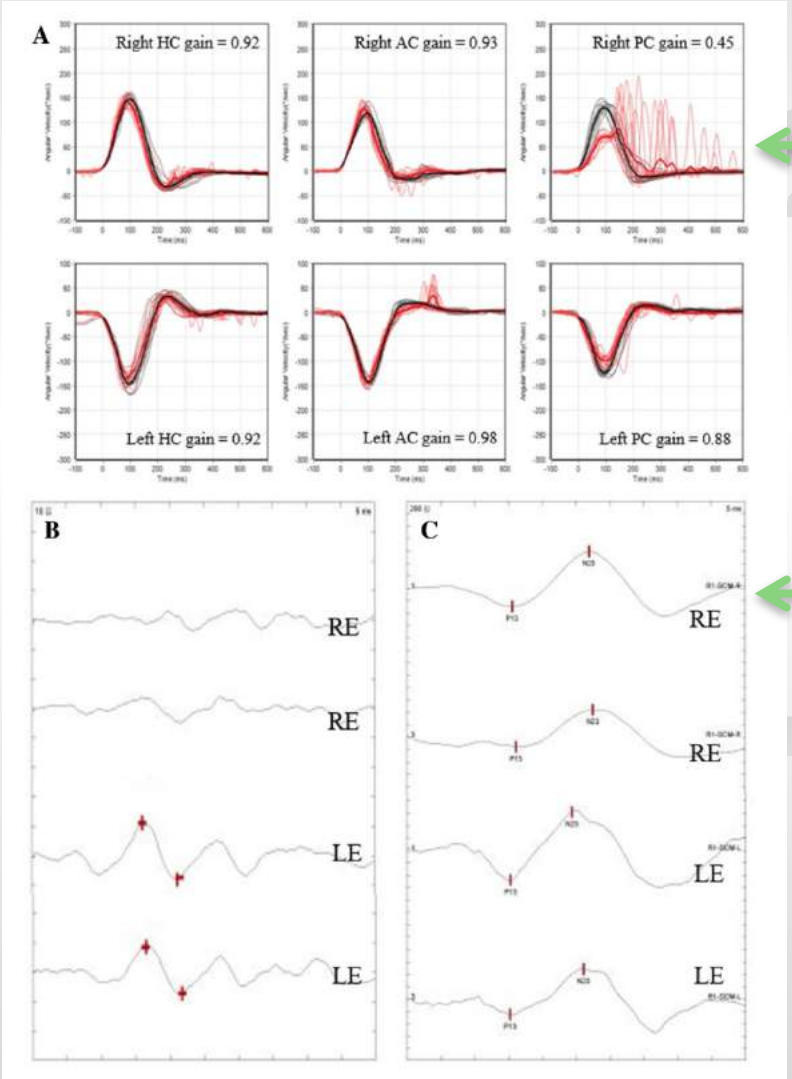


Sintomatico
Piccolo nistagmo downbeat torsionale
Danno utricolare a destra (cervical VEMP non evocabile)
(Canale semicircolare posteriore all' epoca non valutabile)

Vestibular neuritis selectively involving posterior canal and utricle

Ji-Yun Park[1] · Seo Young Choi[2] · Jae-Hwan Choi[3] · Kwang-Dong Choi[2]

Received: 28 May 2018 / Revised: 18 June 2018 / Accepted: 20 June 2018

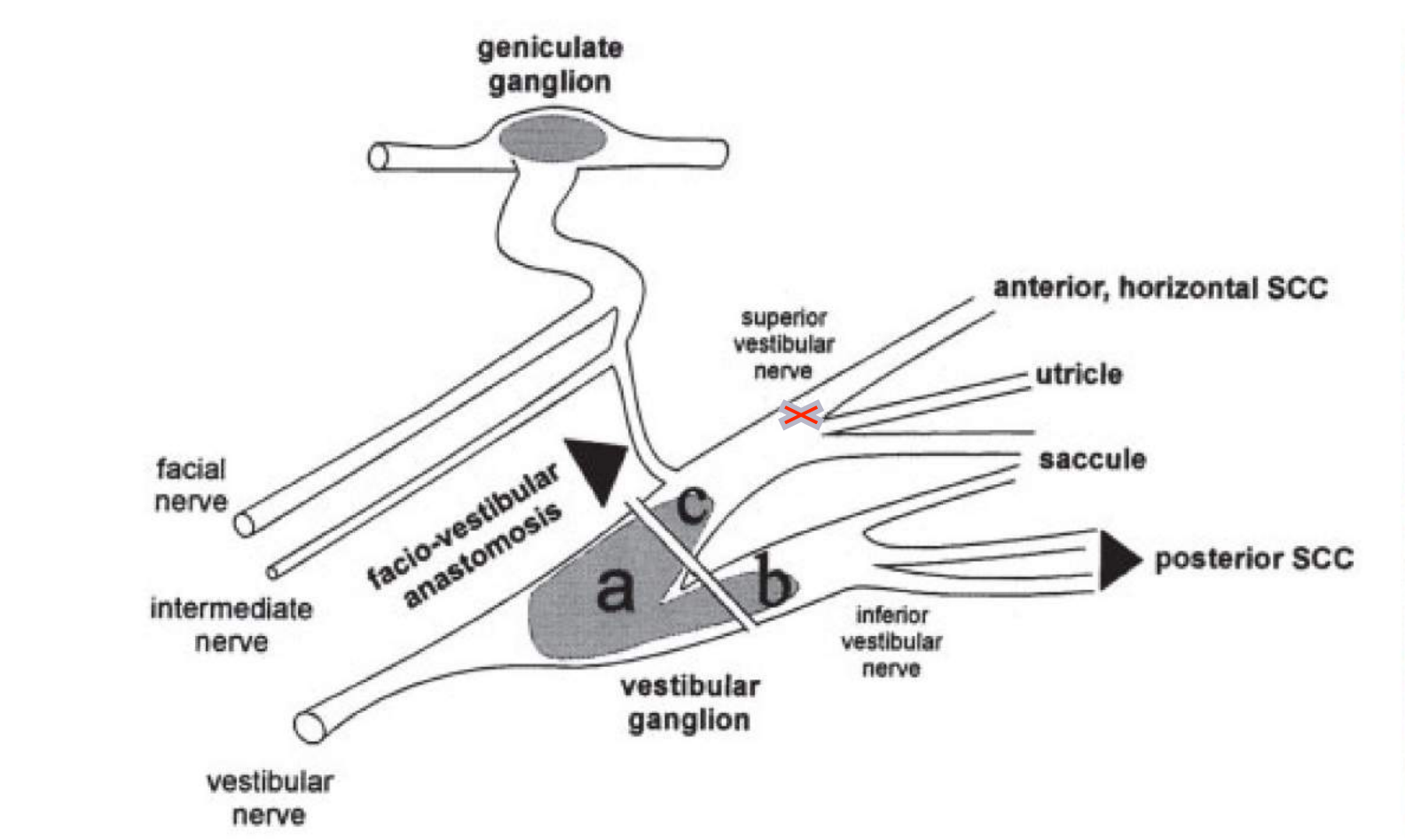


Deficit selettivo del csp destro

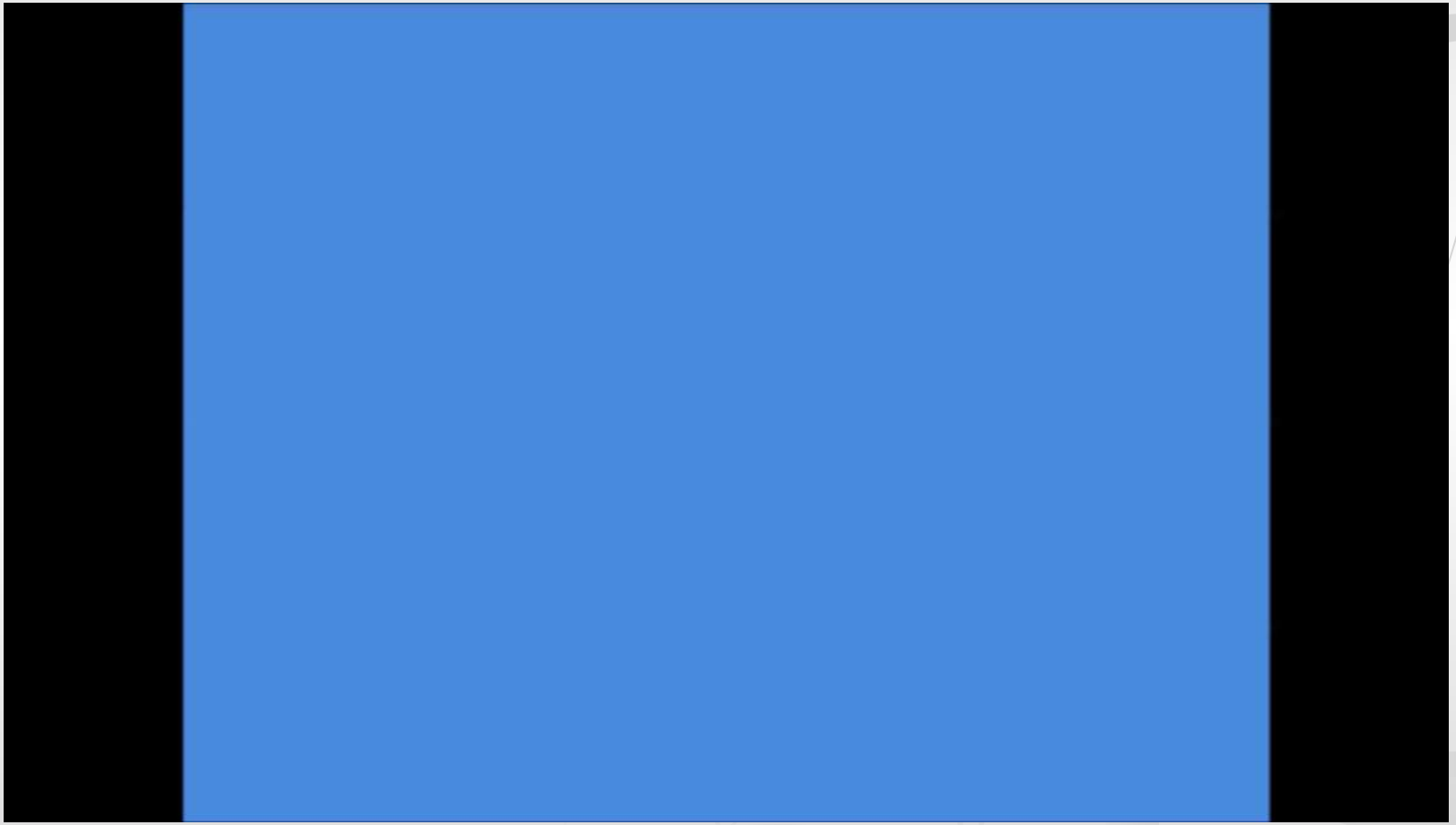
Deficit selettivo dell' utriculo destro

Sacculo destro attivo

Deficit selettivo non «paradigmatico» del nervo vestibolare superiore di sinistra



DVA acuto a sinistra



Uphill/downhill nystagmus

Nistagmo in salita e nistagmo in discesa

M. GUFONI

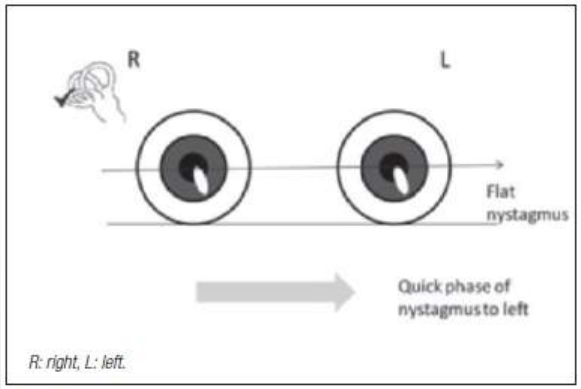


Fig. 3. The right lateral semicircular canal is damaged and a spontaneous horizontal nystagmus arises directed to the left, but the right utriculus is spared. As a result, the plane on which nystagmus beats is horizontal ("flat" nystagmus).

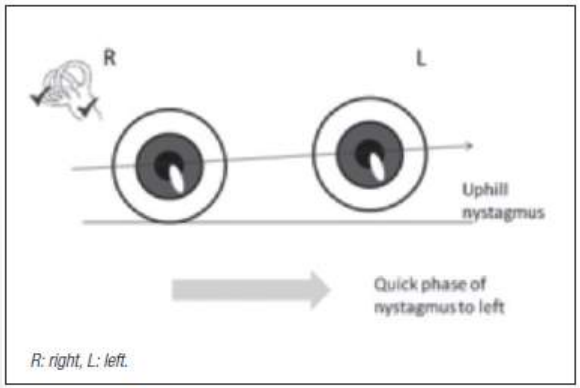


Fig. 1. The right lateral semicircular canal is damaged and a spontaneous horizontal nystagmus arises to the left. At the same time, right utriculus is injured (by the same disease) and an ocular tilt reaction is present, with hypotropia of the right eye. As a result, the plane on which nystagmus beats is inclined upward ("uphill").

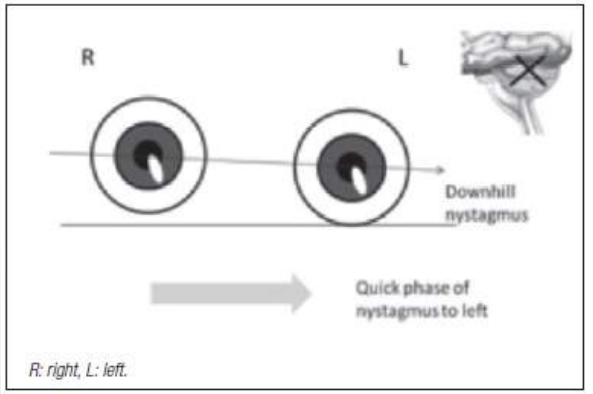
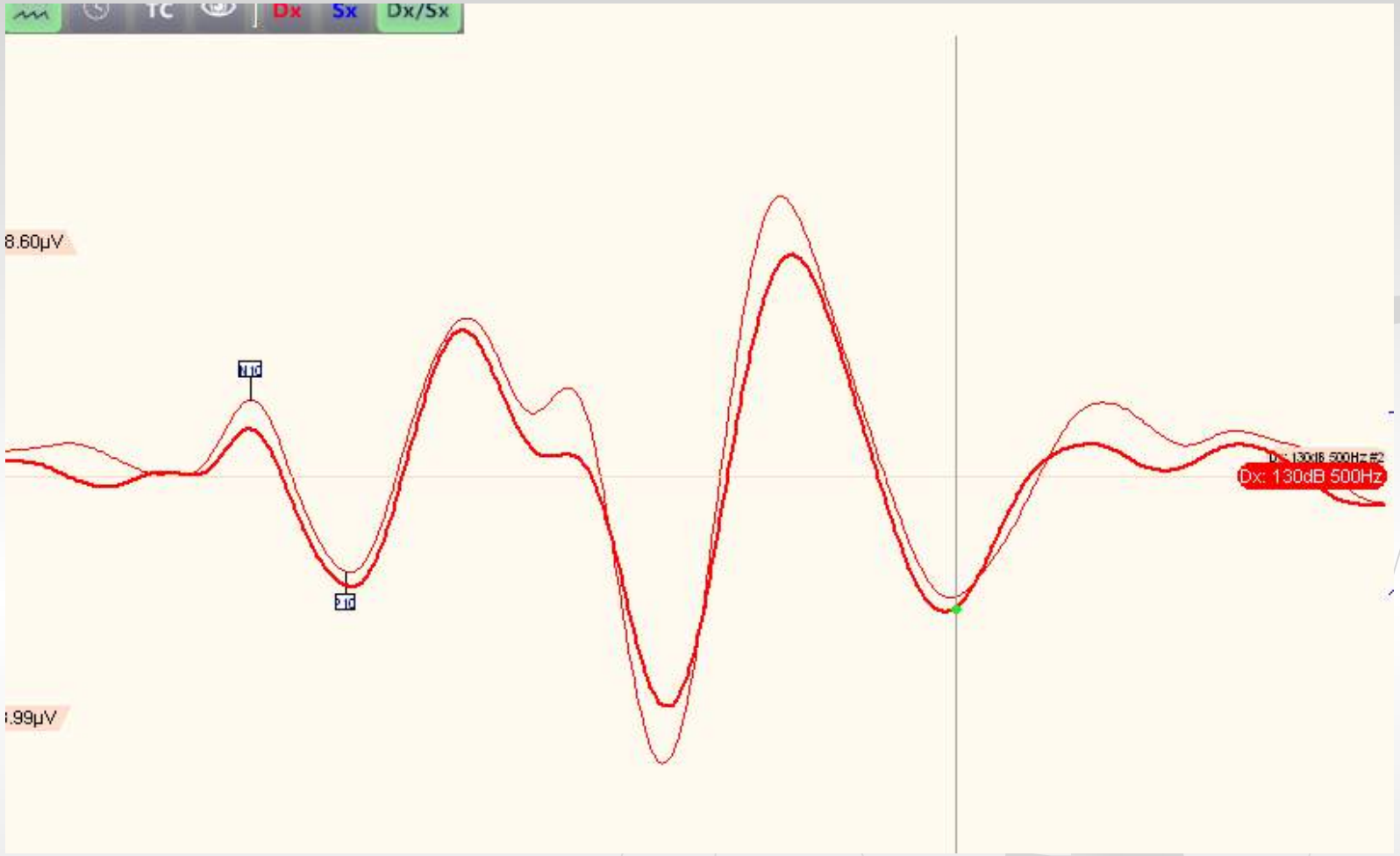
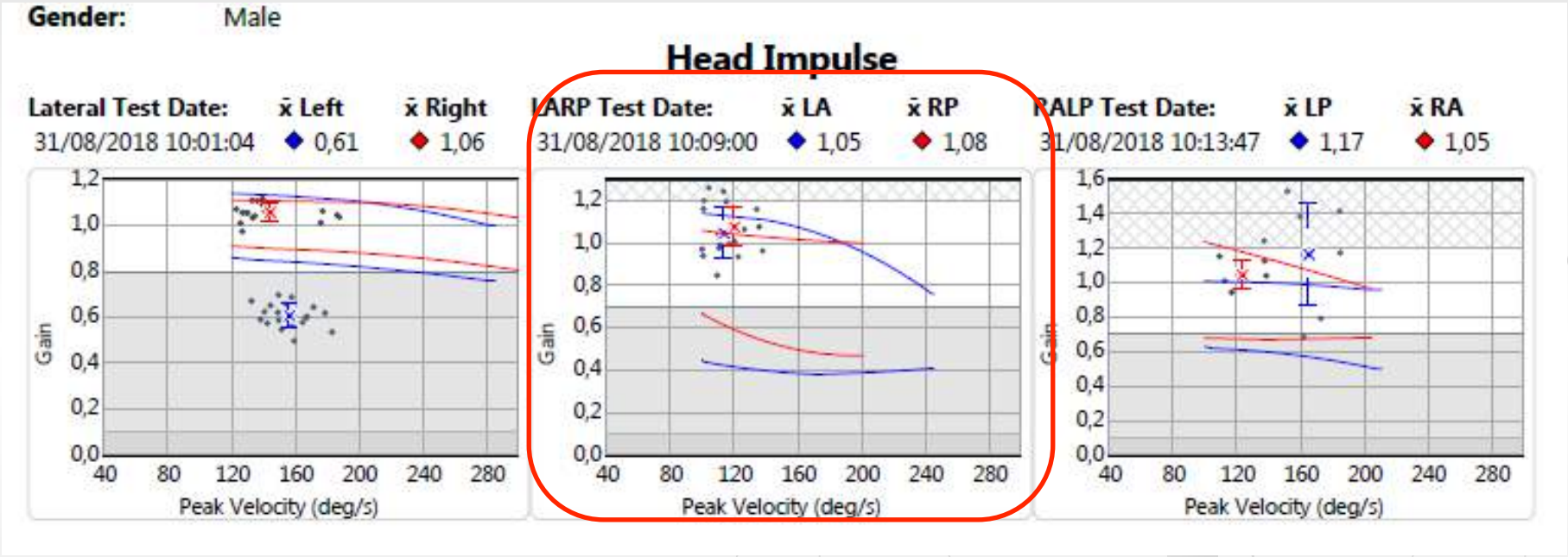


Fig. 2. A lesion is present after decussation of the utricular afferent fibres. Ocular tilt reaction is referred to the opposite side. The (central) resulting nystagmus is tilted "downhill".

Assenza di strabismo verticale



Ocular VEMP evocabile da sinistra : via utricolare sinistra conservata



Riduzione del gain del canale laterale di sinistra
Gain del canale anteriore di sinistra nella norma

Quadro clinico di deficit vestibolare acuto sinistro: vertigine acuta, nistagmo spontaneo destro

- Normoreflessia calorica a sinistra.
- Gain del VOR del canale laterale sinistro 0.61
- Gain del VOR del canale anteriore sinistro 1.05
- Ocular Vemp PRESENTE da sinistra *(utriculo attivo)*
- Cervical VEMP di sinistra presente *(sacculo attivo)*

ACTA OTO-LARYNGOLOGICA
https://doi.org/10.1080/00016489.2018.1481523

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RESEARCH ARTICLE

Check for updates

Changes of video head impulse test results in lateral semicircular canal plane by different peak head velocities in patients with vestibular neuritis

Tae Su Kim[a]*, Hyun Woo Lim[b]*, Chan Joo Yang[c], Yong Han Kim[d], Woo Ri Choi[d], Yeh Ree Kim[d], Jun Woo Park[d], Byung Chul Kang[e] and Hong Ju Park[d]

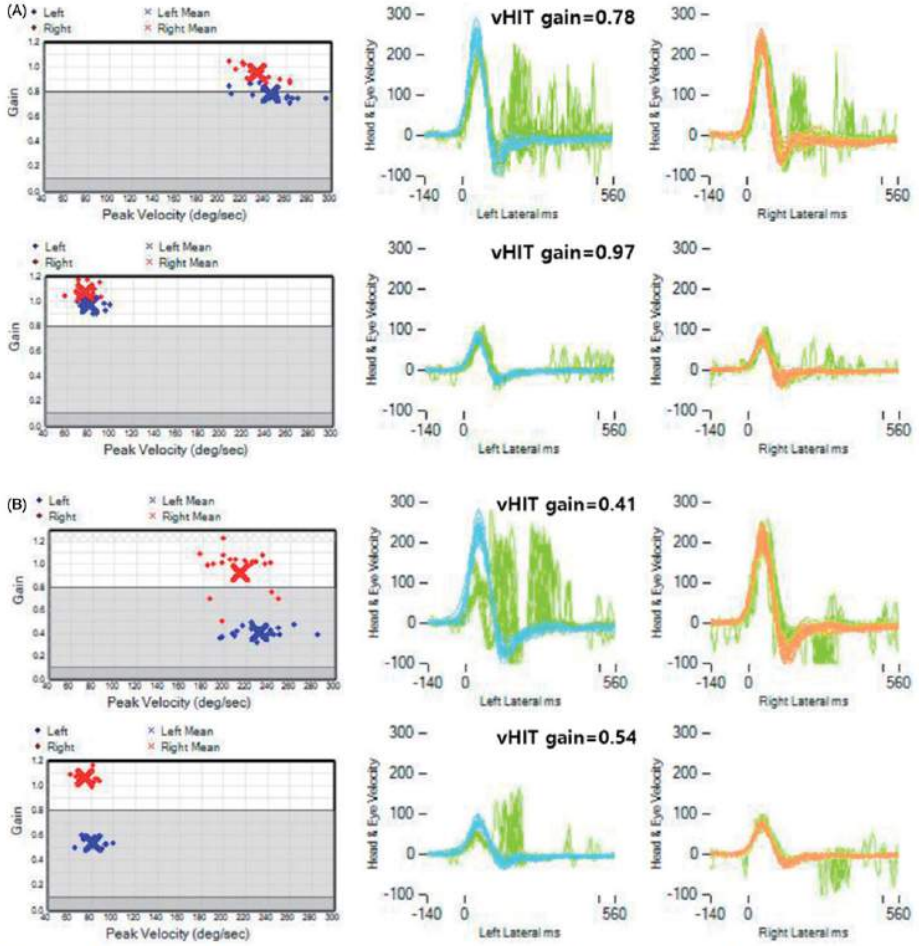


Figure 1. Results of vHIT testing at two different head impulse peak velocities in patients with left vestibular neuritis. A. In the high-peak velocity (mean 245°/s) vHIT (upper panel), the vHIT gain (0.78) and peak velocity (210°/s) of the corrective saccades (CS) was abnormal, but this was within normal limits in the low-peak velocity (mean 80°/s) vHIT (lower panel). B. The vHIT gains and large CSs were found to be abnormal at two different peak head velocities.

E' necessario saturare la componente inibitoria del canale laterale controlaterale (nonché eventuali impulsi propriocettivi cervicali e visivi compensatori) per ottenere la migliora risposta compensatoria (saccadi) e la più sensisbile quantificazione del gain canalare

The peak angular velocity of the head impulses conducted by the examiner is critical when performing vHIT, because head impulses at a high acceleration can better reveal VOR deficits and elicited a larger CS in patients with unilateral vestibular hypofunction [5]. In our present study, vHIT gain decreased and covert and overt CSs occurred more frequently and their peak velocities increased when the mean peak head impulse velocity increased from 80°/s to 240°/s, resulting in higher abnormal rate detection by 17–20% using the high-peak head velocity vHIT. Our findings show the clinical evidence for the superiority of high-peak velocity compared to low-peak velocity head impulses.

The benefit and need for high-peak head impulse velocities can be explained as follows [10]. If the head is rotated slowly to the left, then the counter-rotation of the eyes to the right will be the result of visual, vestibular, and cervical reflexes. If the head is rotated quickly then visual and cervical reflexes cannot respond quickly enough, so that the compensatory counter-rotation of the eyes to the right will be produced exclusively by VOR. Furthermore, if the head rotation is very quick then vestibular afferents from the right lateral SCC will be driven into inhibitory saturation and the VOR will be produced predominantly by afferents from the left lateral SCC. If the left lateral SCC is impaired (low VOR

Eur Arch Otorhinolaryngol
DOI 10.1007/s00405-016-4055-9

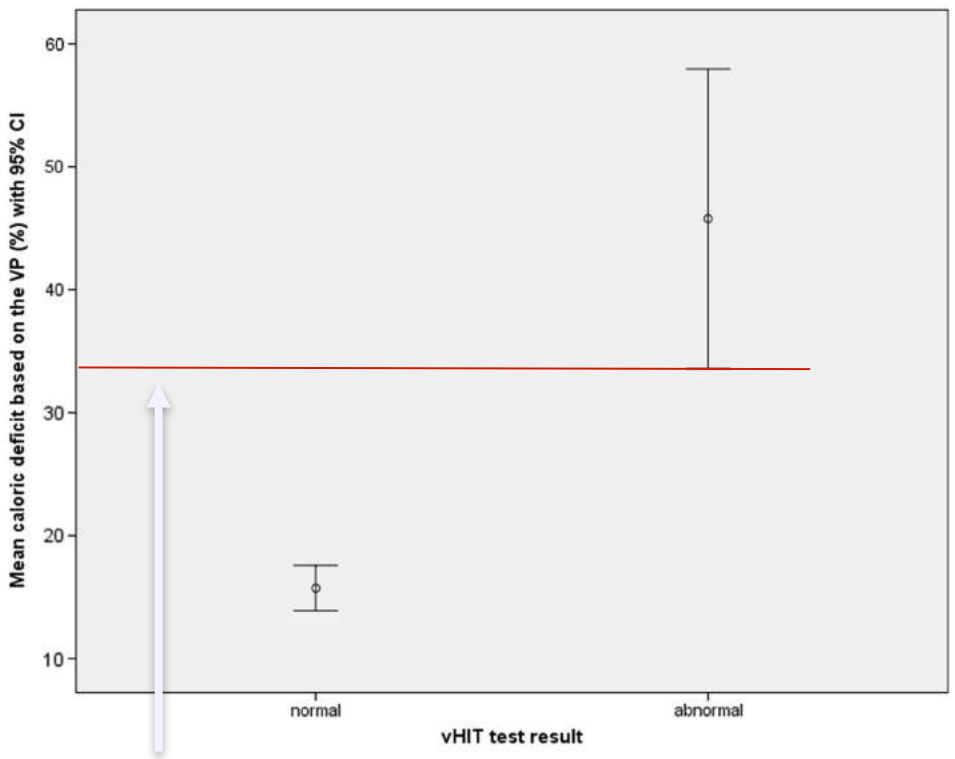
CrossMark

OTOLOGY

Determining vestibular hypofunction: start with the video-head impulse test

B. F. van Esch[1] · G. E. A. J. Nobel-Hoff[2] · P. P. G. van Benthem[3] · H. J. van der Zaag-Loonen[1] · Tj. D. Bruintjes[1]

Fig. 3 Mean canal paresis deficit (based on the vestibular preponderance) as a function of the vHIT test result. Data represent mean and corresponding 95 % confidence intervals



Il V-HIT sembra abbassare notevolmente il livello di paresi calorica con cui si appaia il deficit alle frequenze di stimolo più alte

La possibilità dello studio di tutti e cinque i recettori canalari ha rotto la vecchia schematica distinzione tra «deficit vestibolare acuto superiore vs. inferiore»

Possibili

- deficit globali
- deficit «paradigmatici» (superiore vs inferiore)
- deficit “non paradigmatici”
- deficit «frequenziali» selettivi» (a-VOR ad alta frequenza-VHIT-vs. calorici)

The extent of vestibular impairment is important in recovery of canal paresis of patients with vestibular neuritis

Kyurin Hwang [a], Bo Gyung Kim [b], Jong Dae Lee [b,*], Eek-Sung Lee [c], Tae Kyeong Lee [c], Ki-Bum Sung [c]

Our data suggest that lesions involving both the superior and inferior branch may show poor functional recovery. That is, patients with VN with superior and inferior vestibular nerve involvement showed poor recovery in the caloric test than SVN patients. Although initial CP value was similar between two groups, we speculate that inflammatory response of VN with superior and inferior vestibular nerve involvement may be more severe and it may affect the caloric recovery resulting from a greater degree of nerve atrophy.

CME

Low recurrence rate of vestibular neuritis: A long-term follow-up

Abstract—We examined 103 patients with vestibular neuritis (VN) in a follow-up study (5.7 to 20.5 years, mean 9.8 years). Two patients (1.9%) had developed a second occurrence of VN 29 to 39 months after the first. VN affected the contralateral ear in both and caused less severe distressing vertigo and postural imbalance. Unlike Bell's palsy and sudden hearing loss, a relapse in the same ear did not occur.

NEUROLOGY 2006;67:1870–1871

D. Huppert, MD; M. Strupp, MD; D. Theil, DVM; M. Glaser; and T. Brandt, MD, FRCP

Discussion. In this long-term follow-up, the recurrence rate of VN amounted to 1.9% in the ear contralateral to the initial manifestation, and there was no single relapse in the originally affected ear. Compared with the annual frequency of VN in the normal population of 3.5 per 100,000,[5] the frequency of recurrence is considerably higher (odds ratio OR 55).

testing after VN (unpublished). The rate of permanent paresis of the eighth nerve is considerably higher than in patients with Bell's palsy, in which severe, permanent palsy occurs in 4% and mild or moderate in 13%.[10] This may be due to axonal sprouting of the facial nerve. Thus, a relapse may go undetected in those with a persistent and complete unilateral vestibular deficit; however, it should become symptomatic in those with an incomplete unilateral deficit.

La recidiva controlaterale è in circa il 2% dei casi. Le recidive ipsilaterali possono essere rese meno evidenti sintomatologicamente dal pregresso danno vestibolare

Vestibular Neuritis

Michael Strupp, M.D.,[1] and Thomas Brandt[2Q1]

MANAGEMENT

The management of vestibular neuritis involves (1) symptomatic treatment with antivertiginous drugs (e.g., dimenhydrinate, scopolamine) to attenuate vertigo, dizziness, and nausea/vomiting; (2) causal treatment with corticosteroids to improve recovery of peripheral vestibular function; and (3) physical therapy (vestibular exercises and balance training) to improve central vestibular compensation.[69]

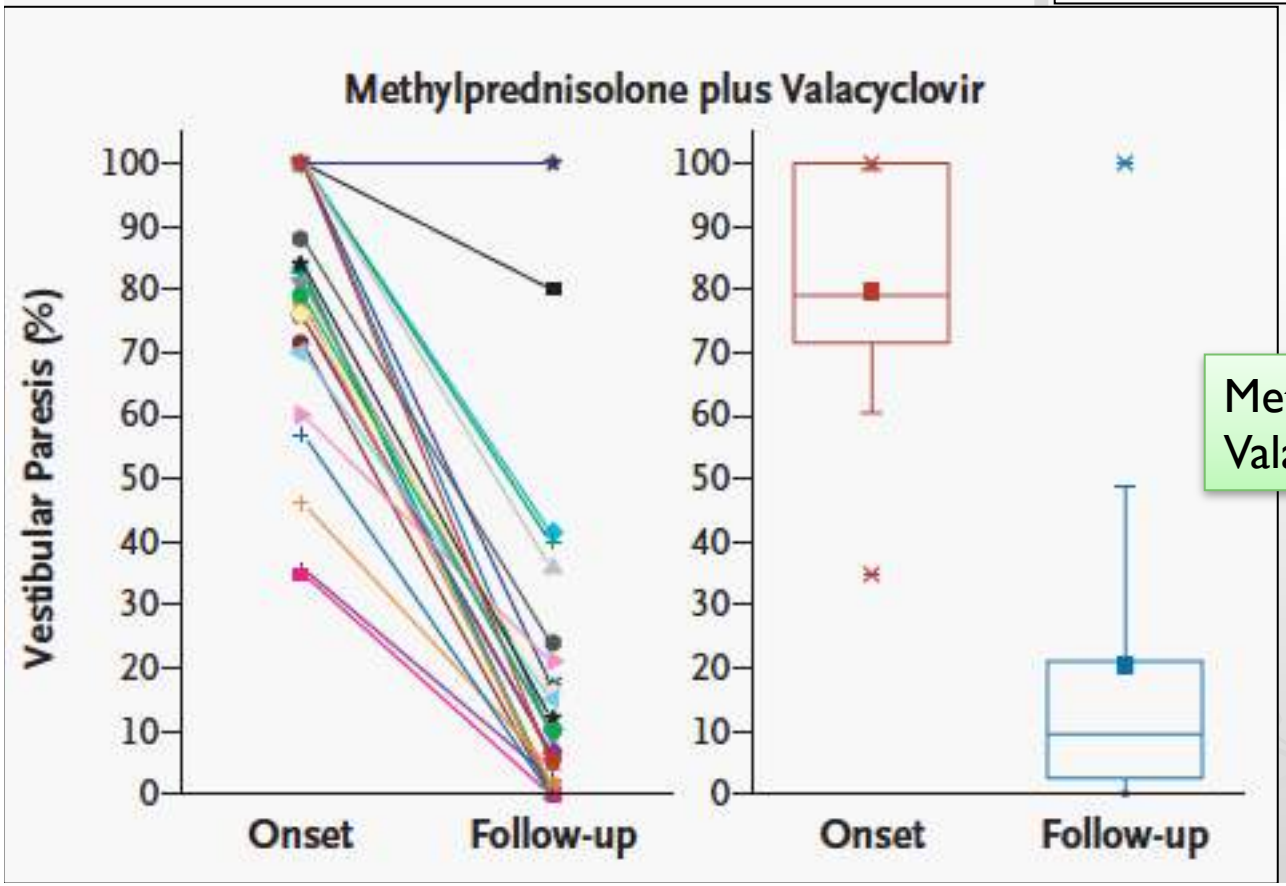
La terapia medica

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Methylprednisolone, Valacyclovir, or the Combination for Vestibular Neuritis

Michael Strupp, M.D., Vera Carina Zingler, M.D., Viktor Arbusow, M.D., Daniel Niklas, Klaus Peter Maag, M.D., Ph.D., Marianne Dieterich, M.D., Sandra Bense, M.D., Diethilde Theil, D.V.M., Klaus Jahn, M.D., and Thomas Brandt, M.D.



Metilprednisolone 60 mg/die
Valacyclovir 1000 mg X 3/die

Vestibular Neuritis

Michael Strupp, M.D.,[1] and Thomas Brandt[2Q1]

SEMINARS IN NEUROLOGY/VOLUME 29, NUMBER 5 2009

and valacyclovir (Fig. 7).[72] Glucocorticoids (methylprednisolone) should be given within 3 days after symptom onset and for 3 weeks (initially 100 mg/day and then tapered off by 20 mg every 3 days). As in Bell palsy, the benefit of steroids might be due to their antiinflammatory effects, which reduce the swelling and cause a mechanical compression of the vestibular nerve within the temporal bone. Thus, steroids but not antiviral agents can be recommended as a treatment for acute vestibular neuritis, as they cause a significant functional improvement. These findings are also supported by an ongoing trial in Sweden (Michael Karlberg, personal communication). Steroids have been demonstrated to be efficacious in two prospective, randomized, double-blind, placebo-controlled studies on Bell palsy, which is also considered an HSV-1 disorder.[73,74] A recent prospective study in only a small number of patients, however, reported that prednisone therapy might enhance earlier recovery, but it does not improve the long-term prognosis of vestibular neuritis.[75]

Expert Opinion

Medical treatment of vestibular disorders

Thomas Brandt†, Andreas Zwergal & Michael Strupp
Ludwig-Maximilians-University, Department of Neurology, Munich, Germany

[redacted] The antiviral drug did not improve the outcome in patients with vestibular neuritis, despite the assumed viral cause. It is conceivable that replication of HSV-1 in the vestibular ganglia may already have occurred at the time the antiviral drug was initiated – that is, within 3 days after the onset of symptoms. Therefore, this study showed that methylprednisolone alone significantly improves the recovery of peripheral vestibular function in patients with vestibular neuritis, whereas valacyclovir is not required [10].

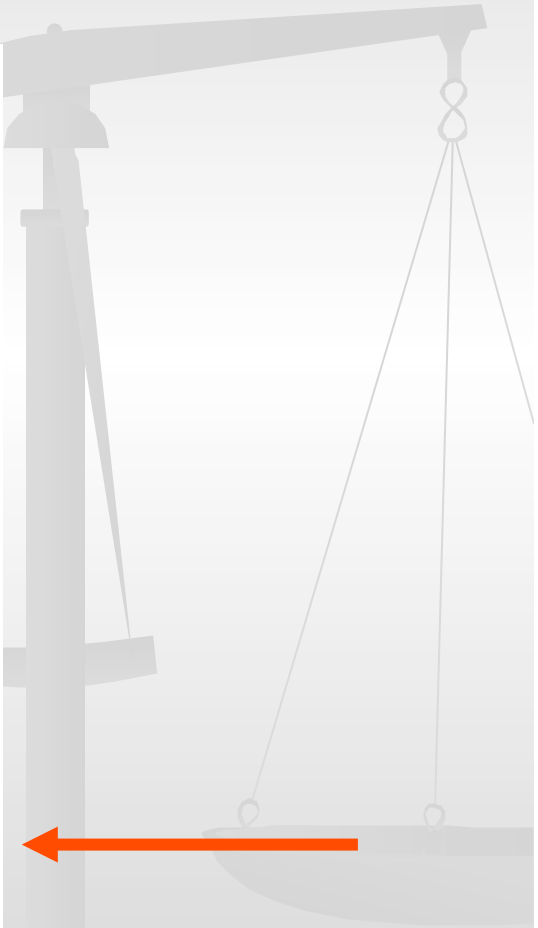
Vestibular Neuritis

Michael Strupp, M.D.,[1] and Thomas Brandt[2Q1]

SEMINARS IN NEUROLOGY/VOLUME 29, NUMBER 5 2009

Symptomatic Treatment

During the first 1 to 3 days, when nausea is pronounced, vestibular sedatives such as antihistamine dimenhydrinate 50 to 100 mg every 6 hours or the anticholinergic scopolamine can be administered. Their major side effect is general sedation. Transdermal application of scopolamine hydrobromide avoids some of the side effects of the conventional means of administration. The most probable sites of primary action are the synapses of the vestibular nuclei, which exhibit a reduced discharge and diminished neural reaction to body rotation. These drugs should not be given for longer than 3 days because they prolong the time required to achieve central compensation.[70,71]



Treatment of Vertigo

RANDY SWARTZ, M.D., *University of California, San Diego, School of Medicine, La Jolla, California*
PAXTON LONGWELL, M.D., *Corpus Christi, Texas*

TABLE 3
Medications Commonly Used In Patients with Acute Vertigo and Associated Nausea and Emesis

Medication	*Dosage*	*Sedation*	*Antiemesis*	*Pregnancy category*
Meclizine* (Antivert)	12.5 to 50 mg orally every 4 to 8 hours	++	+	B
Dimenhydrinate* (Dramamine)	25 to 100 mg orally, IM, or IV every 4 to 8 hours	+	++	B
Diazepam (Valium)	2 to 10 mg orally or IV every 4 to 8 hours	++	+	D
Lorazepam (Ativan)	0.5 to 2 mg orally, IM, or IV every 4 to 8 hours	++	+	D
Metoclopramide (Reglan)	5 to 10 mg orally every 6 hours 5 to 10 mg by slow IV every 6 hours	+	+++	B
Prochlorperazine (Compazine)	5 to 10 mg orally or IM every 6 to 8 hours 25 mg rectally every 12 hours 5 to 10 mg by slow IV over 2 minutes	+	+++	C
Promethazine (Phenergan)	12.5 to 25 mg orally, IM, or rectally every 4 to 12 hours	+++	++	C

**—Available over the counter.*

+ = mild; ++ = moderate; +++ = prominent; IM = intramuscular; IV = intravenous.

Information from references 6 and 7.

Sintomatici

Alla lista aggiungiamo la levosulpiride...

Improvement of vestibular compensation by Levo-Sulpiride in acute unilateral labyrinthine dysfunction

Facilitazione del compenso vestibolare indotto da Levosulpiride nelle lesioni vestibolari acute periferiche

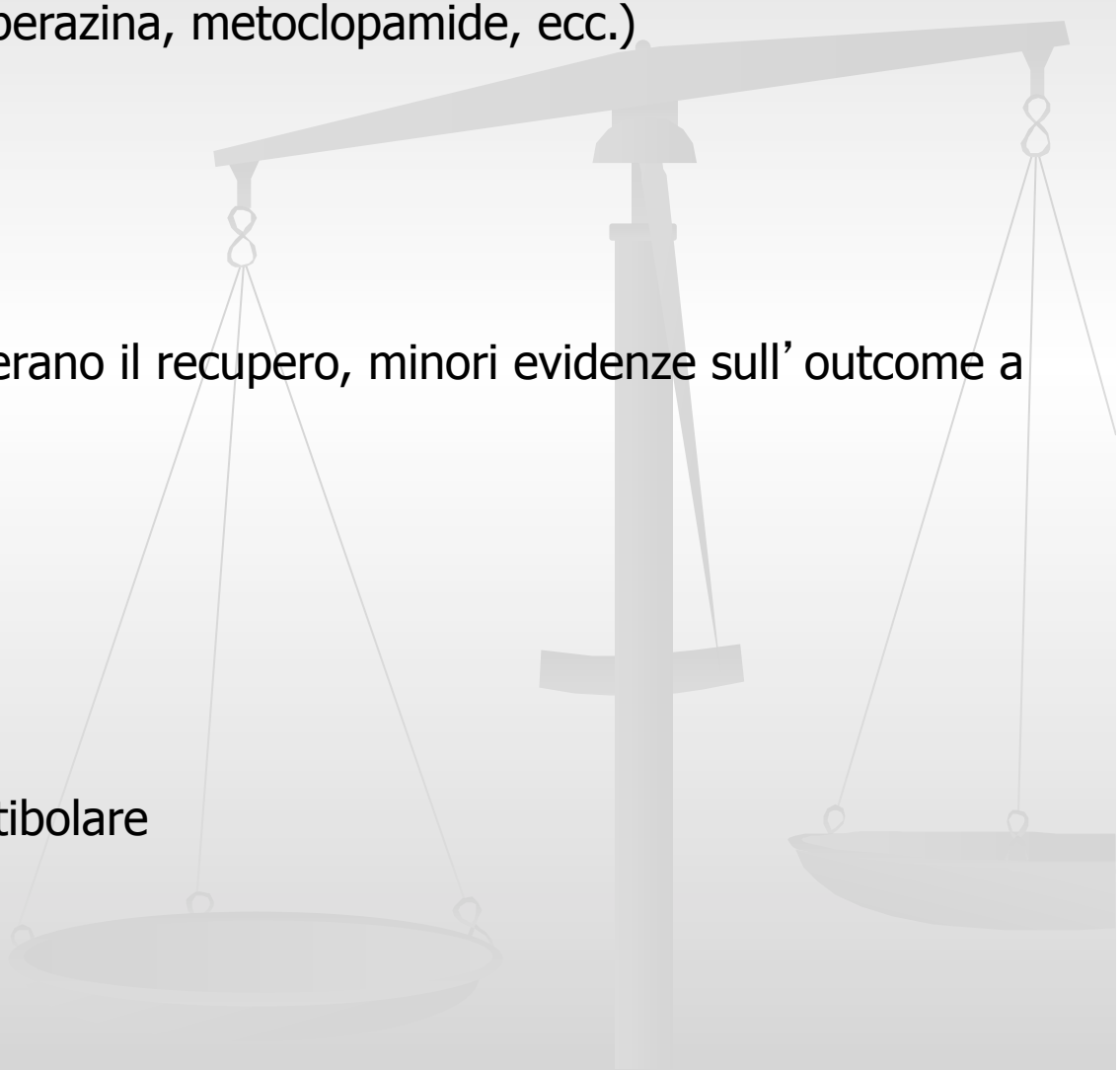
D. ZANETTI, N. CIVIERO, C. BALZANELLI, M. TONINI[1], A.R. ANTONELLI
Otorhinolaryngology Department, University of Brescia; [1] Department of Internal Medicine and Therapy, Section of Pharmacology and Toxicology, University of Pavia, Italy

In this study, L-sulpiride has been found to exert a significant anti-vertiginous action in clinical practice. This action may be derived from enhancement of the inhibitory GABAergic transmission to the MVN, from suppression of the histaminergic transmission at central and vestibular level or from both mechanisms.

Absence of side-effects, at the low doses employed (25 mg t.i.d.), and the induction of fast and steady vestibular compensation strongly recommend its use as the drug of choice for the control of acute vertigo attacks, being more effective than the routinely em-

Uno schema temporal-terapeutico

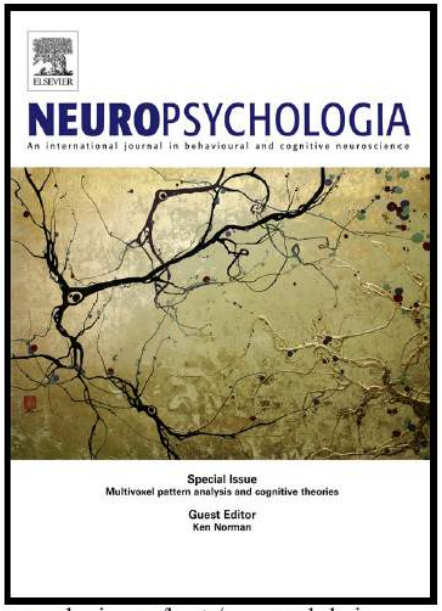
1-3 gg	Vestibulosoppressori, antiemetici, sedativi (Diazepam,tioetilperazina, metoclopamide, ecc.)
1-10 gg	Levosupiride
1-15-30 gg	Cortisonici (accelerano il recupero, minori evidenze sull' outcome a lungo termine)
1-15 gg	Antivirali (???)
30-60 gg	Riabilitazione vestibolare



Author's Accepted Manuscript

Impaired math achievement in patients with acute vestibular neuritis

Ivan Moser, Dominique Vibert, Marco D. Caversaccio, Fred W. Mast



www.elsevier.com/locate/neuropsychologia

Our findings of impaired **math performance** are consistent with the frequent subjective complaints of patients with PVD (Harun et al., 2015). They are also in line with the existing, yet scarce empirical evidence of impaired numerical cognition in vestibular patients (Andersson, Hagman, Talianzadeh, Svedberg, & Larsen, 2003; Bessot, Denise, Toupet, Van Nechel, & Chavoix, 2012; Risey & Briner, 1990; Yardley et al., 2002). Unlike previous studies, we demonstrated impaired arithmetic performance under static conditions. Thus, we provide evidence that previously reported numerical deficits cannot be exclusively attributed to dual-task interference (i.e., impaired cognitive performance due to prioritization of an ongoing postural challenge). This idea is supported by other studies showing various cognitive deficits in PVD in static conditions even though the deficits were more pronounced with an ongoing postural challenge (Redfern et al., 2004; Talkowski et al., 2005; Yardley et al., 2001).

We hypothesize that the observed pattern of arithmetic deficits and normal magnitude processing might be a consequence of metabolic changes in parietal areas during acute PVD. It has been shown that acute PVD leads to a complex activation-deactivation pattern of glucose metabolism. While glucose metabolism is increased in the parieto-insular vestibular cortex, metabolic downregulations are observed in the inferior parietal cortex (Bense et al., 2004;

doi:10.1093/brain/awh617 *Brain* (2005), **128,** 2732–2741

Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans

Thomas Brandt,[1] Franz Schautzer,[2] Derek A. Hamilton,[3] Roland Brüning,[4] Hans J. Markowitsch,[5] Roger Kalla,[1] Cynthia Darlington,[6] Paul Smith[6] and Michael Strupp[1]

HIPPOCAMPUS 17:471–485 (2007)

Spatial Memory and Hippocampal Volume in Humans With Unilateral Vestibular Deafferentation

Katharina Hüfner,[1*] Derek A. Hamilton,[2] Roger Kalla,[1] Thomas Stephan,[1] Stefan Glasauer,[1,3] Jun Ma,[4] Roland Brüning,[4] Hans J. Markowitsch,[5] Kirsten Labudda,[5] Christian Schichor,[6] Michael Strupp,[1] and Thomas Brandt[1,3]

Does vestibular damage cause cognitive dysfunction in humans?

Paul F. Smith[a,*], Yiwen Zheng[a], Arata Horii[b] and Cynthia L. Darlington[a]
[a] *Department of Pharmacology and Toxicology, School of Medical Sciences, University of Otago, Dunedin, New Zealand*
[b] *Department of Otolaryngology, Osaka University Medical School, Japan*

SEMBREREBBE CHE
SIAMO ALLA FINE, MA
PURTROPPO PER VOI
SIAMO SOLO ALL'INIZIO

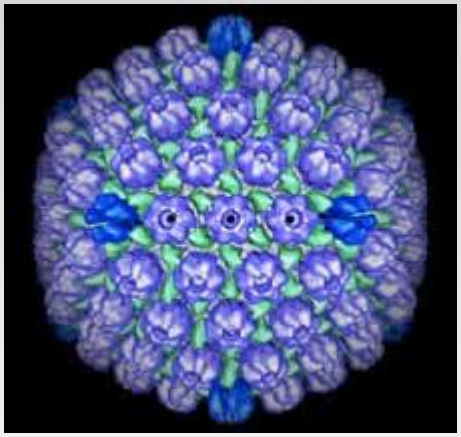
Prednisone Treatment for Vestibular Neuritis

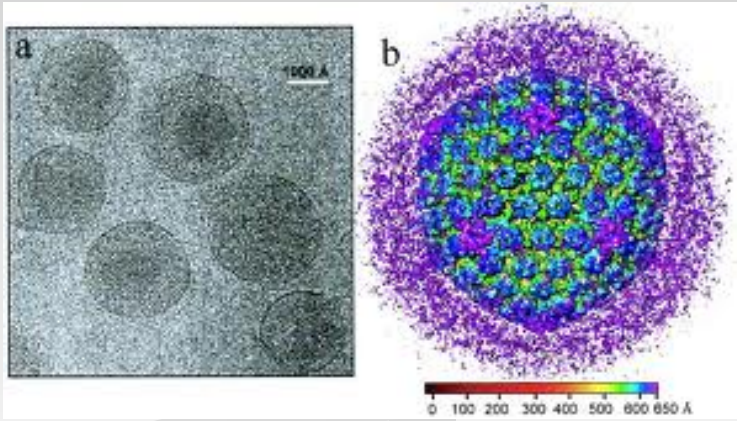
*Avi Shupak, *Anthony Issa, †‡Avishay Golz, *Margalit Kaminer, and ‡§Itzhak Braverman

Vestibular neuritis is considered to have a benign course. The static rotatory vertigo and disequilibrium, present even when the patient is completely at rest, subside in most patients within a few days, and a gradual return to daily activities is the rule. However, it has been shown that there is generally incomplete restoration of peripheral function, and clinical recovery is achieved via proprioceptive and visual substitution for the unilateral vestibular deficit combined with central vestibular compensation of the imbalance in vestibular tone (6,7).

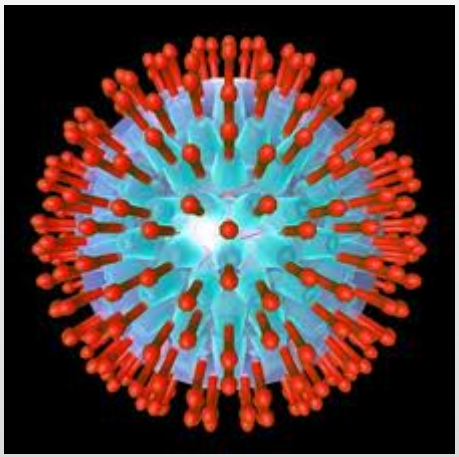
La neurite vestibolare non è quindi una patologia che debba farci paura….

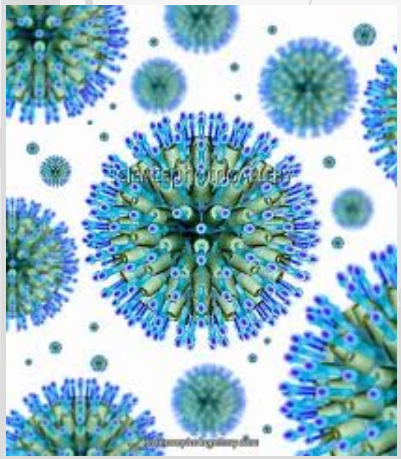
…. ***a condizione che siamo sicuri che sia effettivamente una neurite vestibolare!!!***





Sempre e solo Virus?





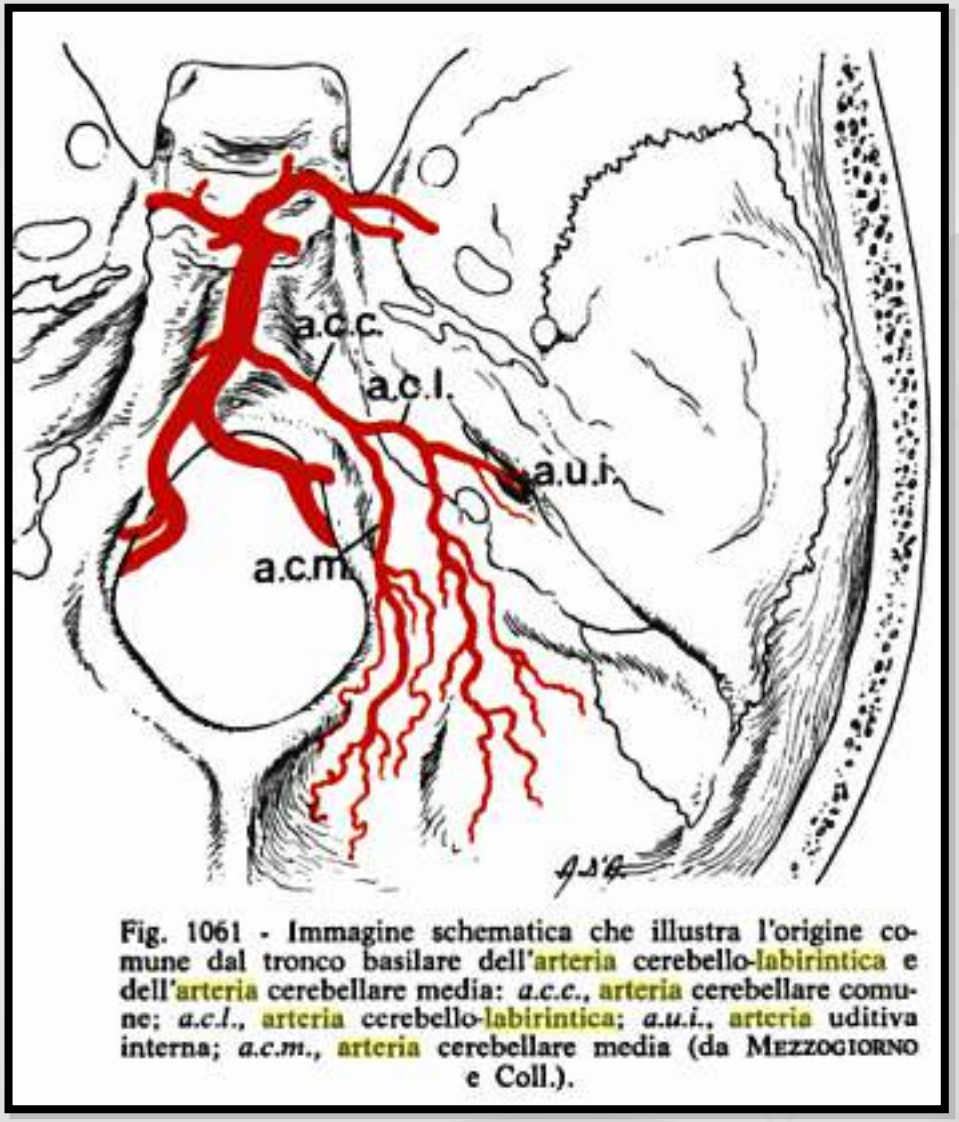
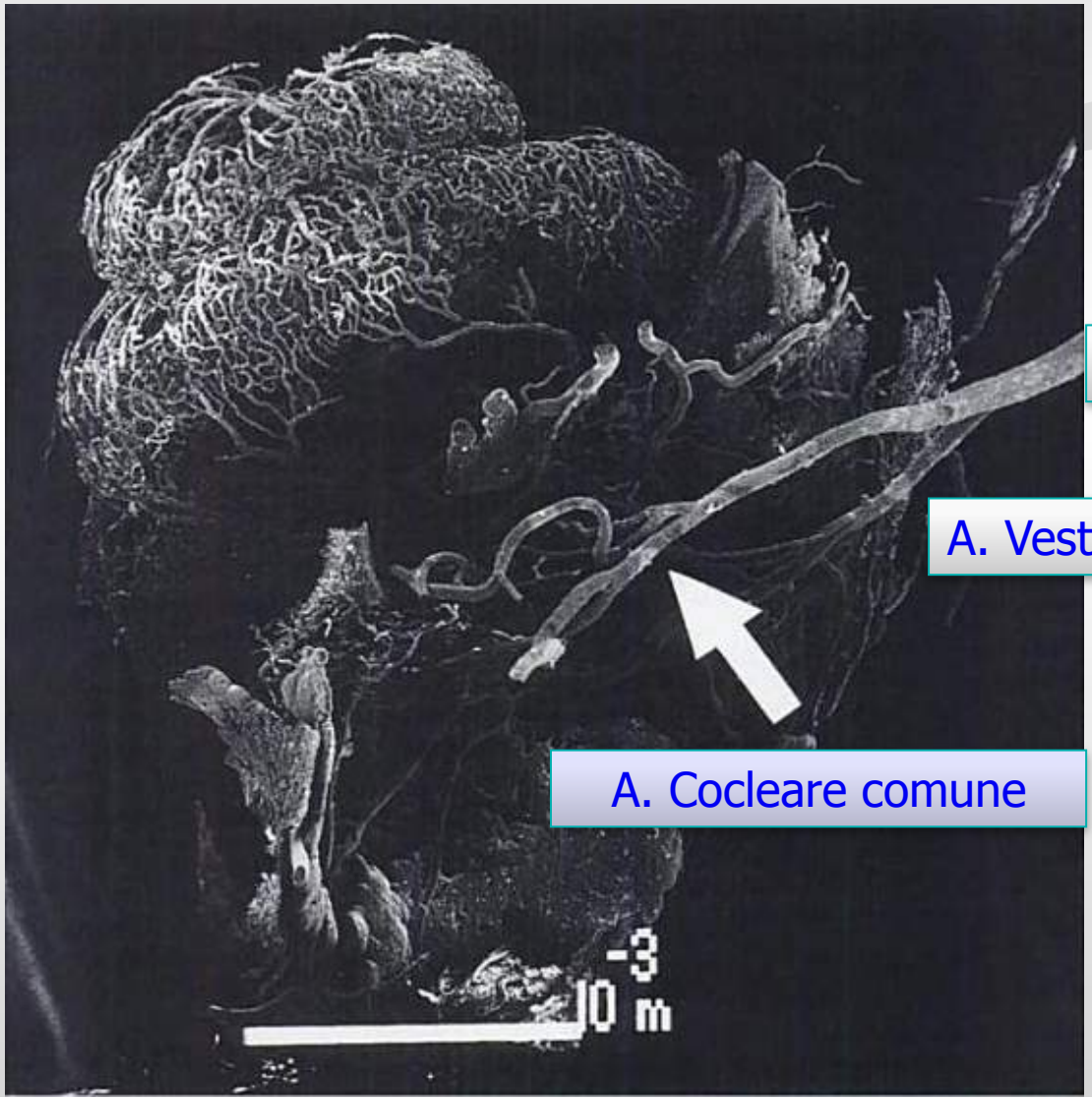


Fig. 1061 - Immagine schematica che illustra l'origine comune dal tronco basilare dell'arteria cerebello-labirintica e dell'arteria cerebellare media: *a.c.c.*, arteria cerebellare comune; *a.c.l.*, arteria cerebello-labirintica; *a.u.i.*, arteria uditiva interna; *a.c.m.*, arteria cerebellare media (da MEZZOGIORNO e Coll.).

VASCULAR STRUCTURES OF THE INNER EAR

R.A. TANGE

Department of Otorhinolaryngology, Academic Medical Centre, University of Amsterdam, Amsterdam, The Netherlands



Arteria Labirintica

A. Vestibolare anteriore

A. Cocleare comune

VASCULAR STRUCTURES OF THE INNER EAR

R.A. TANGE

Department of Otorhinolaryngology, Academic Medical Centre, University of Amsterdam, Amsterdam, The Netherlands

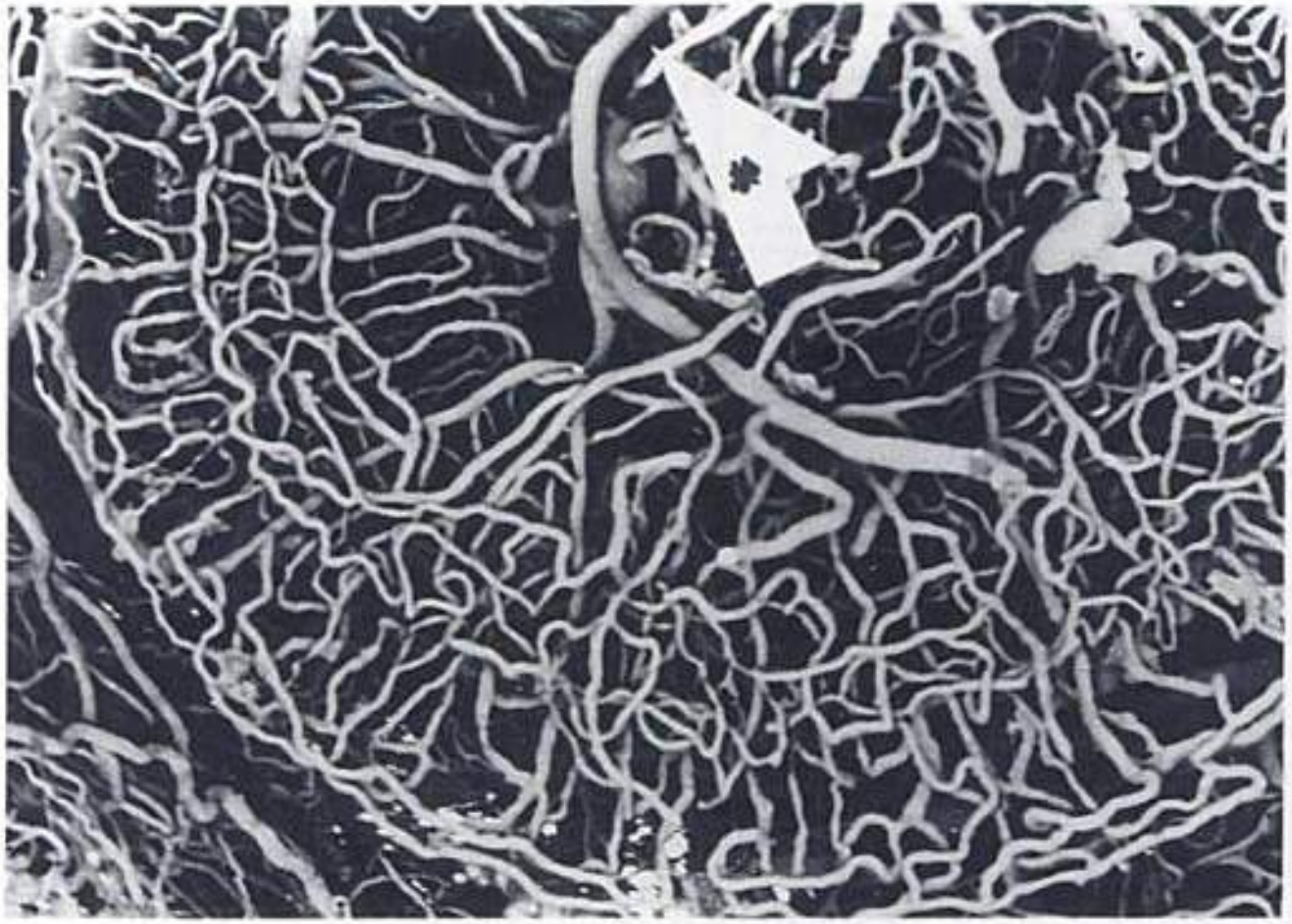
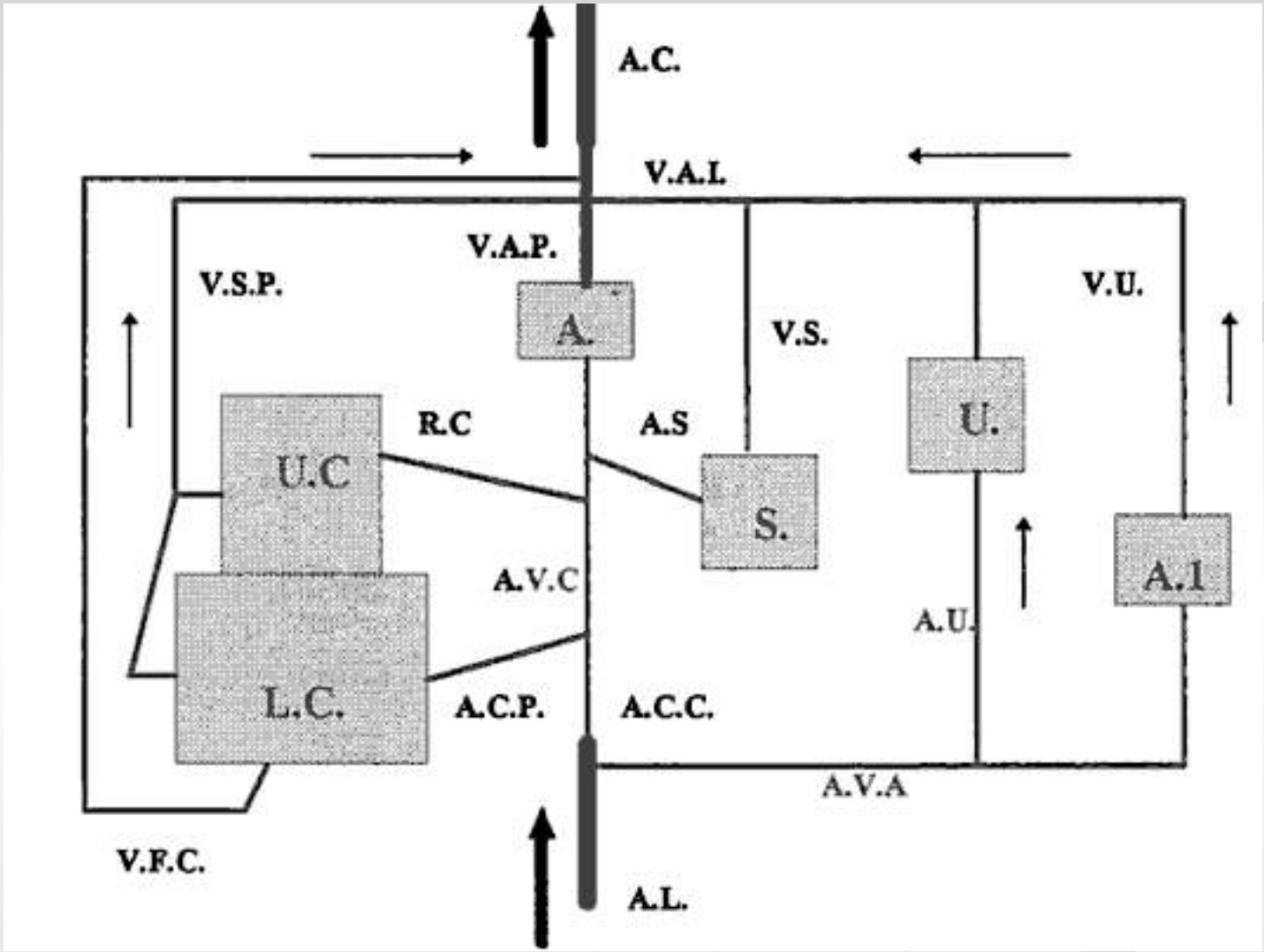
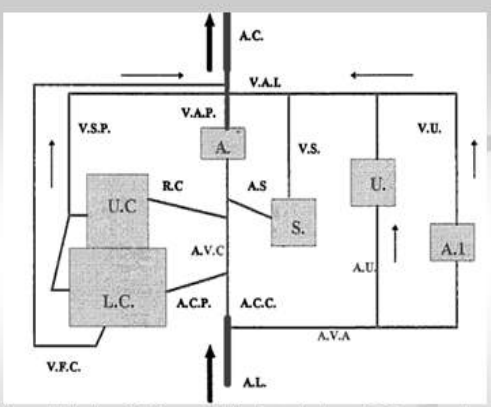


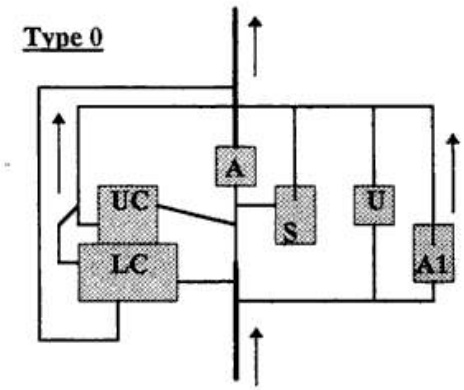
Fig. 2. The *Ramus cochlearis* entering the modiolus in the upper part of the cochlea supplying the organ of Corti (*arrow).



A.L.=a. labirintica. A.V.A.=a. vestibolare anteriore. A.C.C.= a. cocleare comune. A.C.P.= a. cocleare propria. A.V.C.=a. vestibolare comune. R.C.= ramo cocleare. A.S.= A. sacculare. A.U.= a. utriculare. A.C.=Acquedotto della coclea

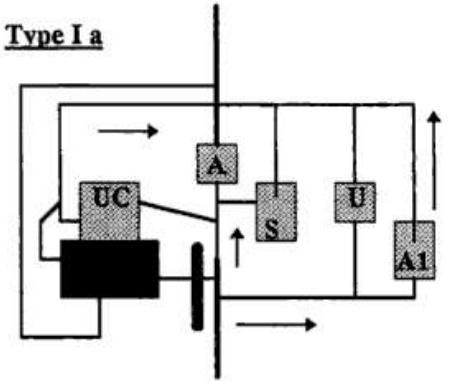


A.L.=a. labirintica. A.V.A.=a. vestibolare anteriore. A.C.C.= a. cocleare comune. A.C.P.= a. cocleare propria. A.V.C.=a. vestibolare comune. R.C.= ramo cocleare. A.S.= A. sacculare. A.U.= a. utriculare. A.C.=Acquedotto della coclea



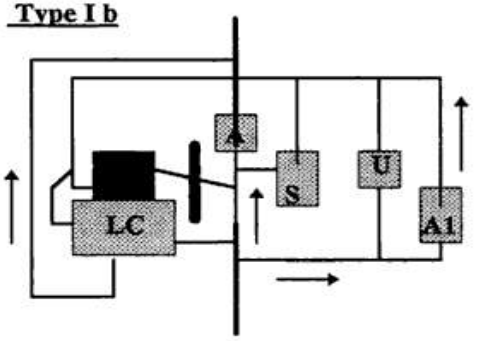
Hearing loss		Vertigo
High tone loss	Low tone loss	
-	-	-

a

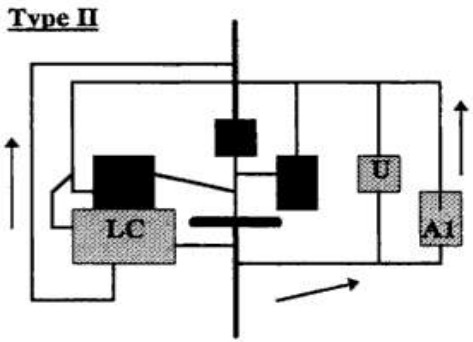


Hearing loss		Vertigo
High tone loss	Low tone loss	
+	-	-

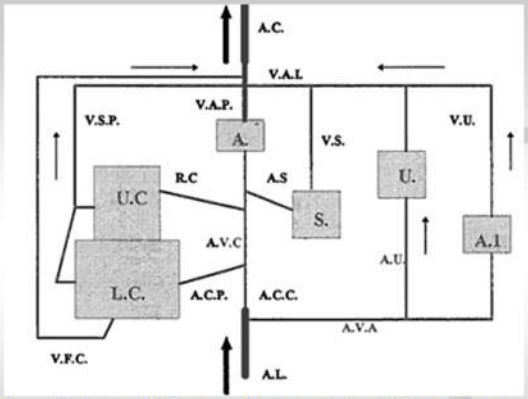
b



Hearing loss		Vertigo
High tone loss	Low tone loss	
-	+	-

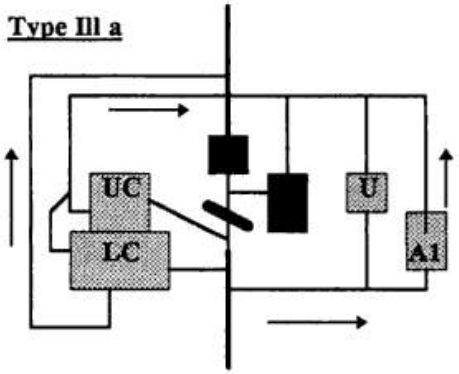


Hearing loss		Vertigo
High tone loss	Low tone loss	
-	+	+/-



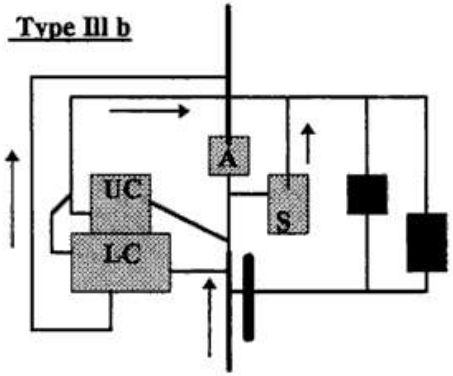
A.L.=a. labirintica. A.V.A.=a. vestibolare anteriore. A.C.C.= a. cocleare comune. A.C.P.= a. cocleare propria. A.V.C.=a. vestibolare comune. R.C.= ramo cocleare. A.S.= A. sacculare. A.U.= a. utriculare. A.C.=Acquedotto della coclea

Type Ill a



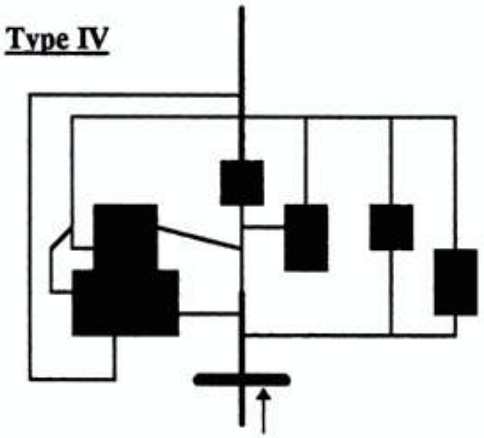
Hearing loss		Vertigo
High tone loss	Low tone loss	
-	-	+

Type Ill b



Hearing loss		Vertigo
High tone loss	Low tone loss	
-	-	+

Type IV



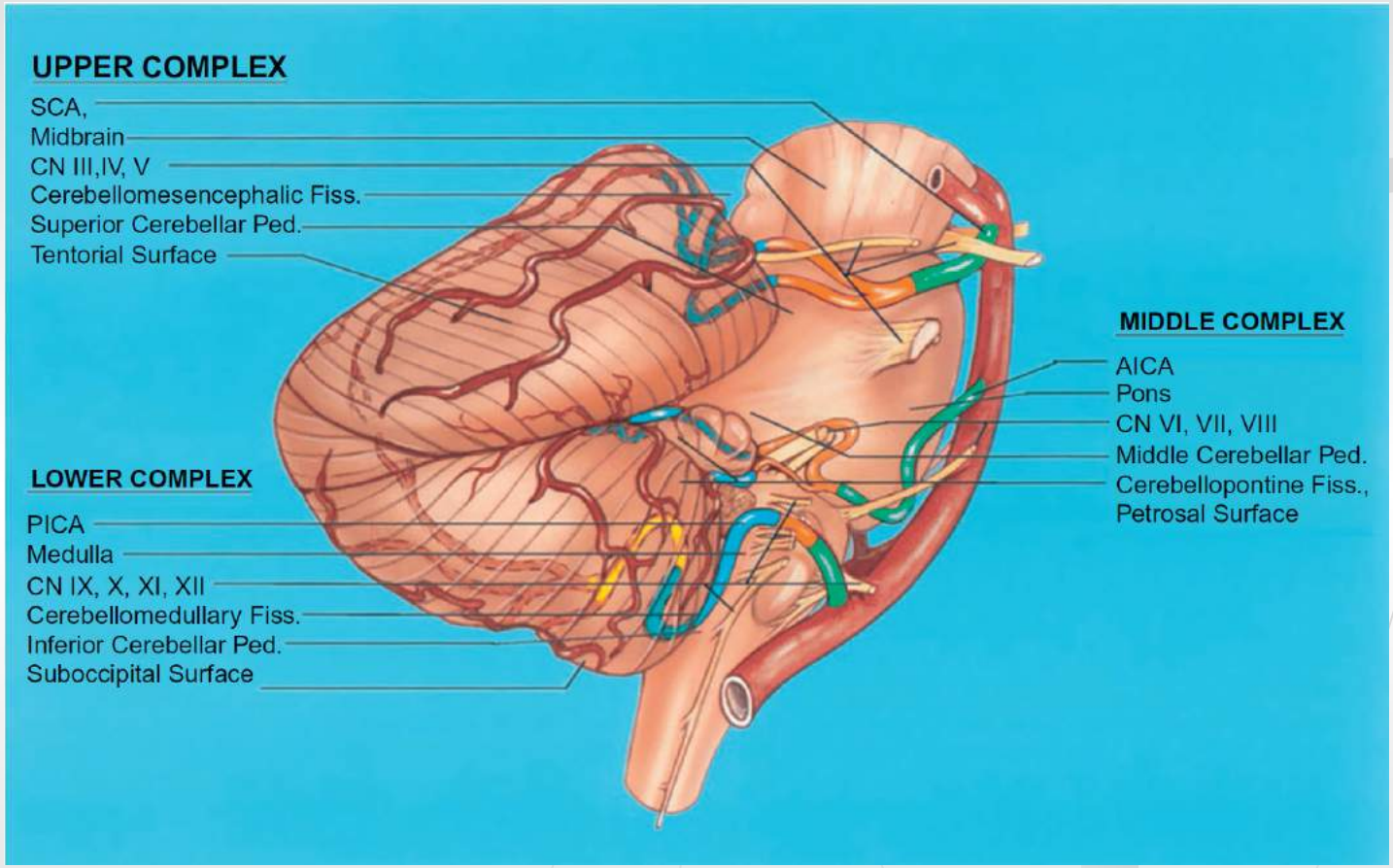
Hearing loss		Vertigo
High tone loss	Low tone loss	
+	+	+

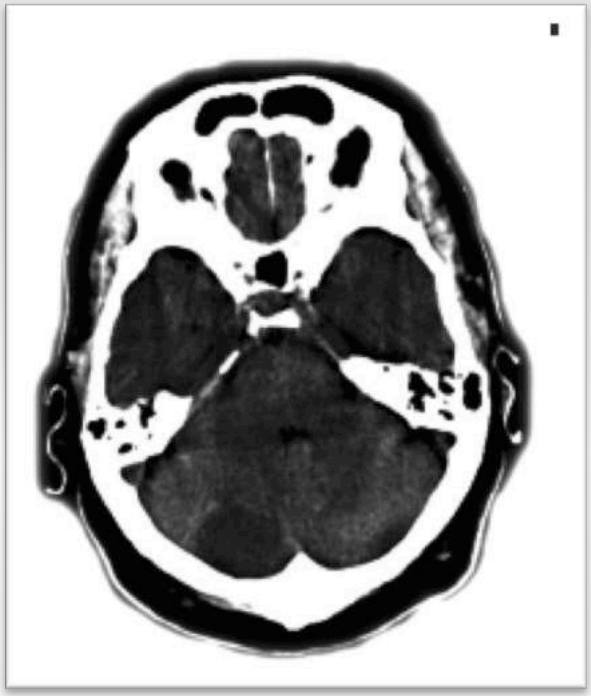
…in un paziente anziano e/o in un paziente con fattori di rischio

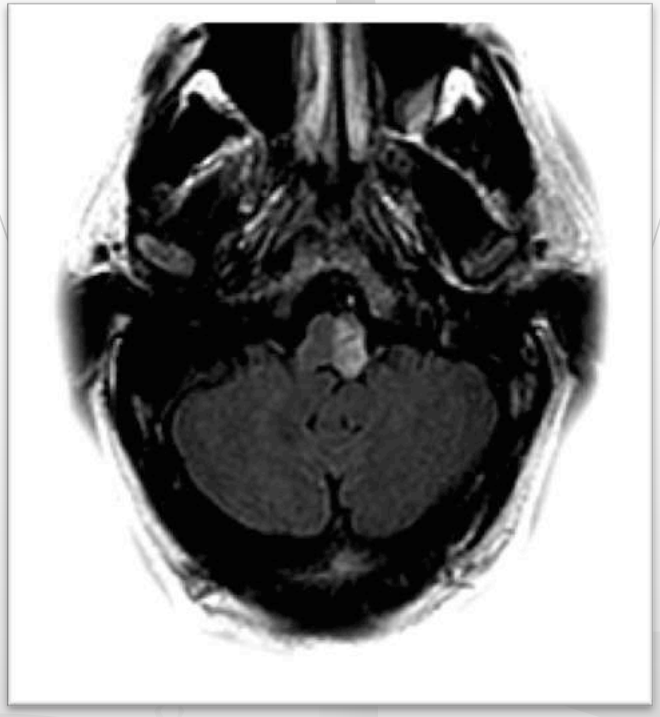
- Ipertensione
- Obesità
- Diabete
- Precedenti cerebrovascolari

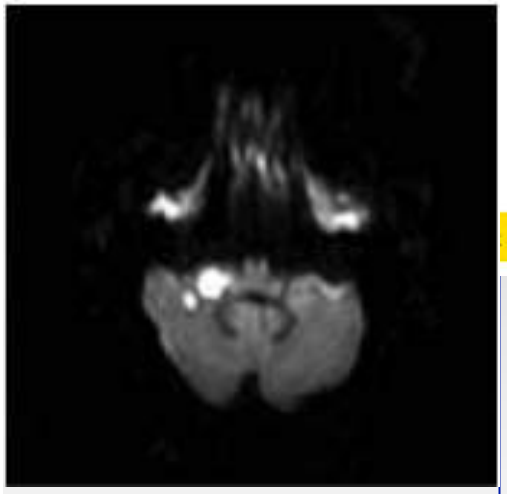
SOSPETTARE SEMPRE UNA GENESI VASCOLARE, CENTRALE e/o PERIFERICA

La neurite vestibolare non fa morire, l'accidente cerebro-vascolare talora sì!!


UPPER COMPLEX
SCA,
Midbrain
CN III,IV, V
Cerebellomesencephalic Fiss.
Superior Cerebellar Ped.
Tentorial Surface
MIDDLE COMPLEX
AICA
Pons
CN VI, VII, VIII
Middle Cerebellar Ped.
Cerebellopontine Fiss.,
Petrosal Surface
LOWER COMPLEX
PICA
Medulla
CN IX, X, XI, XII
Cerebellomedullary Fiss.
Inferior Cerebellar Ped.
Suboccipital Surface







Stroke
NAL OF THE AMERICAN HEART ASSOCIATION

American Stroke Association
A Division of American Heart Association

Infarction in the territory of anterior inferior cerebellar artery: spectrum of audiovestibular loss.

Lee H, Kim JS, Chung EJ, Yi HA, Chung IS, Lee SR, Shin JY.

Stroke. 2009 Dec;40(12):3745-51. Epub 2009 Sep 24.

Infarction in the anterior inferior cerebellar artery territory can present with a broad spectrum of audiovestibular dysfunctions. Unlike a viral cause, labyrinthine dysfunction of a vascular cause usually leads to combined loss of both auditory and vestibular functions.

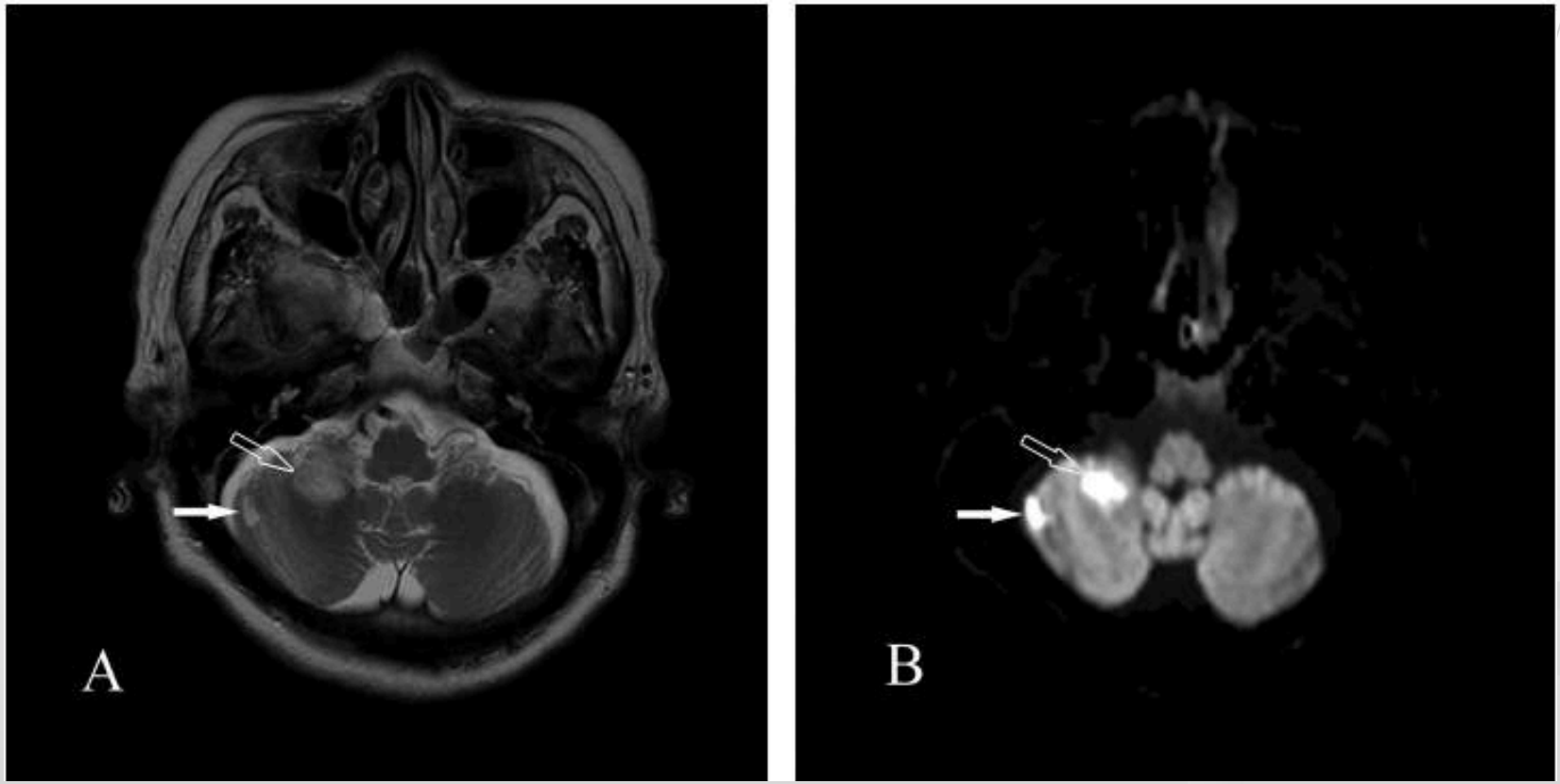
In most patients (80 of 82 [98%]), acute spontaneous prolonged vertigo (>24 hours) with nausea/vomiting was the presenting or main symptom. The spontaneous nystagmus was predominantly horizontal and 55 (67%) patients showed spontaneous nystagmus beating away from (84% [46 of 55]) or toward (11% [6 of 55]) the side of the lesion.

The complete AICA syndrome described by Adams was found in only 2 (2%)

The Laryngoscope
Lippincott Williams & Wilkins, Inc.

Anterior Inferior Cerebellar Artery Infarction Presenting With Sudden Hearing Loss and Vertigo

Eun Jin Son, MD; Jung Hwan Bang, MD; Jae-Goo Kang, MD



Vertigo and the anterior inferior cerebellar artery syndrome.

Oas JG, Baloh RW.

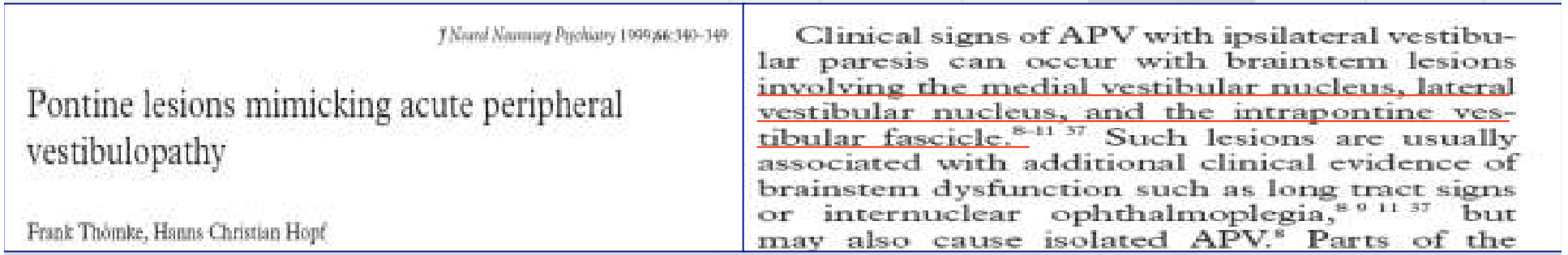
Neurology. 1992 Dec;42(12):2274-9.

Sudden deafness with vertigo as a sole manifestation of anterior inferior cerebellar artery infarction.

Lee H, Ahn BH, Baloh RW.

J Neurol Sci. 2004 Jul 15;222(1-2):105-7.

Since the blood supply to the inner ear and the vestibulocochlear nerve arises from AICA, a combination of peripheral and central symptoms and signs is characteristic of the AICA infarction syndrome. The vertigo that preceded infarction may have resulted from transient ischemia to the inner ear or the vestibular nerve.



J Neurol Neurosurg Psychiatry 1999;66:340–349

Pontine lesions mimicking acute peripheral vestibulopathy

Frank Thömke, Hanns Christian Hopf

Clinical signs of APV with ipsilateral vestibular paresis can occur with brainstem lesions involving the medial vestibular nucleus, lateral vestibular nucleus, and the intrapontine vestibular fascicle.[8–11 37] Such lesions are usually associated with additional clinical evidence of brainstem dysfunction such as long tract signs or internuclear ophthalmoplegia,[8 9 11 37] but may also cause isolated APV.[8] Parts of the

Isolated Nodular Infarction

In Soo Moon, Ji Soo Kim, Kwang Dong Choi, Min-Jeong Kim, Sun-Young Oh, Hyung Lee, Hak-Seung Lee and Seong-Ho Park

Stroke 2009;40;487-491; originally published online Dec 24, 2008;

Conclusions—Isolated nodular infarction mostly presents with isolated vertigo mimicking acute peripheral vestibulopathy. However, severe imbalance and a negative head impulse test are important clinical discriminants between nodular infarcts and peripheral vestibular dysfunction. The findings of isolated nodular infarctions are consistent with impaired gravito-inertial processing of the vestibular signals and disrupted nodular inhibition on the vestibular secondary neurons

Bilateral sudden profound hearing loss and vertigo as a unique manifestation of bilateral symmetric inferior pontine infarctions.

Bovo R, Ortore R, Ciorba A, Berto A, Martini A.
Department of Audiology, Ferrara University, Ferrara, Italy.

Ann Otol Rhinol Laryngol. 2007 Jun; 116(6):407-10.

Acute vertigo and sudden deafness in a patient with known cerebrovascular occlusive disease may represent the warning signs of an impending brain stem or cerebellar infarction, even when other neurologic signs are absent. These events are fortunately very rare, but should be considered by clinicians who see patients with vertigo.

Laryngoscope. 1980 Mar;90(3):505-14.

Cerebellar infarction--a presentation of vertigo.

Rubenstein RL, Norman DM, Schindler RA, Kaseff L.

Computerized tomography should be included in the routine work-up of acute vertigo with any associated neurologic findings to aid in proper diagnosis and effect appropriate treatment.

Acta Otolaryngol Suppl. 1993;503:64-6.

Cerebellar infarctions and 'vestibular neuritis'.

Magnusson M, Norrving B.

Consecutive subjects 50 to 75 years of age with sudden onset of vertigo but without cochlear or neurological symptoms were investigated with neuro-imaging techniques. Doppler sonography of the vertebral and carotid arteries and recording of voluntary saccades and pursuit eye movements, caloric, spontaneous, gaze and optokinetic nystagmus. Among those studied, 6 out of 24 subjects could be demonstrated to have cerebellar infarctions, another 2 subjects had occlusion of one vertebral artery.

Otolaryngol Head Neck Surg. 2013 Mar;148(3):475-81. doi: 10.1177/0194599812472614. Epub 2013 Jan 10.

Cerebellar infarctions mimicking acute peripheral vertigo: how to avoid misdiagnosis?

Casani AP, Dallan I, Cerchiai N, Lenzi R, Cosottini M, Sellari-Franceschini S.

Department of Neurosciences, Otorhinolaryngology Unit, Pisa University Hospital, Pisa, Italy. a.casani@med.unipi.it

CONCLUSIONS: Pseudo-APV is not an uncommon diagnosis in otoneurological practice. The presence of moderate-severe imbalance and the persistence of **vertigo** for more than 72 h from the onset, together with the results of bedside examination tests (spontaneous nystagmus and Head Impulse Test), are useful indicators for recognizing a cerebellar ischemic origin in cases of acute **vertigo**.

J Neurol Sci. 2014 Mar 15;338(1-2):23-9. doi: 10.1016/j.jns.2013.12.048. Epub 2014 Jan 10.

Recent advances in acute hearing loss due to posterior circulation ischemic stroke.

Lee H[1].

Abstract

Acute hearing loss (AHL) has traditionally been considered to be a neglected and underestimated symptom of stroke. However, because the blood supply to the auditory system originates from the vertebrobasilar system, stroke in the distribution of the vertebrobasilar circulation can present with acute hearing loss (AHL) and/or tinnitus. Approximately one-tenth of vertebrobasilar ischemic stroke (VBIS) is accompanied by AHL. Sometimes, AHL is a warning symptom of impending VBIS (mainly in the anterior inferior cerebellar artery). In this case, the MRI is normal, and the clinician must rely on other clinical features to make the diagnosis. This review summarizes the current advances in the clinical syndromes and signs of AHL due to VBIS.

Otol Neurotol. 2013 Feb;34(2):e6-7. doi: 10.1097/MAO.0b013e31826dba43.

Sudden onset hearing loss and vertigo just before posterior inferior cerebellar artery infarction (lateral medulla syndrome).

Kanzaki S[1], Suzuki T, Suzuki S, Suzuki N, Ogawa K.

J Clin Neurosci. 2013 Jan;20(1):177-9. doi: 10.1016/j.jocn.2012.01.043. Epub 2012 Sep 16.

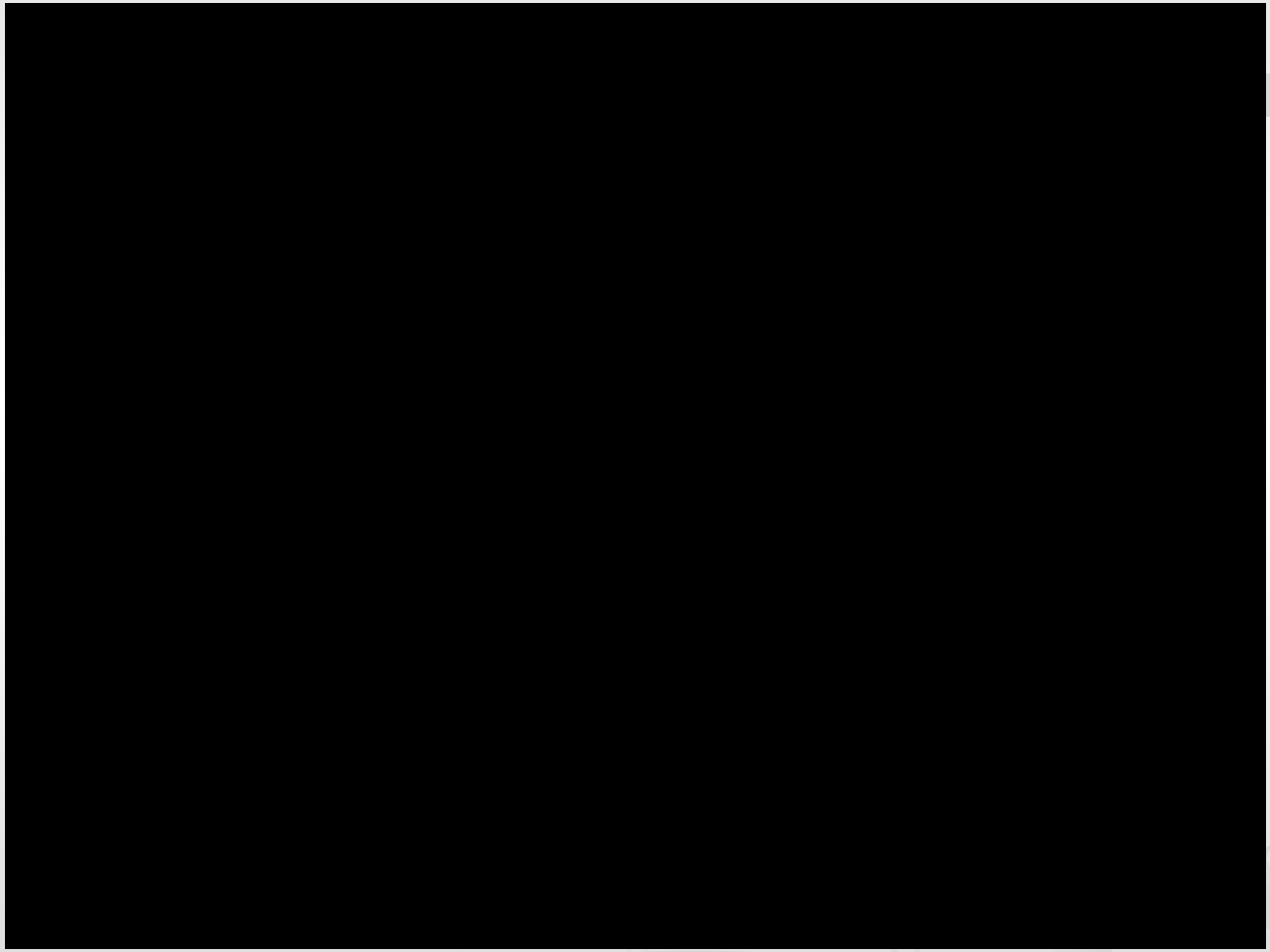
Acute bilateral hearing loss as a "worsening sign" in a patient with critical basilar artery stenosis.

Chiang CI[1], Chou CH, Hsueh CJ, Cheng CA, Peng GS.

Abstract

We report a patient who presented with an acute-onset transient vertigo and unsteady gait with bilateral hearing loss. Brain MRI revealed a critical basilar artery (BA) stenosis at the lower pons and infarction in various areas on both sides in the territories of the posterior inferior cerebellar arteries (PICA). Further, we could not visualize the right anterior inferior cerebellar artery (AICA). The bilateral hearing loss may be ascribed to stroke due to the critical BA stenosis, causing hypoperfusion injury extending from the PICA to the AICA on both sides. Local intra-arterial thrombolytic therapy with the administration of 1×10(6) IU of urokinase aided partial recanalization of the BA, after which the right AICA reappeared. The neurological function of the patient recovered to normal, and no hemorrhagic complications were observed. Therefore, practitioners should be alert when treating patients with acute bilateral hearing loss, which may be related to an underlying catastrophic stroke.

Due vecchi video di accessi dal Pronto Soccorso



….e quindi?

- **La concomitanza anamnestica di fattori di rischio vascolare (l'età stessa può esserlo!)**
- **La concomitanza di altri segni neurologici**
- **La concomitanza di danno uditivo (solitamente assente nel DVA postvirale) o la presenza di danno uditivo bilaterale**

devono indurre al sospetto di lesione vascolare o espansiva con possibile interessamento tronco-cerebellare…

… pur in presenza di un quadro periferico iniziale "macroscopico" coerente con una "Neurite Vestibolare" (Nistagmo spontaneo orizzontale-rotatorio deficitario stazionario e persistente), senza cioè nistagmi di tipo centrale

NB: dati quali vertigine oggettiva vs. vertigine soggettiva, grande vertigine vs. instabilità, sintomi neurovegetativi imponenti vs. scarsi sintomi neurovegetativi, non hanno NESSUN significato né diagnostico, né diagnostico-differenziale

Domande fondamentali

E' possibile distinguere in un esame bed-side una vertigine periferica da una centrale?

Quando indicare il neuroimaging mirato nel sospetto di una genesi centrale?

J Emerg Med. 2018 April ; 54(4): 469–483. doi:10.1016/j.jemermed.2017.12.024.

A NEW DIAGNOSTIC APPROACH TO THE ADULT PATIENT WITH ACUTE DIZZINESS

Jonathan A. Edlow, MD[*,†], **Kiersten L. Gurley, MD**[*,†,‡], and **David E. Newman-Toker, MD, PHD**[§]

Use of the Physical Examination to Diagnose Patients With Acute Vestibular Syndrome

Exam Component	Peripheral (All Must Be Present to Diagnose Vestibular Neuritis)	Central (Any One of These Findings Suggests Posterior Fossa Stroke)
Nystagmus (straight-ahead gaze and rightward and leftward gaze)	Dominantly horizontal, direction-fixed, beating away from the affected side [*]	Dominantly vertical or torsional or dominantly horizontal, direction-changing on left/right gaze [†]
Test of Skew (alternate cover test)	Normal vertical eye alignment and no corrective vertical movement (i.e., no skew deviation)	Skew deviation (small vertical correction on uncovering the eye) [‡]
Head Impulse Test	Unilaterally abnormal with head moving towards the affected side (presence of a corrective re-fixation saccade towards the normal side) [§]	Usually bilaterally normal (no corrective saccade)
Targeted neurologic examination (see text)	No cranial nerve, brainstem, or cerebellar signs	Presence of limb ataxia, dysarthria, diplopia, ptosis, anisocoria, facial sensory loss (pain/temperature), unilateral decreased hearing
Gait and truncal ataxia	Able to walk unassisted and to sit up in stretcher without holding on or leaning against bed or rails	Unable to walk unassisted or sit up in stretcher without holding on or leaning against bed or rails

Elementi cardine

- *(Positional testing)*
- HINTS (HINTS-plus, Standing)
- Deviazione coniugata laterale dello sguardo

A clinical sign of canal paresis.

Halmagyi GM, Curthoys IS

Department of Neurology, Royal Prince Alfred Hospital, Camperdown, Sydney, New South Wales, Aust

Arch Neurol. 1988 Jul;45(7):737-9.

head velocity) of the VOR.[8] Consider a patient with left vestibular neuritis: if the patient fixates a distant earth-fixed target while the clinician rapidly rotates the patient's head to the left, the passive head-impulse test, the patient will make 1 or 2 observable, compensatory, that is, rightward, saccades just after the head impulse is over.

This is the head-impulse sign and it indicates that the gain of the horizontal VOR, which is generated from inhibition of the sole functioning, right, lateral SCC, rather than from excitation of the nonfunctioning left lateral SCC, is severely defective. In other words, the VOR gain is much less than 1.0. In contrast, when the clinician rotates the

Bedside differentiation of vestibular neuritis from central "vestibular pseudoneuritis"

C D Cnyrim,[1] D Newman-Toker,[2] C Karch,[1] T Brandt,[1] Michael Strupp[1]

J Neurol Neurosurg Psychiatry 2008;**79**:458–460.

Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis

David E. Newman-Toker, MD, PhD*
Jorge C. Kattah, MD*
Jorge E. Alvernia, MD
David Z. Wang, DO

Neurology, 70; June 2008 (Part 2 of 2)

Head impulse test in unilateral vestibular loss

Vestibulo-ocular reflex and catch-up saccades

K.P. Weber, MD
S.T. Aw, PhD
M.J. Todd, MBiomedE
L.A. McGarvie, MBiomedE
I.S. Curthoys, PhD
G.M. Halmagyi, MD

Neurology, 70; February 2008

I principi di fondo

Un improvviso spostamento impulsivo della testa, indotto da una forza esterna, ad alta velocità (> 150°/ sec) e ad alta accelerazione sull'asse orizzontale stimola i recettori del canale semircolare laterale del lato verso cui avviene l'impulso, cimentando l'efficienza del VOR nel riportare l'occhio indietro su una mira di riferimento con una latenza di 5-7 msec.

(Lo stesso concetto è applicabile anche per le coppie di canali verticali "LARP" e "RALP").

Valutare la posizione finale dell'occhio alla fine dell'impulso e l' eventuale movimento correttivo (saccade) verso una mira "fixed to earth" è alla base dell'HIT clinico ("Occhio vs. Occhio")

Positività = comparsa di saccade/i correttiva/e quando, con paziente che fissa una mira, la testa è sottoposta *passivamente* a piccolo e rapido "thrust" verso il lato malato

HINTS

Head Impulse Test

Test positivo in fase acuta

Test positivo in fase di compenso

Management of common central vestibular disorders

Thomas Lempert[a] and Adolfo Bronstein[b]

Current Opinion in Otolaryngology & Head and Neck Surgery 2010, 18:436–440

Missing a posterior fossa stroke in a patient with an acute vestibular syndrome is a classical emergency room nightmare. A normal head impulse test is regarded as the most reliable sign for an intact periphery, thus suggesting a central lesion [23]. However, lateral pontine infarctions may encompass the root entry zone of the 8th nerve, resulting in an abnormal head impulse test, which falsely suggests a peripheral lesion. A refined bedside examination protocol that combined head impulse testing with search for contralateral gaze-evoked nystagmus or skew deviation was 100% sensitive and 96% specific for identification of stroke, whereas the initial diffusion weighted MRI missed 12% of them [24••].

- Vertigine acuta da tre giorni; andatura incerta
- Nistagmo spontaneo orizzontale destro (in discesa)

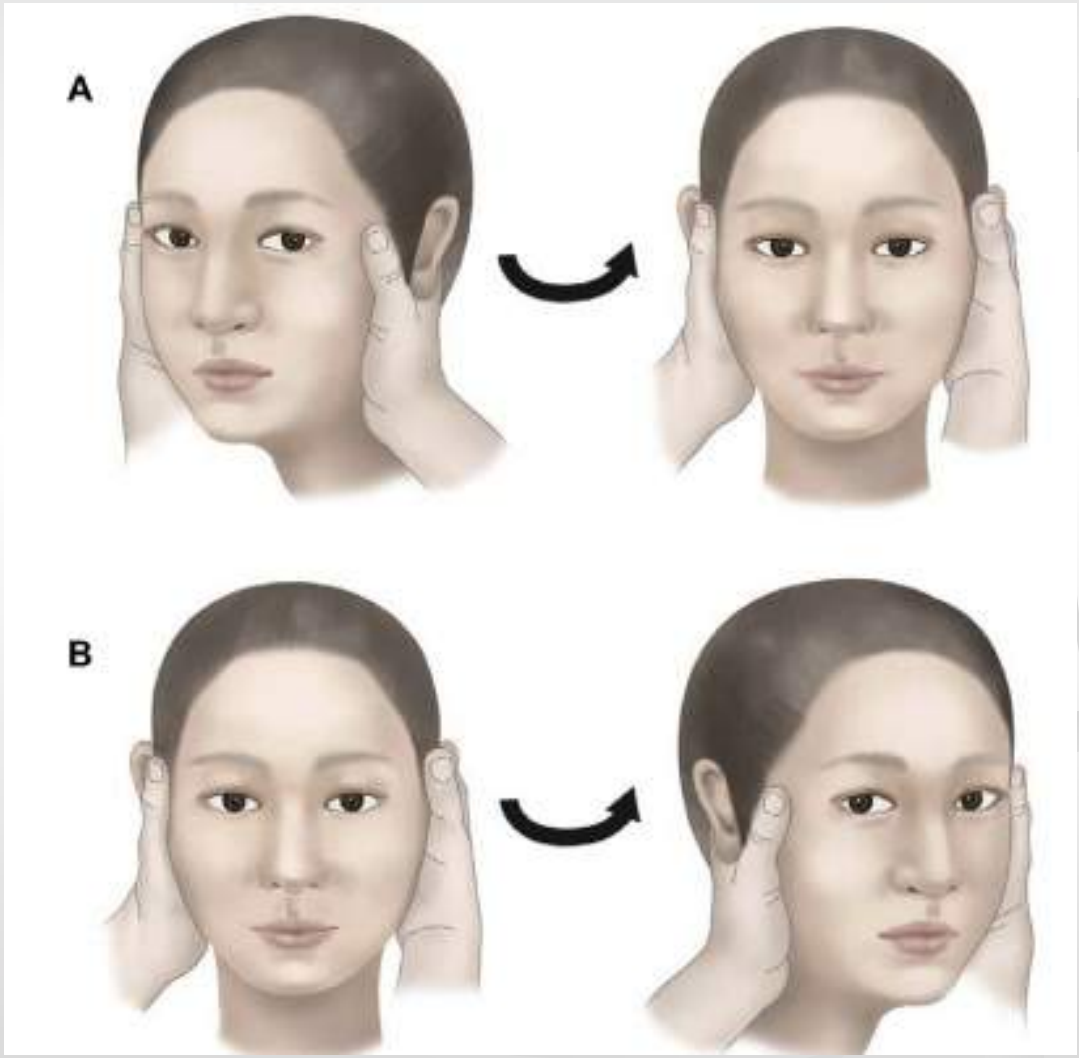
completamente inibito dalla fissazione visiva!

Infarto cerebellare

N.B.: Negatività è diverso da normalità

Compensatory saccade differences between outward *versus* inward head impulses in chronic unilateral vestibular hypofunction

Seung-Han Lee [a,b,c], David E. Newman-Toker [a,d], David S. Zee [a,d], Michael C. Schubert [d,e,*]



Clinical Study

Compensatory saccade differences between outward *versus* inward head impulses in chronic unilateral vestibular hypofunction

Seung-Han Lee [a,b,c], David E. Newman-Toker [a,d], David S. Zee [a,d], Michael C. Schubert [d,e,*]

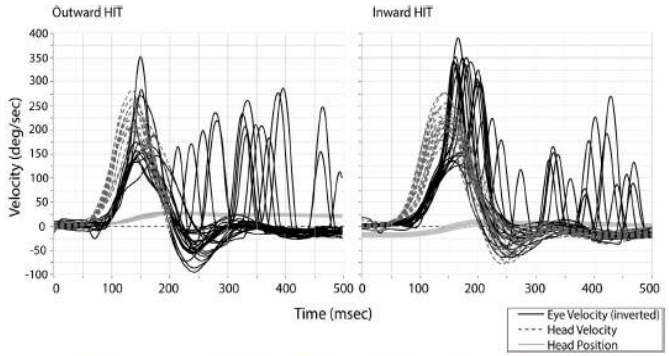


Fig. 2. Outward *versus* inward head impulse test (HIT) in one patient with left unilateral vestibular hypofunction. Eye velocity trace is inverted for ease of comparison. (Left) For the outward HIT, 13 trials are displayed and the head position changes from an initially centered head position (stippled gray line at 0 velocity) and then is moved to the left (positive/upward deflection). (Right) For the inward HIT, 16 trials are displayed and the head is initially positioned to the right (negative/downward deflection) and then moved leftward/towards the center (stippled gray line at 0 velocity). In this patient, a higher proportion of covert saccades occurred with the inward HIT. deg = degrees, msec = milliseconds, sec = seconds.

A B S T R A C T

The horizontal head impulse test (HIT) is a valuable clinical tool that can help identify peripheral vestibular hypofunction by the refixation (compensatory) saccade that returns the eyes to the target of interest after the head has stopped. We asked if there were differences in the compensatory saccade responses during the HIT when the head was rotated away or toward straight ahead (outward *versus* inward). We also investigated the influence of a fixation target. Using scleral search-coils, we tested five patients with chronic unilateral vestibular hypofunction (UVH) and three healthy control subjects. In UVH patients, the latencies of both overt and covert saccades were longer when the head was rotated inward from an initially eccentric position, regardless of a visual target. The proportion of HIT with covert saccades was independent of a visual target. In control subjects no compensatory saccades were observed and there were no differences in either angular vestibulo-ocular reflex gain or latency between inward and outward HIT. Our data suggest that inward applied HIT in chronic UVH is more likely to include an overt compensatory saccade based on its lengthened latency. Neither latency nor the occurrence of covert compensatory saccades during HIT depended on a visual target, suggesting they have become a learned behavior in response to chronic UVH.

CORRISPETTIVO CLINICO: L'HEAD IMPULSE VERSO LA POSIZIONE CENTRALE DELLA TESTA DETERMINA NEL SOGGETTO CON DEFICIT VESTIBOLARE PERIFERICO MAGGIORE EVIDENZA DELLE «OVERT SACCADES» PER AUMENTO DELLA LORO LATENZA

- **Abrupt, brief duration, high acceleration**
- **Small excursion**
- **BEGIN from eccentric and stop at center (to prevent gaze-evoked nystagmus from obscuring the deficit or,**
- **BEGIN from center and stop at eccentric to prevent anticipation from obscuring the deficit**

Courtesy, David S. Zee

Head Impulse Test sec Halmagyi

- Appare dalla Letteratura il Test che meglio distingue una vestibulopatia deficitaria periferica da una centrale, quando manchino nistagmi patognomonici di una sofferenza centrale (gaze ny, rebound ny, PA- ny, gaze paretic ny, nistagmo dissociato, ecc.)
- Necessità però di una marcata iporeflessia vestibolare per elicitare il segno (Halmagyi, Beynon, Schubert)
- Possibili falsi negativi : problema delle occult (o covert) saccades, saccadi *durante* piuttosto che *dopo* il thrust. Il V-HTT annulla tale problema; l'»inward» Head Impulse lo riduce anche in valutazione bed-side
- Possibili positivi in patologie cerebellari e latero-pontine (da coinvolgimento cocleolabirintico associato) (Newmann-Toker: 9%; Cnyrim: 39%!!)
- La positività del test è più specifica per sofferenza periferica rispetto a quanto lo sia la negatività per sofferenza centrale

J Neurol (2013) 260:1576–1582
DOI 10.1007/s00415-013-6837-y

ORIGINAL COMMUNICATION

Isolated floccular infarction: impaired vestibular responses to horizontal head impulse

Hong-Kyun Park · Ji-Soo Kim · Michael Strupp · David S. Zee

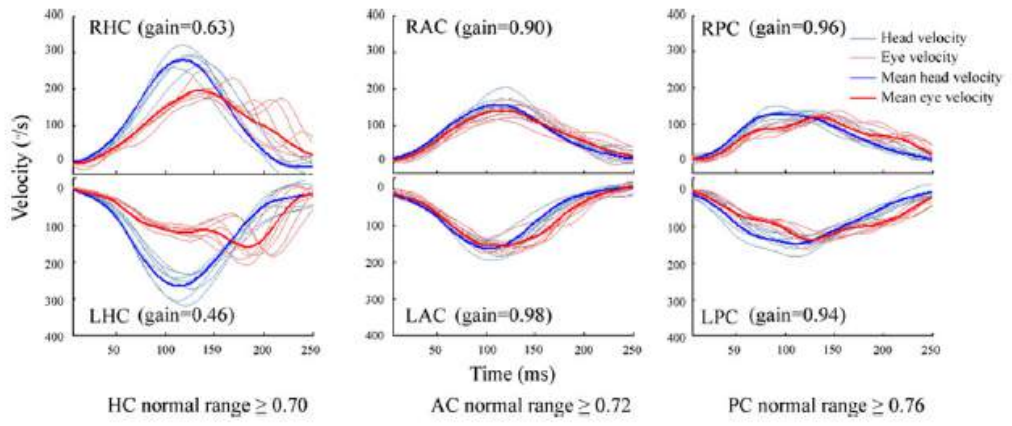


Fig. 2 During the head impulse test using the scleral search coil technique, there were impaired vestibular responses only during stimulation of both horizontal semicircular canals. *AC* anterior semicircular canal, *HC* horizontal semicircular canal, *PC* posterior semicircular canal

In conclusion, our patient with the rare syndrome of an isolated unilateral floccular infarction showed normal caloric responses, increased horizontal VOR gains during the low-frequency rotatory chair test and decreased horizontal VOR gains during the high-frequency, high acceleration HIT. These results suggest that the flocculus inhibits the horizontal VOR during low-frequency stimulation and facilitates it during high-frequency stimulation. This finding also supports the clinical dictum that in a patient with an acute vestibular syndrome an abnormal head impulse response does not exclude a central lesion [26]. Here we show specifically that involvement of the flocculus may produce this sign.

HINTS
Nystagmus

Periferico

Unidirezionale

- presente nello sguardo primario
- **Non cambia direzione**
- Rispetta la **legge di Alexander**

Centrale

Direction-changing

- **Gaze-evoked; Rebound; P-A**

Spontaneo

- Verticale puro (upbeat-downbeat)
- Torsionale puro

HINTS
Nystagmus

HINTS
Nystagmus

[redacted]

[redacted]

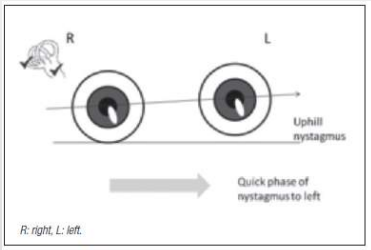


Fig. 1. The right lateral semicircular canal is damaged and a spontaneous horizontal nystagmus arises to the left. At the same time, right utriculus is injured (by the same disease) and an ocular tilt reaction is present, with hypotropia of the right eye. As a result, the plane on which nystagmus beats is inclined upward ("uphill").

Uphill/downhill nystagmus

Nistagmo in salita e nistagmo in discesa

M. GUFONI

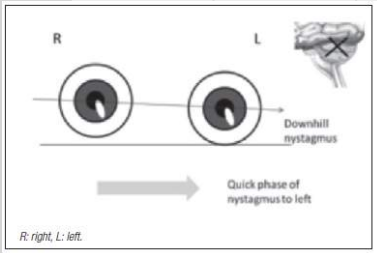
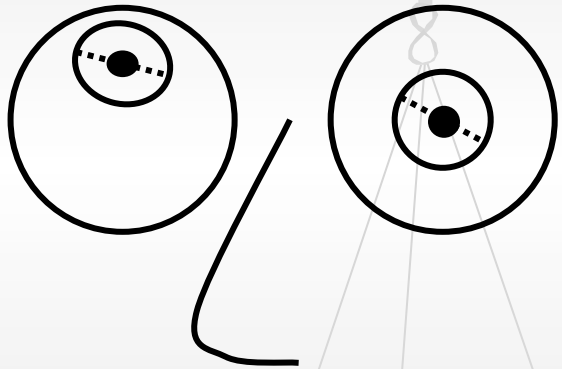


Fig. 2. A lesion is present after decussation of the utricular afferent fibres. Ocular tilt reaction is referred to the opposite side. The (central) resulting nystagmus is tilted "downhill".

HINTS
Test of Skew

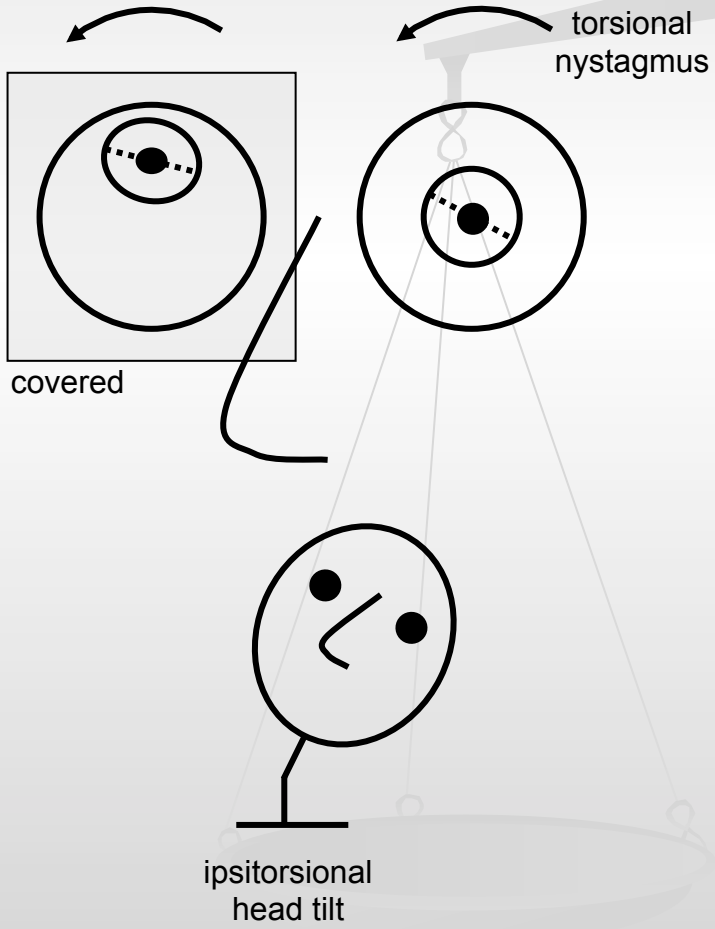


skew deviation

= vertical misalignment of the eyes

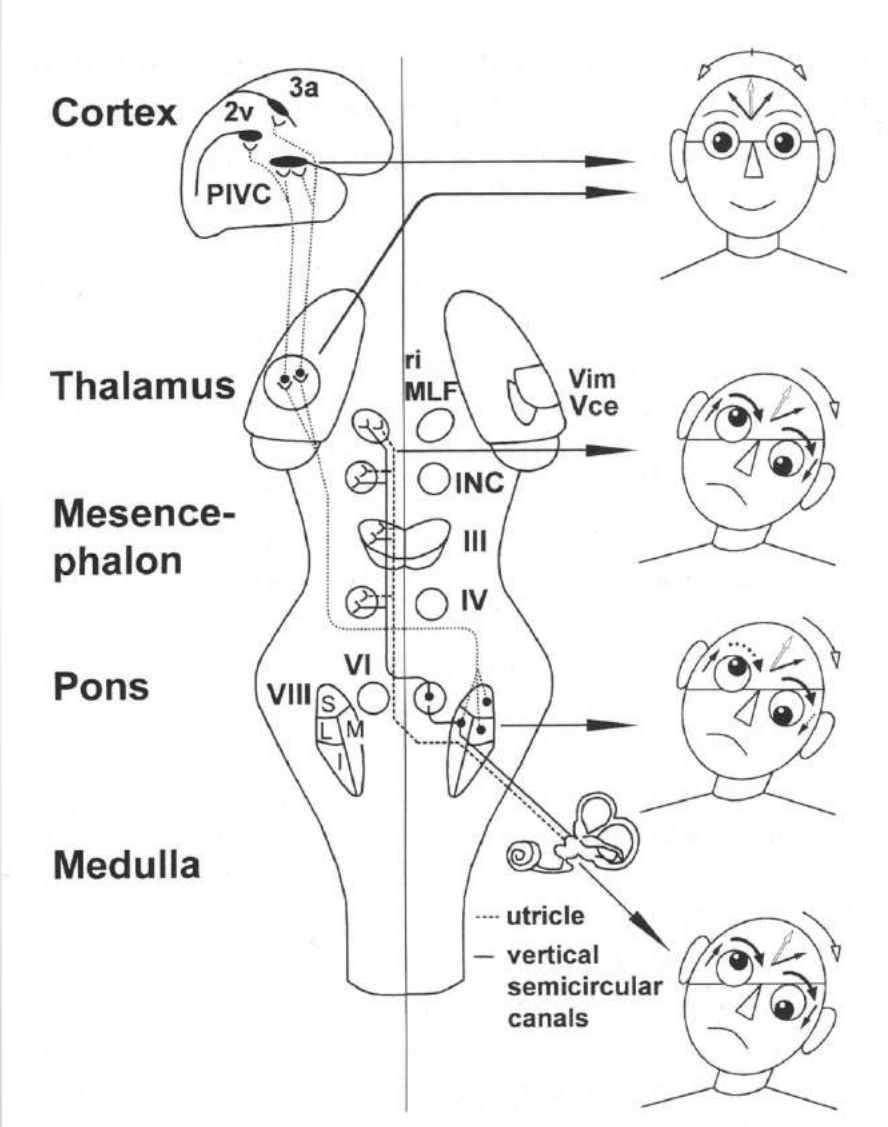
Skew deviation

ocular tilt reaction



Vestibular disorders in frontal (Roll) plane (da Brandt)

OTR (ocular tilt reaction): sincinesia posturale caratterizzata da inclinazione del capo, torsione oculare coniugata e asimmetrica ed ipotropia, tutte verso lo stesso lato



La deviazione è controversiva(occhio controlaterale in basso) nelle lesioni rostrali pontomesencefaliche e nelle lesioni corticali (insula)

La deviazione è "ipsiversiva"(occhio ipsilaterale in basso) nelle lesioni pontomedullari caudali

VESTIBOLOGY

Binocular cyclotorsion in superior vestibular neuritis

La ciclotorsione binoculare nella neurite vestibolare superiore

R. LAPENNA[1], A. PELLEGRINO[2], G. RICCI[1], C. CAGINI[2], M. FARALLI[1]

Department of Surgical and Biomedical Sciences, [1] Section of Otorhinolaryngology, [2] Section of Ophthalmology, University of Perugia, Italy

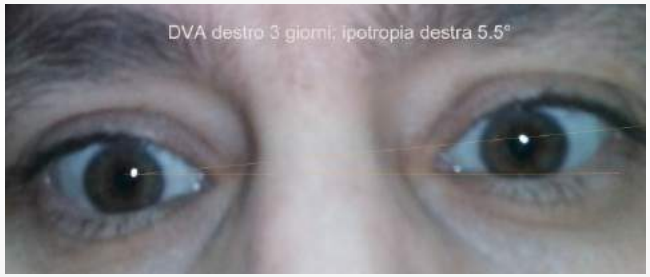
La ciclotorsione coniugata degli occhi verso il lato malato può comunemente essere osservata in corso di neurite vestibolare. Lo scopo di questo studio è stato quello di valutare le differenze nell'entità della ciclotorsione tra l'occhio ipsi- e contra- lesionale in caso di un coinvolgimento selettivo della branca superiore del nervo vestibolare. Abbiamo studiato la ciclotorsione binoculare ottenendo fotografie del fondo oculare in 10 pazienti affetti da neurite vestibolare superiore acuta (NVS). La ciclotorsione è stata studiata anche in 20 soggetti normali. Tutti i pazienti con NVS hanno mostrato una cicloversione ipsilesionale degli occhi. I soggetti normali invece hanno mostrato una exciclovergenza lieve costante (6,42 ± 2,34 °). Nei pazienti con NVS, l'inciclotorsione controlaterale (8.4 ± 8.14 °) era minore e non normalmente distribuita tra i soggetti rispetto alla exciclotorsione dell'occhio ipsilaterale (17,9 ± 4,36 °). Non è stata osservata correlazione significativa tra la ciclotorsione dei due occhi. La differenza interoculare della ciclotorsione potrebbe essere legato in parte alla presenza della exciclovergenza fisiologica, in parte ai diversi effetti tonici sui muscoli estrinseci dei due occhi, e infine alla diversa influenza del nistagmo spontaneo sulla ciclotorsione dei due occhi. Alla luce di questo studio consigliamo di far riferimento solo alla exciclotorsione ipsilaterale nella valutazione della funzione utricolare durante una NVS e del suo successivo compenso. Ulteriori studi sono necessari per determinare il comportamento della ciclotorsione binoculare in caso di coinvolgimento selettivo delle altre componenti del nervo vestibolare.

HINTS
Test of Skew

Spostamenti ampi sono indicatori di lesioni centrali

Valutazione statica della ipotropia

Nistagmo spontaneo "in salita" rispetto all'ipotropia: lesione periferica



Valutazione statica della ipotropia

Nistagmo spontaneo "in discesa" rispetto all'ipotropia: lesione centrale

EVOLUZIONE IN SINDROME DI WALLENBERG

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

Jorge C. Kattah, MD; Arun V. Talkad, MD; David Z. Wang, DO; Yu-Hsiang Hsieh, PhD, MS; David E. Newman-Toker, MD, PhD

Results

- HINTS (**normal** head impulse test and / or **direction-changing** nystagmus and / or **skew deviation**)
  - 100% sensitive and 96% specific for stroke
- early (< 24h) MRI with DWI
  - 72% sensitive and 100% specific for stroke

Conclusions

- In acute vestibular syndrome a 3-step bedside oculomotor examination (HINTS) is more sensitive than an early MRI with DWI.

ORIGINAL RESEARCH CONTRIBUTION

CME HINTS Outperforms ABCD2 to Screen for Stroke in Acute Continuous Vertigo and Dizziness

David E. Newman-Toker, MD, PhD, Kevin A. Kerber, MD, MS, Yu-Hsiang Hsieh, PhD, John H. Pula, MD, Rodney Omron, MD, Ali S. Saber Tehrani, MD, Georgios Mantokoudis, MD, Daniel F. Hanley, MD, David S. Zee, MD, and Jorge C. Kattah, MD

Table 1
ABCD2 and H.I.N.T.S. Elements and Stroke Findings

Five-item ABCD2 risk score	Stroke findings: risk score ≥ 4
• Age	• A ≥60 years = 1
• Blood pressure	• B systolic ≥140 or diastolic ≥/90 = 1
• Clinical features	• C unilateral weakness = 2, speech disturbance without weakness = 1, any other symptom = 0
• Duration of symptoms	• D <10 min = 0; 10–59 min = 1; ≥60 min = 2
• Diabetes	• D present = 1
Three-step "H.I.N.T.S." eye examination*	Stroke findings: "I.N.F.A.R.C.T." (any of these)†
• Head Impulse (right- and leftward)	• Impulse Normal (bilaterally normal)
• Nystagmus type (gaze testing)	• Fast-phase Alternating (direction-changing)
• Test of Skew (alternate cover test)	• Refixation on Cover Test (skew deviation)

*A fourth step (H.I.N.T.S. "plus") includes assessing the presence of new hearing loss, generally unilateral and on the side of the abnormal head impulse test (the side opposite the fast phase of the nystagmus). Recent evidence suggests that, counter to traditional teaching, the presence of such hearing loss more often indicates a vascular (labyrinthine or lateral pontine infarction) rather than viral (labyrinthitis) cause of the AVS presentation.[8,18,19]

ORIGINAL RESEARCH CONTRIBUTION

CME

HINTS Outperforms ABCD2 to Screen for Stroke in Acute Continuous Vertigo and Dizziness

David E. Newman-Toker, MD, PhD, Kevin A. Kerber, MD, MS, Yu-Hsiang Hsieh, PhD, John H. Pula, MD, Rodney Omron, MD, Ali S. Saber Tehrani, MD, Georgios Mantokoudis, MD, Daniel F. Hanley, MD, David S. Zee, MD, and Jorge C. Kattah, MD

old (28.9%). HINTS stroke sensitivity was 96.5%, specificity was 84.4%, LR+ was 6.19, and LR– was 0.04 and did not vary by age. For any central lesion, sensitivity was 96.8%, specificity was 98.5%, LR+ was 63.9, and LR– was 0.03 for HINTS, and sensitivity was 99.2%, specificity was 97.0%, LR+ was 32.7, and LR– was 0.01 for HINTS "plus" (any new hearing loss added to HINTS). Initial MRIs were falsely negative in 15 of 105 (14.3%) infarctions; all but one was obtained before 48 hours after onset, and all were confirmed by delayed MRI.

Conclusions: HINTS substantially outperforms ABCD2 for stroke diagnosis in ED patients with AVS. It also outperforms MRI obtained within the first 2 days after symptom onset. While HINTS testing has traditionally been performed by specialists, methods for empowering emergency physicians (EPs) to leverage this approach for stroke screening in dizziness should be investigated.

ACADEMIC EMERGENCY MEDICINE 2013; 20:987–996 © 2013 by the Society for Academic Emergency Medicine

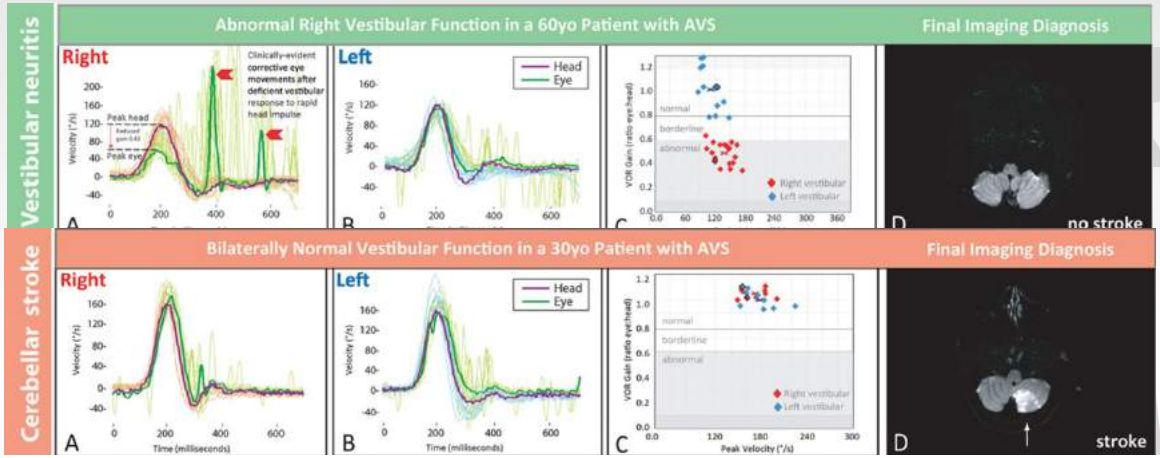
IL PROBLEMA DELLA FORMAZIONE DEI MEDICI DELL'EMERGENZA PER UN CORRETTO APPROCCIO AL PAZIENTE VERTIGINOSO E' COMUNE ANCHE ALLA REALTA' AMERICANA

Stroke, 2013; 44: 1158-1161

Quantitative Video-Oculography to Help Diagnose Stroke in Acute Vertigo and Dizziness

Toward an ECG for the Eyes

David E. Newman-Toker, MD, PhD; Ali S. Saber Tehrani, MD; Georgios Mantokoudis, MD; John H. Pula, MD; Cynthia I. Guede, RN, BSN; Kevin A. Kerber, MD, MS; Ari Blitz, MD; Sarah H. Ying, MD; Yu-Hsiang Hsieh, PhD; Richard E. Rothman, MD, PhD; Daniel F. Hanley, MD; David S. Zee, MD; Jorge C. Kattah, MD



Oculomotor Finding on Clinical "H.I.N.T.S." Battery (Head Impulse, Nystagmus, Test of Skew)	Vertebrobasilar Stroke	Vestibular Neuritis
Impulse Normal†: horizontal head impulse test (h-HIT) measures normal VOR response[2]	99% (PICA/SCA); 62% (AICA‡)	5%
Fast-phase Alternating: direction-changing horizontal nystagmus[3] evoked by lateral gaze right and left	38%	8%
Refixation on Cover Test: vertical skew deviation[4] as assessed by alternate cover testing	30%	2%
No 'I.N.F.A.R.C.T.' eye signs: unilateral abnormal h-HIT, direction-fixed horizontal nystagmus, no skew deviation	2%	85%

AICA – anterior inferior cerebellar artery; PICA – posterior inferior cerebellar artery; SCA – superior cerebellar artery; VOR – vestibulo-ocular reflex

HIT NORMALE

Nistagmo di tipo centrale

Vertical Skew deviation

Dato complessivo

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

Jorge C. Kattah, MD; Arun V. Talkad, MD; David Z. Wang, DO; Yu-Hsiang Hsieh, PhD, MS; David E. Newman-Toker, MD, PhD

Conclusions—Skew predicts brainstem involvement in AVS and can identify stroke when an abnormal horizontal head impulse test falsely suggests a peripheral lesion. A 3-step bedside oculomotor examination (HINTS: Head-Impulse—Nystagmus—Test-of-Skew) appears more sensitive for stroke than early MRI in AVS. (***Stroke*****. 2009;40:3504-3510.)**

Can Bedside Oculomotor (HINTS) Testing Differentiate Central From Peripheral Causes of Vertigo?

Brian Cohn, MD

The current literature suggests that it is reasonable to use the 3-step HINTS examination as a tool to help determine whether vertigo is peripheral or central in cause. All of the studies used strict inclusion criteria, resulting in moderate- to high-risk populations; caution should be used when applying HINTS to patients at lower risk. Additionally, examiners specifically trained in HINTS performed the examination in all of these studies. Emergency physicians would likely need to undergo additional training to correctly perform and interpret the HINTS examination. The use of the quantitative video-oculography device would allow easy interpretation of the horizontal head impulse test, arguably the most difficult component of the examination,[16] but requires expensive equipment and will need to be studied further. In the meantime, specialty consultation will

Avoiding "HINTS Positive/Negative" to Minimize Diagnostic Confusion in Acute Vertigo and Dizziness

TABLE 1. HINTS Findings That Indicate a Peripheral Versus. Central Lesion in AVS

Lesion	HIT	Nystagmus	Test-of-Skew
Vestibular neuritis	Unilaterally abnormal, in the direction opposite the fast phase of spontaneous nystagmus	Spontaneous unidirectional nystagmus,[a] predominantly horizontal, often with a small torsional component	Normal vertical eye alignment (ie, skew deviation absent)
Stroke or other central nervous system lesion	Usually bilaterally normal (but may be abnormal either unilaterally or bilaterally)	Nystagmus may change direction in different gaze positions (or be unidirectional)	Eyes may be misaligned vertically, indicating presence of skew deviation (or eyes may be normally aligned)

TABLE 2. "Do's and Don'ts" for Documenting HINTS Results

Documentation	HIT	Nystagmus	Test-of-Skew	HINTS Battery
Words to use	Normal vs abnormal vestibular reflexes; note the specific canal plane and side tested (eg, right horizontal HIT normal, left horizontal HIT abnormal)	Nystagmus present vs absent; if nystagmus present, note whether spontaneous or provoked, the nystagmus vector, the direction of the fast phase, and any changes with gaze or position	Skew deviation present vs absent; if skew present, note which eye is higher, and, ideally, measure the degree of deviation in prism diopters	Peripheral vs central pattern; always note the individual components in addition to the overall impression
Words not to use	Positive vs negative; central vs peripheral	Normal vs abnormal; avoid general descriptions such as "direction-changing" or "central" without further details	Normal vs abnormal; central vs peripheral	Positive vs negative; normal vs abnormal

When (and when not) to use HINTS: HINTS is valid only in patients who have continuous vertigo or dizziness and spontaneous nystagmus (ie, AVS). HINTS should not be used in patients with positional vertigo, or its results will be misleading.[8] For example, if HINTS is erroneously applied in a patient with typical posterior canal benign paroxysmal positional vertigo (BPPV), the HIT will reveal normal vestibular reflexes bilaterally, leading to a false interpretation of "stroke."

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VESTIBOLOGY

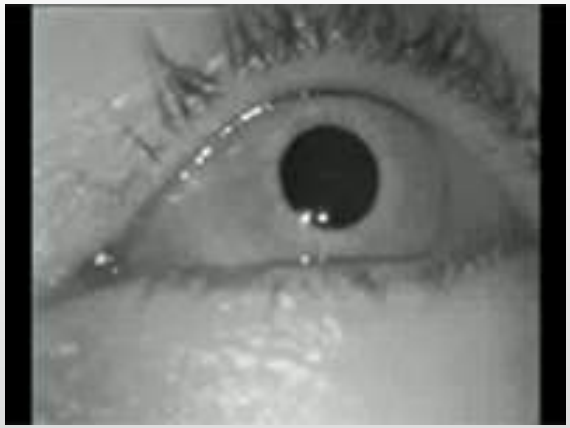
STANDING, a four-step bedside algorithm for differential diagnosis of acute vertigo in the Emergency Department

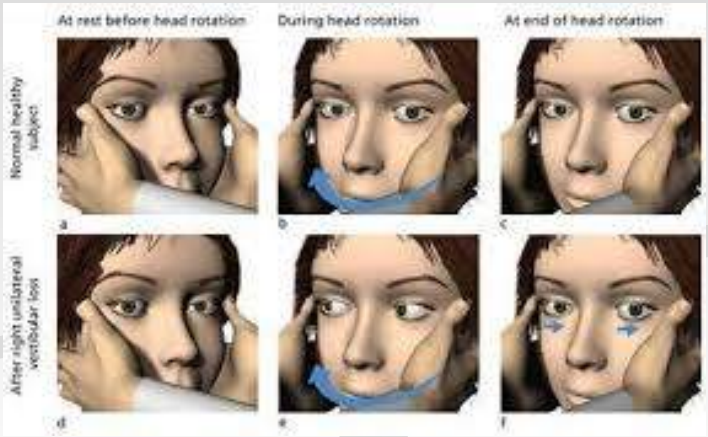
Lo STANDING, un algoritmo bedside a quattro step per la diagnosi differenziale delle vertigini acute nel Dipartimento di Emergenza

S. VANNI[1], R. PECCI[2], C. CASATI[1], F. MORONI[1], M. RISSO[1], M. OTTAVIANI[1], P. NAZERIAN[1], S. GRIFONI[1], P. VANNUCCHI[2]

[1] Department of Emergency Medicine, Careggi Hospital, University of Firenze, Italy; [2] Department of Surgical Sciences and Translational Medicine, Unit of Audiology, Careggi Hospital, University of Firenze, Italy

The aim of this pilot study was to preliminarily assess the reliability and diagnostic accuracy of a simple structured clinical algorithm (STANDING: SponTAneous Nystagmus, Direction, head Impulse test, standiNG) that we developed to differentiate central from non-central AV in the emergency setting, and to evaluate in an explorative fashion if its use might be associated with a reduction of the neuroimaging burden and hospitalisation.





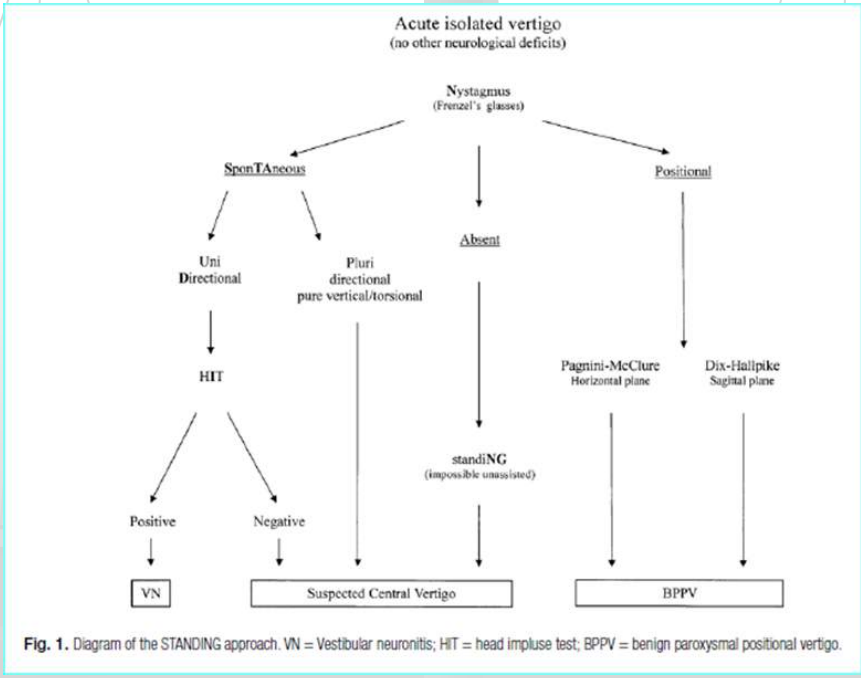


Fig. 1. Diagram of the STANDING approach. VN = Vestibular neuronitis; HIT = head impluse test; BPPV = benign paroxysmal positional vertigo.

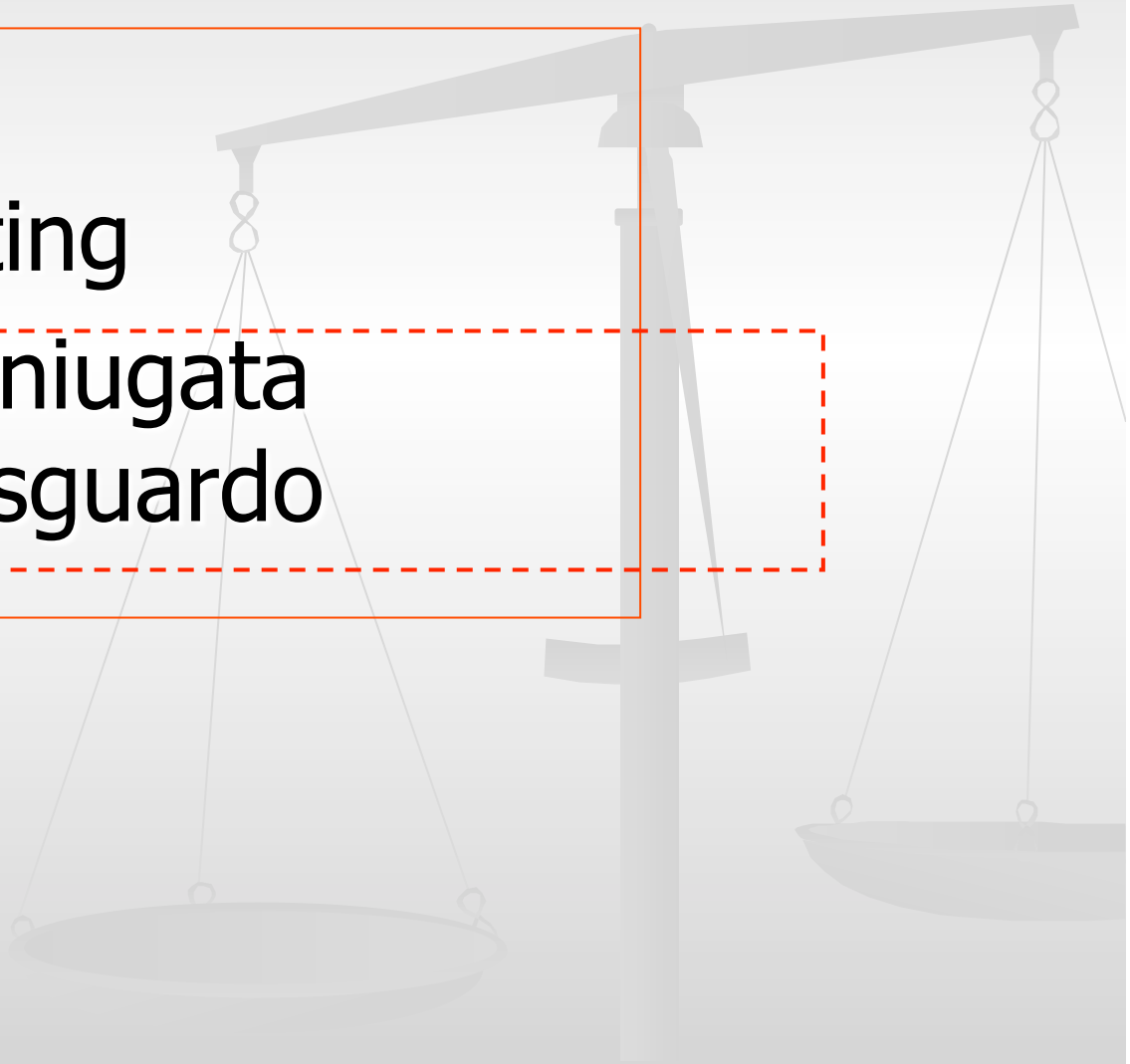
Periferico o centrale?

I limiti di ognuna di queste metodiche sono

- La possibile associazione tra danno vascolare acuto periferico e centrale
- La possibile sequenzialità evolutiva del danno periferico-centrale
- Per l'HTT clinico:
  - La tecnica di esecuzione (testa flessa di 30°; impulsi"random" non prevedibili)
  - l'interpretazione:ad occhio nudo la saccade è rapida, piccola e fugace! Il V-HIT migliora la sensibilità
  - Individuazione di un danno labirintico preesistente

Elementi cardine

- HINTS
- Positional testing
- Deviazione coniugata laterale dello sguardo



H-CGD in posizione seduta

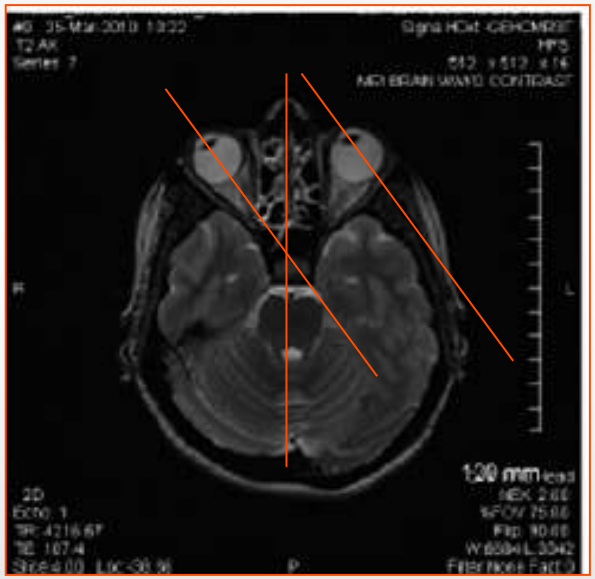
Ocular lateropulsion

- common in lateral medullary infarcts (Baloh et al, *Ann NY Acad Sci*, 1981; Waespe and Wichmann, *Brain*, 1990)
- conjugate eye movement to the side of infarct
- best observed after removal of visual fixation (look for corrective eye movement to the midline after brief sustained eye closure)

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES
Issue: *Basic and Clinical Ocular Motor and Vestibular Research*

Ocular lateropulsion as a central oculomotor sign in acute vestibular syndrome is not posturally dependent

Jorge C. Kattah,[1] John Pula,[1] and David E. Newman-Toker[2]



Vestibular neuritis

Table 1. Ocular lateropulsion and radiographic h-CGD in patients with acute vestibular syndrome

Patient	Diagnosis	Lesion side	Clinical OL OL direction	Image 1 type, h-CGD side (mean degrees)	Image 2 type, h-CGD side (mean degrees)	Imaging concordance (time interval)
1	Stroke (multiterritory cerebellar hemisphere)	Left	Right	CT, right (38.5)	NA [no MRI, pacemaker]	NA
2	Vestibular neuritis	Right	Absent	CT, right (24.2)	MRI, right (32.6)	Yes (8 h)
3	Stroke (inferolateral cerebellum)	Left	Absent	MRI, absent (0.75)	NA	NA
4	Stroke (MCP, nodulus, dentate nucleus)	Left	Right	CT, right (25.1)	MRI, right (33.9)	Yes (24 h)
5	Stroke (inferolateral cerebellum)	Right	Left	CT, left (35.5)	MRI, left (19.8)	Yes (5 h)
6	Vestibular neuritis	Right	Absent	MRI, right (24.6)	NA	NA
7	Stroke (lateral medulla, inferior cerebellum)	Left	Left	MRI, left (28.1)	CT, left (30.2)	Yes (4 days)
8	Vestibular neuritis	Left	Absent	CT, left (24.6)	NA (no MRI, wide shoulders)	NA
9	Stroke (lateral medulla)	Right	Right	MRI, right (19.5)	CT, right (22.3)	Yes (10 h)
10	Stroke (lateral medulla, inferior cerebellum)	Right	Right	MRI, right (32.1)	CT, right (39.5)	Yes (24 h)
11	Vestibular neuritis	Left	Absent	CT, left (25.5)	MRI, left (20.5)	Yes (2 weeks)
12	Demyelinating plaque (near dentate nucleus)	Right	Left	MRI, left (19.9)	NA	NA
13	Vestibular neuritis	Right	Absent	MRI, right (28.3)	NA	NA
14	Stroke (lateral medulla)	Left	Absent	MRI, absent (1.7)	MRI, absent (12.0)	Yes (72 h)
15	Stroke (lateral medulla)	Left	Left	MRI, left (26.3)	NA	NA
16	Stroke (lateral medulla, inferior cerebellum)	Right	Absent	MRI, absent (13.6)	NA	NA
17	Vestibular neuritis	Right	Absent	CT, NA (poor image quality)	MRI, right (17.2)	NA

Abbreviations: OL, ocular lateropulsion; CT, computerized tomography; MCP, middle cerebellar peduncle; MRI, magnetic resonance imaging; NA, not available.

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Ocular lateropulsion as a central oculomotor sign in acute vestibular syndrome is not posturally dependent

Jorge C. Kattah,[1] John Pula,[1] and David E. Newman-Toker[2]

Results

- Supine h-CGD in supine position was found in ~ equal peripheral vestibular (38-57%) and central posterior fossa(38%) lesions
- Upright h-CGD found only in central posterior fossa lesions

Conclusions

- In the acute vestibular syndrome upright h-CGD is present only in stroke patients.
- **Clinicians should routinely assess upright h-CGD after brief sustained eye closure in patients with acute vestibular syndrome.**

H-CGD in upright position

Audio-vestibular findings after AICA infarction

Lee et al, *Stroke*, 2009

- Results in 82 patients with AICA infarction:
  - 60% of combined audio-vestibular involvement
  - 5% of isolated vestibular labyrinth involvement
  - 4% of isolated cochlear labyrinth involvement
- Conclusions
  - combined loss of auditory and vestibular function should call attention to vascular involvement
  - viral illness rather presents with isolated vestibular or cochlear loss

Periferico o centrale?

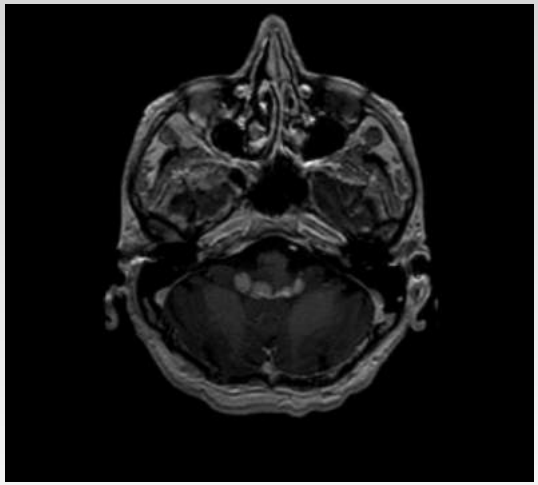
Quando la RMN ”ragionata” per vertigine acuta senza iniziali sintomi/segni neurologici?

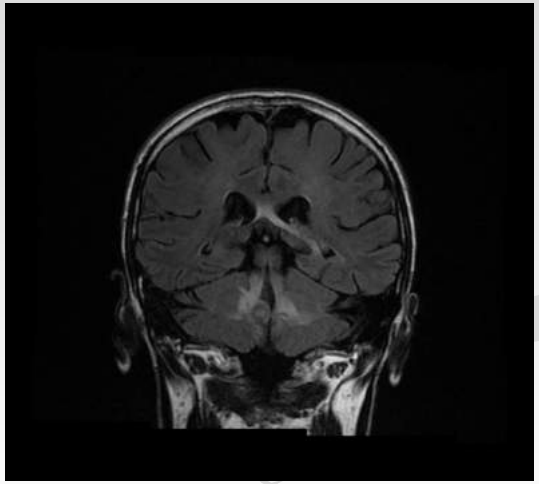
- **Nistagmi a valore topodiagnostico centrale**
- **HTT negativo**
- **Vertical Skew deviation**
- **H-GCD presente in posizione eretta/seduta**
- **Ice test simultaneo negativo**
- **Gravi/multipli fattori di rischio vascolare**
- **Persistenza/ingravescenza della sintomatologia con il passare dei giorni**
- **Più ovviamente se comparsi altri sintomi neurologici**

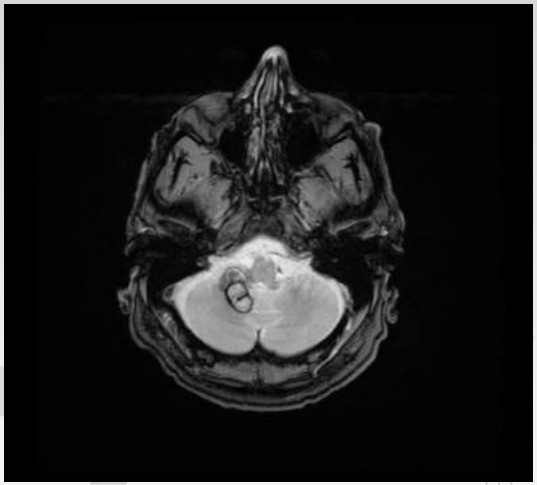
Neuroimaging

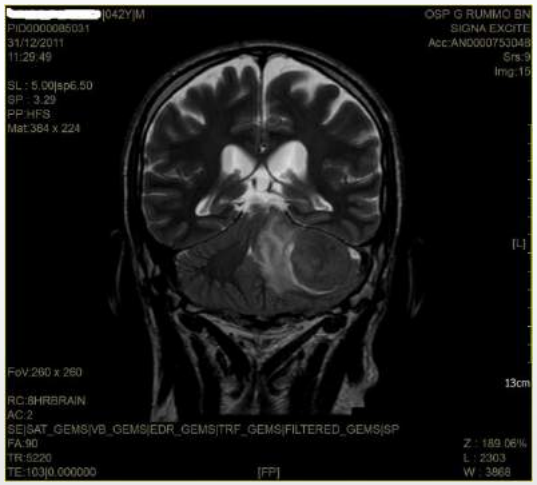
- **CT scan sensitivity** for identifying a posterior fossa stroke in the acute setting is **26%** (Chalela et al, *Lancet*, 2007)
- **MRI with DWI within 48h** of acute vestibular symptom onset is **false negative in 12%** of brainstem and cerebellar infarctions (Kattah et al, Stroke , 2009; Oppenheim et al, *AJNR Am J Neuroradiol*, 2000)

- *Recommendation*: The presence of vascular risk factors, particularly in an older patient, should tilt the balance towards obtaining an MRI of the brain, even if clinical features are consistent with a peripheral cause.





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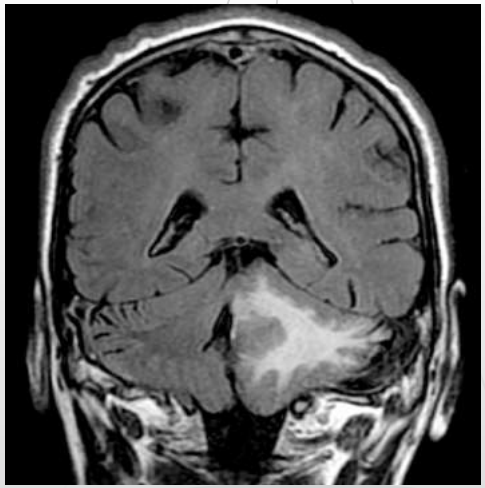

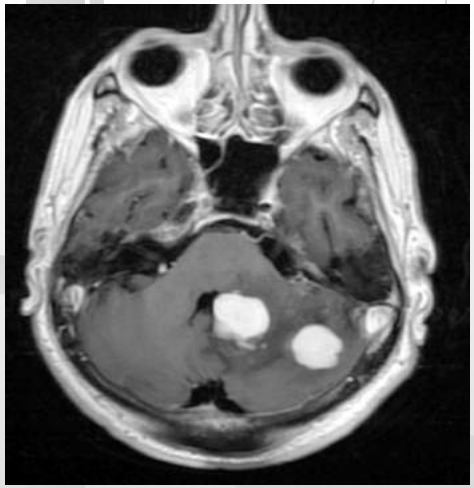

Periferico o centrale che sia…

Nel soggetto anziano è sempre necessaria l'osservazione clinica in ambiente ospedaliero, anche per l'impatto che il sintomo vertigine ha sul paziente, sulla sua nutrizione ed idratazione, *sulla sua compliance verso le terapie orali già in atto*, ecc.

Table 3. Absolute and relative frequency of the number of medications used by 120 elderly patients with chronic vestibular dysfunction.

	Categories	Absolute Frequency (n)	Relative Frequency (%)
Number of medications	Non-user	4	3,3
	1 or 2 medications	35	29,2
	3 or 4 medications	37	30,8
	5 or more medications	44	36,7

Clinical evaluation of elderly people with chronic vestibular disorder

Juliana Maria Gazzola[I], Fernanda Freitas Ganança[I], Mayra Cristina Aratani[II], Monica Rodrigues Perracini[III], Maurício Malavasi Ganança[IV]

Take-home messages

- Il DVA è la seconda causa di vertigine acuta
- Esso può interessare tutte le fasce di età
- La diagnosi NECESSITA della presenza di un nistagmo spontaneo deficitario e delle sue "modulazioni"
- L' evoluzione è benigna nelle forme infiammatorie, a possibile rischio-vita nelle forme centrali (cd pseudoneurite)
- La diagnosi clinica differenziale è possibile con un idoneo esame vestibolare e neurologico: utile l'algoritmo HINTS, specie in presenza di nistagmi centrali (Gaze, Rebound ecc.) e l' H-CGD
- La diagnostica per immagini va eseguita in modo mirato
- Mai sottovalutare una vertigine acuta in un paziente anziano o a rischio cardiovascolare

Take-home messages

- Red flags in vertigo patients with sparse symptoms:
  - normal **head impulse test**
  - **skew deviation**, skew torsion, ocular tilt reaction
  - Central spontaneous **nystagmus** (upbeat nystagmus etc.)
  - look for **upright horizontal conjugate gaze deviation** after brief sustained eye closure
- Evaluate **imaging** in 'vasculopathic' patients

Quando ti metterai in viaggio per **Itaca**
devi augurarti che la strada sia lunga
fertile in avventure e in esperienze.
.....

Devi augurarti che la strada sia lunga,
che i mattini d'estate siano tanti
quando nei porti – finalmente e con che gioia -
toccherai terra tu per la prima volta
.....

Sempre devi avere in mente **Itaca** –
Raggiungerla sia il tuo pensiero costante.
Soprattutto, pero', non affrettare il viaggio
.....

E se la trovi povera, non per questo **Itaca**
ti avra' deluso.
Fatto ormai savio, con tutta la tua esperienza
addosso
gia' tu avrai capito cio' che **Itaca** vuole significare

Konstantinos Kavafis