Il Nistagmo da Iperventilazione

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THE DIAGNOSIS OF EAR DISEASES WITHOUT INSTRUMENTS.[1]

By HUNTINGTON RICHARDS, M.D.,

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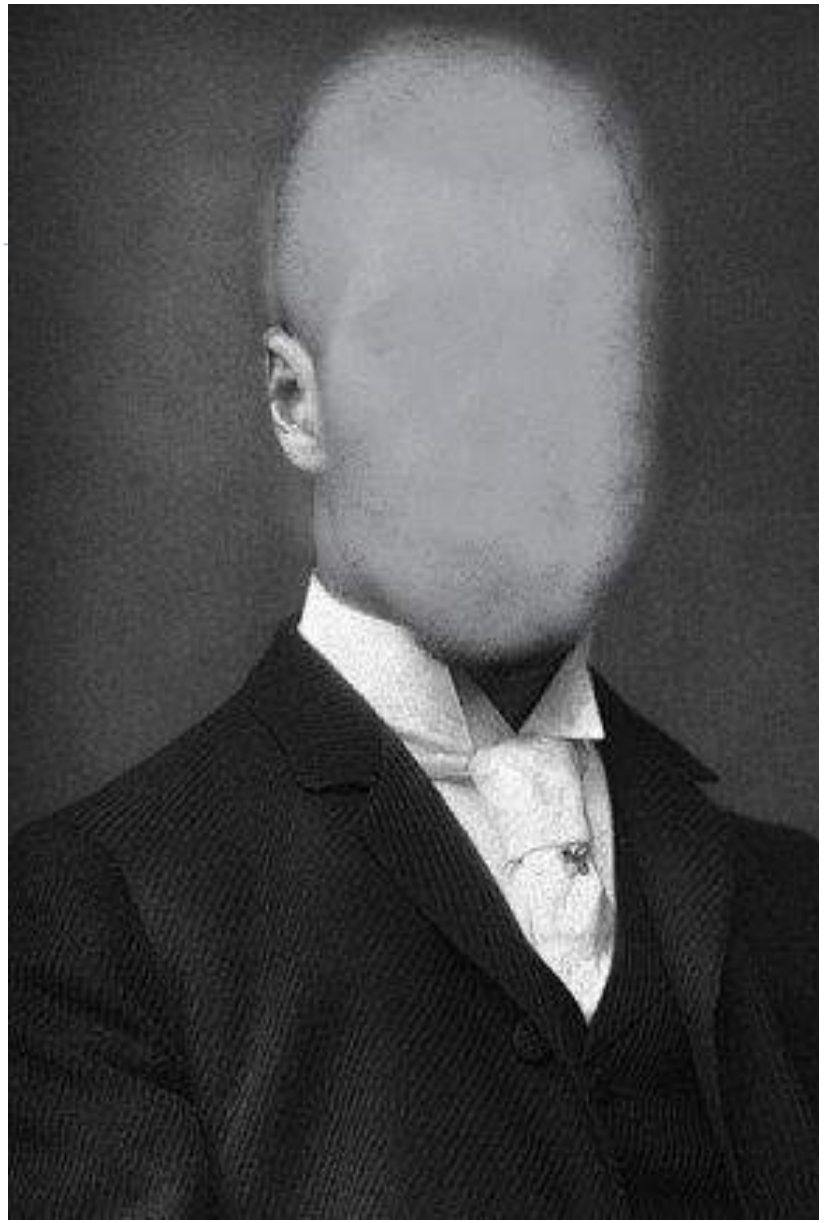
THE title which I have chosen for the following brief paper is one which will surely be accounted a contradiction in terms, both by an otological specialist, and by a general practitioner who has had any considerable experience in diseases of the ear. The subject-matter of the paper is, nevertheless, one which should interest medical men, whether they be "specialists" or "family physicians;" and if it fail to do so in this instance, or if the paper prove quite barren in suggestions of a practical kind, it will be the fault of the writer alone.

Accurate diagnosis, in the case of a vast majority of diseases of the ear, is certainly impossible without the aid afforded by properly-constructed instruments, and is not invariably easy of accomplishment, even with this aid. On the other hand, it will not be disputed that in many cases of aural disease relief is first sought, and must be first sought, at the hands of a physician, who either has no good apparatus for examining the ear, or who lacks experience in the handling of such apparatus. Moreover, in many cases of aural disease, right diagnosis in the very earliest stage of the disease is a matter of the greatest importance. Particularly is this true of that class of cases wherein a disturbance in the function of hearing is the leading symptom; for, unless the family physician be on the watch for the first beginning of the disease and knows how to recognize its symptoms, an incurable deafness is but too often the result.

Medical Record; New York, Vol 33, Fasc 13 , (Mar 31, 1888): 354



- Author caption: By Huntington Richards, M. D., *New York, N. Y.*
- Medical degree, College of Physicians and Surgeons, New York, 1877.
- Postgraduate studies, Vienna, 1879-80.
- Assistant surgeon, New York Eye and Ear Infirmary, 1881.
- Aural surgeon, New York Eye and Ear Infirmary, 1886.
- Chief aural clinician, Vanderbilt Clinic.

L'iperventilazione è l'aumento della ventilazione polmonare eccedente le effettive necessità metaboliche di eliminazione di CO_2

Figura 1

Fisiopatologia dell'iperventilazione

- Diminuzione della Pa CO_2
- Aumento del pH arterioso
- Diminuzione del Ca^{++} libero ionizzato extracellulare
- Vasocostrizione arteriolare da ipocapnia con ipossia cellulare
- Ipereccitabilità neuronale
- Diminuzione del flusso cerebrale e labirintico
- Diminuzione della pressione liquorale

Fisiopatologia dell'iperventilazione

- L'alcalosi plasmatica interferisce con la curva di dissociazione dell'Hb contribuendo ulteriormente all'ipossia cellulare
- L' alcalosi attiva canali del Ca++ voltaggio dipendenti favorendo l'ingresso dello ione nelle cellule, in scambio con K+ e Na+
- Le cellule per compensare l'alcalosi extra cellulare cedono H+ (alcalosi intracellulare)
- L'ipocapnia e l'alcalosi hanno effetto eccitatorio sui tessuti neurali

Precisando che…

- Esiste una notevole resistenza della barriera emato-encefalica alle variazioni di Ca++ plasmatico
- Tale barriera viene focalmente meno in condizioni patologiche quali la SM ed il neurinoma per la presenza di foci di demielinizzazione (Davis)….
- …ma anche nelle neurite vestibolare (Matsuo, 1986,1989), per un periodo temporalmente limitato (Kuwabara, 2006, in polineuropatie periferiche)

Iperventilazione e nistagmo

Attraverso i meccanismi metabolici ed acido-base suddetti l'iperventilazione è capace di

- **rompere** i meccanismi centrali di compenso di un'asimmetria vestibolare periferica, determinando la comparsa o il rinforzo di un ny "deficitario" *(Sakellari, Bance)*
- **peggiorare** la performance inibitoria cerebellare, slatentizzando/rinforzando un Down-beat ny *(Walker)*
- **migliorare focalmente e temporaneamente** la conduzione lungo vie demielinizzate, determinando la comparsa di ny "irritativo" o l'inibizione di un ny spontaneo preesistente *(Leigh e Zee, Bance, Walker, Minor, Choi, Califano)*

Iperventilazione ed induzione di dizziness (o altro)

- Nei (non molti) casi di comparsa di sensazioni soggettive di dizziness, di testa vuota, di mancamento, ecc. non è stato da noi mai riscontrato nistagmo
- Non è stata da noi mai osservata tetania, talvolta solo fugaci parestesie alle estremità *(nel caso, far respirare in un sacchetto di carta)*
- Meglio evitare negli epilettici

Standardizzazione dell'iperventilazione

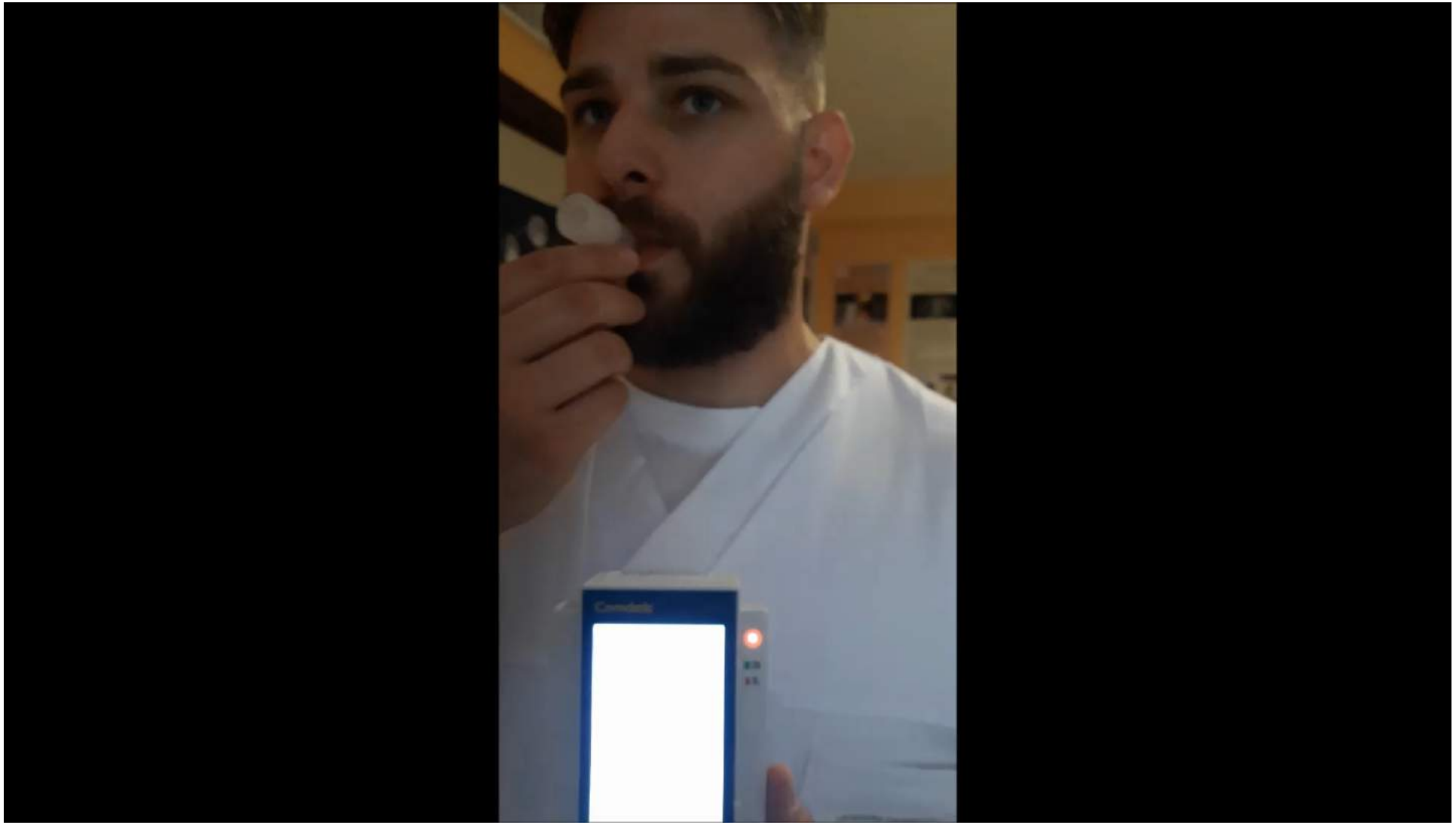
- Durata della iperventilazione: 60-90"
- Walker e Sakellari praticano 70" a cicli di circa 6", enfatizzando la profondità dell'atto respiratorio

- Per una iperventilazione di 30" *(Sakellari)*
  - Riduzione della CO2 plasmatica del 21%
  - Riduzione della pACO2 di 6 mmHg
  - Riduzione Ca++ extracellulare
  - Innalzamento pH liquorale

Standardizzazione dell'iperventilazione

Difficoltà difficilmente superabile è che la profondità e la forza dell'atto respiratorio sono diversi in relazione alle capacità del paziente (giovane sano vs. anziano broncopatico cronico)

Standardizzazione dell'iperventilazione



Standardizzazione dell'iperventilazione

Nostra esperienza di standardizzazione con misurazione «side-stream» della $P_{Et\ CO2}$

TABLE 1. *Mean P_{EtCO2} values (mm Hg) in the control, schwannoma, and chronic vestibular neuritis groups during the HVT*

	Baseline	30 s	60 s	120 s	180 s	Minimum value
Control group	34.1 ±1.28	20.4 ±1.28	12.6 ±1.17	27.7 ±1.86	30.8 ±1.73	12.0 ±1.99
Schwannoma group	33.9 ±1.53	20.2 ±1.37	12.1 ±1.66	28.9 ±1.58	31.7 ±1.38	12.1 ±1.74
Chronic vestibular neuritis group	34.6 ±1.34	20.5 ±1.19	12.5 ±1.21	26.9 ±1.24	30.7 ±1.67	12.2 ±1.71

The P_{EtCO2} differences between the healthy controls and the patients affected by an VIIIth cranial nerve schwannoma or chronic vestibular neuritis were not significant at any time point. The lowest P_{EtCO2} values were registered between 50 and 75 seconds from the beginning of the HVT.

Hyperventilation-Induced Nystagmus in Patients with Vestibular Schwannoma

Luigi Califano, Giuseppina Iorio, Francesca Salafia, Salvatore Mazzone, and Maria Califano

Otology & Neurotology
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Questi valori sono analoghi (dati non pubblicati) a quanto riscontrato anche nel Deficit vestibolare acuto monolaterale ed in patologie centrali

Standardizzazione dell'iperventilazione

In the VS and CVN groups, the P_{EtCO2} value that triggered HVIN was homogeneous, at approximately 16 mm Hg. To standardize the test, we suggest that the HVT should be interrupted at this P_{EtCO2} value if a capnographic measurement is performed; otherwise, more generally, if a capnographic measurement is not performed, a 60-second hyperventilation is sufficient to lower the P_{EtCO2} to levels that act on the vestibular system, if a vestibular disease is present.

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Iperventilazione e nistagmo

La modalità di osservazione influisce sulla sensibilità del test

1° "Three dimensional scleral search coil" elevata sensibilità e possibilità di valutazione quantitativa e qualitativa dei vettori nistagmici (Minor, Walker)

2° Videonistagmoscopia (Bance)

3° Occhiali di Frenzel

4° ENG

Iperventilazione e nistagmo

- Il test ha una bassa percentuale di "falsi positivi" (< 2%)
- L'individuazione di parametri quantitativi di durata e di velocità praticamente li azzera (Bance, Hain, Califano)
- Più la tecnica di osservazione è sensibile, più la specificità potrebbe diminuire

J. Neurol. Neurosurg. Psychiat., 1970, **33**, 723-732

Effect of intravenous sodium bicarbonate, disodium edetate (Na_2EDTA), and hyperventilation on visual and oculomotor signs in multiple sclerosis

FLOYD A. DAVIS, FRANK O. BECKER, JOEL A. MICHAEL, AND ERIC SORENSEN

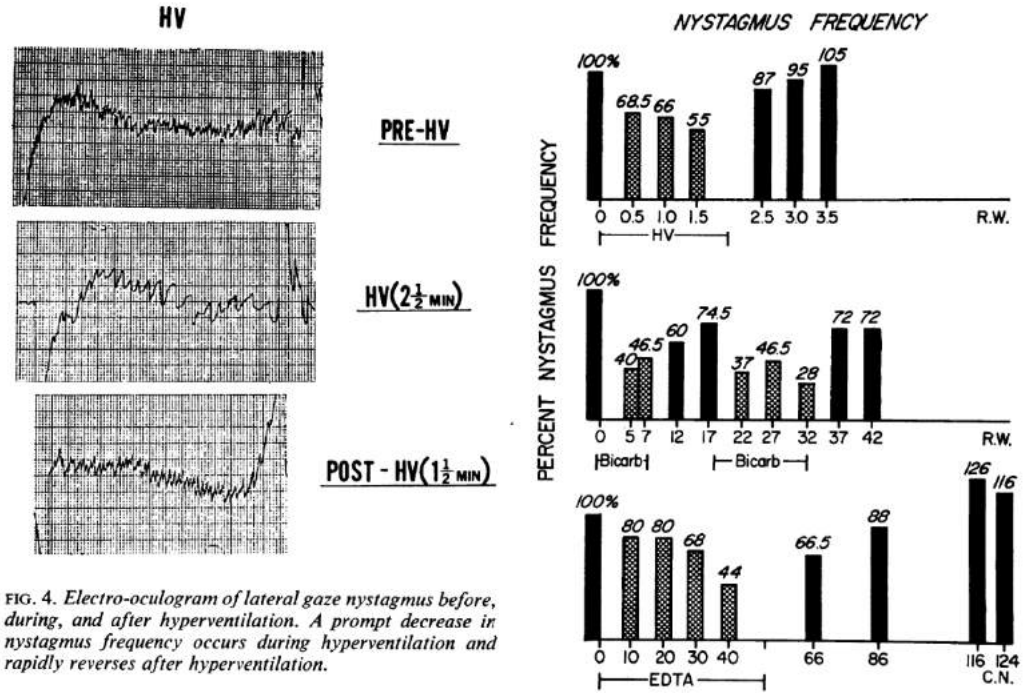

FIG. 4. *Electro-oculogram of lateral gaze nystagmus before, during, and after hyperventilation. A prompt decrease in nystagmus frequency occurs during hyperventilation and rapidly reverses after hyperventilation.*

The improvement in clinical findings in MS with hyperventilation, $NaHCO_3$ and Na_2EDTA is probably due, at least in part, to a lowering of serum ionized calcium, which increases axonal excitability and in turn produces an increase in the conduction safety factor. There is evidence for a formidable blood-CSF barrier to calcium in normals (Soffer and Toribara, 1961; Schain, 1964), multiple sclerosis patients (Merritt and Bauer, 1931), and patients with disorders of calcium metabolism (Hebert, 1933). However, it is conceivable that there is a localized breakdown of the blood-brain barrier to calcium at the site of MS plaques which might not produce a marked change in CSF calcium. This is supported by the finding of Broman (Broman, 1944) that increased uptake of trypan blue occurs in MS plaques. Thus, a low serum ionized calcium might alter brain calcium in MS where it counts most—that is, at the site of the demyelinating lesion. This idea of a breakdown

Brain (1997), **120,** 1659–1673

The effects of hyperventilation on postural control mechanisms

V. Sakellari,[1,*] A. M. Bronstein,[1] S. Corna,[1,†] C. A. Hammon,[1] S. Jones[2] and C. J. Wolsley[1]

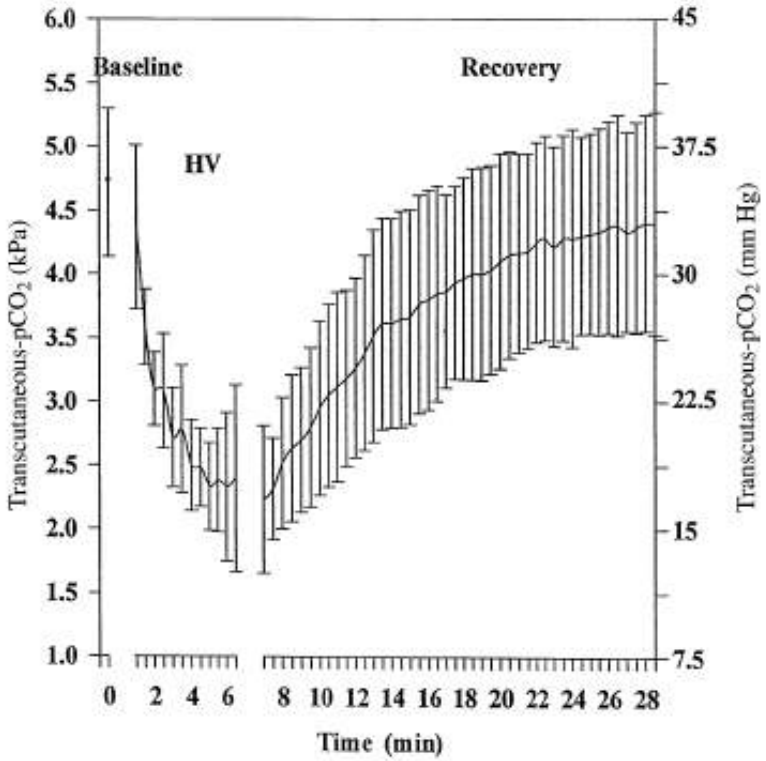

Fig. 2 Transcutaneous CO_2 changes (mean and SD) in six normal subjects during hyperventilation (HV) and during the recovery from hyperventilation, in kPa (left) and mmHg (right). The corresponding changes in peripheral SAPs and scalp SEPs in these subjects are shown in Fig. 3.

Six patients with unilateral peripheral lesions (four female), mean age 57 years, range 49–68 years were tested. Three of them had undergone vestibular neurectomies for treatment of refractory vertigo (2.5, 3 and 12 months before testing), three had long-standing unilateral absence of nystagmic response (canal paresis) to irrigation at 30 and 44°C (one from presumed viral labyrinthitis, one from unilateral Meniere's disease and one a probable Herpes zoster cochleo/vestibular neuritis). Two groups of normal subjects were used as controls for the video-oculography (eye-movement) recordings, a group of six subjects (four females and two males; mean age 28 years, range 22–42 years) and a group of six older subjects (two females, four males; mean age 54 years, range 47–59 years).

Before hyperventilation, none of the patients had consistent nystagmus in the light. In the dark, horizontal beats of nystagmus of 0.5–2°/s were present in three patients. A weak downbeat component was observed in the video-images in two patients. Torsional nystagmus was not clearly observed in any of the patients. Hyperventilation triggered the onset of nystagmus in three patients in whom it was not initially present and established regularly beating nystagmus in the three with spontaneous beats.

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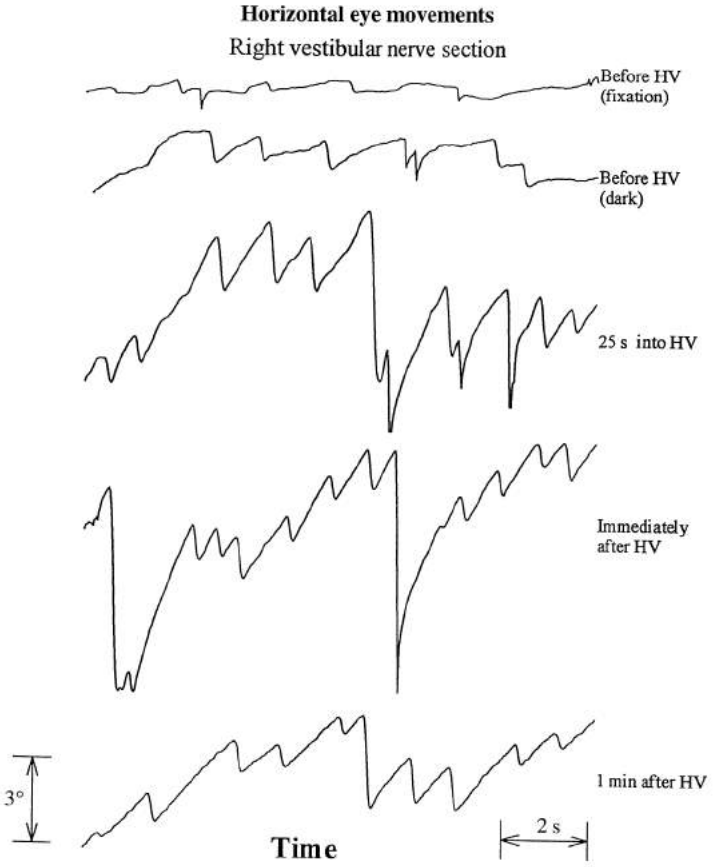

Fig. 8 Spontaneous horizontal eye movements during straight ahead gaze in a patient with right-sided vestibular nerve section, before, during and after hyperventilation (HV). Note the exacerbation of the nystagmus by hyperventilation. Upwards deflections indicate rightwards eye movements.

The direction of beat of the spontaneous nystagmus (fast phase) was towards the healthy side in three patients. In two patients the direction of the nystagmus was reversed (towards the lesion side) and remained so after the hyperventilation. The latter two patients had neither vestibular nerve section nor severe hearing loss. Finally, in one of the patients with vestibular nerve section, the direction of the nystagmus reversed from beating towards the affected side (before hyperventilation in the dark) to beating towards the healthy side (after hyperventilation in the dark).

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In summary, this study confirms that hyperventilation causes an objective disturbance of postural control. The causes of this unsteadiness are likely to be multiple, in the light of the widespread metabolic changes induced by hyperventilation. However, in the main, the effects are not mediated by vestibular mechanisms but rather by interference with somatosensory and vestibular-compensation processes. The findings could have significance for the treatment of patients with balance disorders, since the rehabilitation specialist should be aware that hyperventilation, which could arise as part of anxiety, could aggravate patients' symptoms and signs. Similarly, in the light of our results showing that hyperventilation can decompensate an underlying vestibular disorder, symptoms of dizziness and unsteadiness should only be attributed solely to hyperventilation in the absence of findings in a careful neuro-otological examination.

The effect of hyperventilation on downbeat nystagmus in cerebellar disorders

Article abstract—Hyperventilation can affect nystagmus in patients with vestibular disorders. However, the effects on nystagmus in patients with cerebellar disease have not been systematically studied. Using the magnetic field search coil technique, we studied the effects of hyperventilation on nystagmus in a series of cerebellar patients. In four of eight patients, hyperventilation produced an increase in the slow-phase velocity of downbeat nystagmus. We speculate that this effect may be mediated through metabolic effects on cerebellar calcium channels. **Key words:** Hyperventilation—Nystagmus—Downbeat nystagmus—Cerebellum.

NEUROLOGY 1999;53:1576–1579

Mark F. Walker, MD; and David S. Zee, MD

Discussion. Our results show that hyperventilation may enhance spontaneous downbeat nystagmus in some cerebellar patients. Downbeat nystagmus is a common finding with lesions of the vestibulocerebellum and its brainstem pathways. It has been suggested that there is an underlying upward eye velocity bias in the central vestibular[5] or pursuit system[6] or in the peripheral vestibular system[7]; this bias is assumed normally to be inhibited by the cerebellum. According to this hypothesis, when cerebellar inhibition is disrupted by disease, the upward bias is uncovered, resulting in a spontaneous downbeat nystagmus. Downbeat nystagmus in cerebellar patients may be modified by a number of factors, including orbital position, head position and movement,[5] head shaking,[8] and caloric stimulation.[5] We have now shown that downbeat nystagmus may also be increased by hyperventilation.

Hyperventilation and calcium channels. An increase in downbeat nystagmus during hyperventilation may be related to a functional worsening of the cerebellar lesion, further decreasing inhibitory cerebellar output and thus increasing nystagmus. Perhaps this could be mediated through metabolic effects on cerebellar calcium channels. The vestibulocerebellum is rich in P/Q-type voltage-gated calcium channels, and these channels are known to be important in some forms of cerebellar disease.[9,10] These cerebellar calcium channels are sensitive to pH changes, which may explain the episodic symptoms observed in some of these patients as well as the response to acetazolamide.[10] Perhaps our patients also have abnormal ion channels that render them particularly sensitive to the alkalosis produced by hyperventilation. With this in mind, it would be interesting to know whether patients with defined calcium channel abnormalities (e.g., SCA6 and EA-2) are more susceptible to hyperventilation and whether acetazolamide prevents hyperventilation-induced nystagmus. It would also be of interest to

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Table *Cerebellar patients: Age, sex, diagnosis, slow-phase velocity (SPV) of spontaneous downbeat nystagmus (measured in the dark), and presence or absence of a hyperventilation (HV) effect on vertical SPV*

Patient	Age, y	Sex	Diagnosis	SPV (°/s)	HV effect
1	33	F	Sporadic cerebellar degeneration	3.2	Y
2	43	F	Sporadic cerebellar degeneration	7.8	Y
3	54	M	Vestibulocerebellar syndrome	3.5	N
4	55	M	Probable MSA	0.7	N
5	61	M	Sporadic cerebellar degeneration, bilateral vestibulopathy	7.5	N
6	66	F	Sporadic cerebellar degeneration	3.1	N
7	71	M	Probable paraneoplastic syndrome	1.6	Y
8	75	F	Familial cerebellar degeneration	1.4	Y

MSA = multiple system atrophy.

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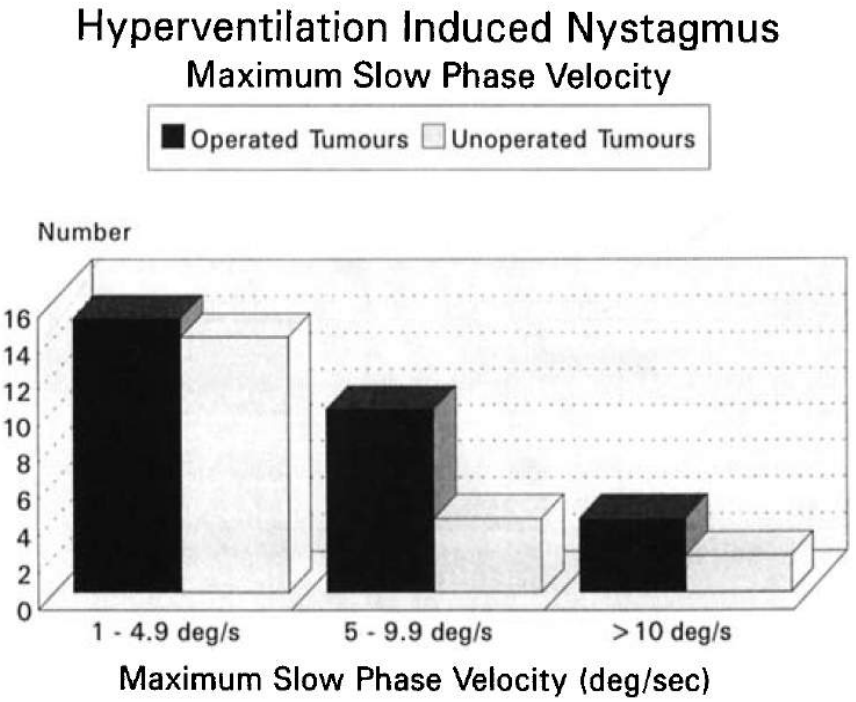
This report further emphasizes the use of hyperventilation in the evaluation of disorders of equilibrium and balance. Not only may it be useful in the identification of a peripheral lesion, such as an acoustic neuroma, but it may also enhance downbeat nystagmus in patients who have a cerebellar cause for their imbalance.

Vestibular Disease Unmasked by Hyperventilation

Manohar L. Bance, MSc, FRCS(C); Martin O'Driscoll, MSc; Nimesh Patel, MB, ChB; Richard T. Ramsden, FRCS

Hyperventilation-induced dizziness is often thought to be psychogenic, but its effects in the presence of known vestibular disease have not been adequately examined. In this study hyperventilation was tested in two models of vestibular disease. These were, first, patients with profound unilateral vestibular deficit (prior translabyrinthine acoustic neuroma resection [postsurgery group]) and, second, patients with variable unilateral vestibular deficit (unoperated unilateral acoustic neuroma [presurgery group]). Patients were hyperventilated for 90 seconds. Using infrared videonystagmography, 100% of the 32 postsurgery patients and 82% of the 28 presurgery patients developed nystagmus with hyperventilation. Hyperventilation was more sensitive than head shake for eliciting nystagmus in these models. The false-positive rate for nystagmus in 29 normal volunteers was 3.5% for hyperventilation and 10% for head shake. Our results show that hyperventilation can unmask underlying vestibular disease.



J Otolaryngol. 2002 Jun;31(3):140-3.

Is hyperventilation-induced nystagmus more common in retrocochlear vestibular disease than in end-organ vestibular disease?

Robichaud J[1], DesRoches H, Bance M.

Hyperventilation-induced nystagmus (HVIN) has previously been shown by the senior author to be common in patients with both acoustic neuromas and following resection. The recurrent study's aim was to examine if HVIN was specific for retrocochlear pathology. To test this, the incidence of HVIN in 24 patients with confirmed acoustic neuroma was compared with its incidence in 38 patients with end-organ vestibular disease (defined as a greater than 25% reduction in caloric testing). Hyperventilation was carried out for 90 seconds. The results showed that 58% of the acoustic neuroma group were positive for HVIN versus 18% of the end-organ group. This difference was very significant on chi-square testing ($p < .002$). Hyperventilation-induced nystagmus appears to be much more prevalent in retrocochlear pathology than in end-organ pathology.

Hyperventilation-induced nystagmus in patients with vestibular schwannoma

Lloyd B. Minor, MD; Thomas Haslwanter, PhD; Dominik Straumann, MD; and David S. Zee, MD

Article abstract—*Objective:* To analyze the nystagmus evoked by hyperventilation in patients with unilateral vestibular schwannoma and to use this information to predict the effects of hyperventilation on individual ampullary nerves. *Methods:* Three-dimensional scleral search coil eye movement recording techniques were used to record the magnitude and time course of eye movements in six patients with unilateral vestibular schwannoma and hyperventilation-induced nystagmus. The presenting complaints in five of these patients were vertigo or dysequilibrium. *Results:* The eye movement response to hyperventilation was a "recovery" nystagmus with slow-phase components corresponding to excitation of the affected vestibular nerve. Projection of the eye velocity vector into the plane of the semicircular canals revealed that fibers arising from the ampulla of the horizontal canal were most affected by hyperventilation with lesser activation of fibers to the superior canal and smaller, more variable responses from posterior canal fibers. *Conclusions:* The three-dimensional characteristics of the nystagmus evoked by hyperventilation in patients with vestibular schwannoma provide insight into the vestibular end organs affected by the tumor and the mechanism responsible for the nystagmus. This finding indicates that hyperventilation resulted in a transient increase in activity from these partially demyelinated axons. **Key words:** Acoustic neuroma—Recovery nystagmus—Three-dimensional eye movements—Hyperventilation—Vestibular schwannoma.

NEUROLOGY 1999;53:2158–2167

Table 3 *Symptoms and signs in patients with hyperventilation-induced nystagmus and vestibular schwannoma*

				Signs		
Patient	Age, y	Sex	Symptoms	General	Hyperventilation-induced nystagmus slow phases	Cranial MRI with gadolinium
1	40	M	1-y history of dizziness and nausea	Normal	Right, up, clockwise	Enhancing mass left IAC with 8-mm extension into CPA
2	62	M	10-mo history of dizziness and imbalance; rotatory vertigo with exertion as when playing tennis	Spontaneous nystagmus, rightward; horizontal head shaking nystagmus, rightward; vertical head shaking nystagmus, leftward	Left, up, counter-clockwise	Enhancing mass right IAC with 8-mm extension into CPA
3	62	M	Dysequilibrium and progressive gait ataxia	Left head tilt; diminished VOR noted with head thrusts to left; spontaneous nystagmus: rightward, clockwise slow phases on left and down gaze	Right, up, clockwise	Enhancing mass left IAC with 5-mm extension into CPA
4	64	M	Unsteadiness and dysequilibrium increasing in severity over 2½-y and worse on exertion	Horizontal head shaking nystagmus, leftward	Right, up, clockwise	Enhancing mass left IAC with 3-mm extension into CPA
5	52	F	Left tinnitus and sensorineural hearing loss	Normal	Right, up, clockwise	Enhancing mass left IAC with 2-mm extension into CPA
6	49	F	Episodic vertigo	Normal	Right, down, counterclockwise	Enhancing mass confined to right IAC

The direction of the nystagmus refers to the slow-phase components.

Hyperventilation-induced nystagmus in patients with vestibular schwannoma

Lloyd B. Minor, MD; Thomas Haslwanter, PhD; Dominik Straumann, MD; and David S. Zee, MD

The direction and alignment of nystagmus noted after hyperventilation can be used to evaluate the hypothesis that these eye movements result from a specific effect, either excitation or inhibition, on vestibular-nerve afferents arising from individual semicircular canals. The vestibular nerve has superior and inferior divisions. Vestibular schwannomas can arise from either the superior division of the vestibular nerve (through which pass fibers that innervate the ampullae of the horizontal and superior semicircular canals as well as the utriculus) or inferior division (through which pass fibers that innervate the ampullae of the posterior semicircular canal and the sacculus). The planar characteristics of the nystagmus elicited by hyperventilation would be expected to correlate with the location of the tumor.

In our patients, the determination of the contributions of individual canals based upon the alternate assumption—stimulation of a single canal leads to eye movements *exactly* in the plane of that canal—did not change the dominance of the contribution of the horizontal canal. The contribution from the vertical canals was, however, more variable when such an assumption was made. Because the underlying pathology in all our patients was similar (a schwannoma that predominantly affected the superior division of the vestibular nerve), the profile of canal activation produced by hyperventilation would also be expected to be similar among patients.

Mechanism of hyperventilation-induced nystagmus. We propose that partial demyelination of the vestibular nerve caused by the schwannoma is the underlying pathology in these cases of hyperventilation-induced nystagmus. The presumed mechanism involves the effects of hyperventilation on the physiology of partially demyelinated axons.

Hyperventilation-induced nystagmus in peripheral vestibulopathy and cerebellopontine angle tumor

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J.-W. Koo, MD
J.H. Kim, MD
C.-Y. Kim, MD
C.W. Oh, MD
H.J. Kee

Methods: We recorded horizontal HIN in 33 patients with CPA tumors and 145 with UPV. The UPV included patients of either acute (7 days or less from symptom onset, n = 47) or chronic (more than 7 days from symptom onset, n = 98) phases.

Results: The incidence of HIN was higher in the CPA tumor than in the UPV group (82 vs 34%, $p < 0.01$) and was also higher in the acute than in the chronic UPV group (60 vs 21%, $p < 0.01$). Furthermore, HIN was more commonly ipsilesional (i-HIN) in the CPA tumor than in the UPV group (52 vs 8%, $p < 0.01$) and more commonly ipsilesional in the acute than in the chronic UPV group (21 vs 1%, $p < 0.01$). The patients with i-HIN and acoustic neuroma had a tendency to harbor smaller tumors and to have less severe caloric asymmetry.

Conclusions: The contribution of hyperventilation on vestibular nystagmus differs depending on the disease phase or underlying pathologies. Our study demonstrates that hyperventilation-induced nystagmus (HIN) beating to the side of reduced caloric response, hearing impairment, or abnormal auditory brainstem response responses may be a valuable sign for bedside detection of cerebellopontine angle (CPA) tumors. CPA tumor should be a prime suspicion in patients with acute vertigo and ipsilesional HIN, especially when the vertigo accompanies hearing impairments.

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C.W. Oh, MD
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HIN in CPA tumors. HIN was observed in 27 (82%) of the 33 patients with CPA tumors, 19 (83%) of the 23 patients with acoustic neuroma, and 8 (80%) of the 10 with other CPA tumors (table 2). The fast phase of HIN beat toward the tumor side (i.e., ipsilesional) in 17 (52%) of the 33 patients, 11 with acoustic neuroma (48%) and 6 with other CPA tumors (60%) (figure 1A), whereas it beat toward the healthy side (i.e., contralesional) in 10 (30%) (figure 1B). The mean

Patients with acoustic neuroma and c-HIN had a tendency to harbor larger tumors and to have more severe caloric asymmetry. Seven of 8 patients with c-HIN had tumors larger than 1.0 cm, whereas 7 of 11 with i-HIN showed tumors less than 1.0 cm.

HIN in UPV. In 28 (60%) of 47 patients with acute UPV, hyperventilation induced nystagmus or affected the horizontal component of the spontaneous nystagmus (table 3). HIN was ipsilesional in 10 patients (21%) (figure 2A and video [on the *Neurology* Web site at www.neurology.org]) and contralesional in 18 (38%) (figure 2B). In the remaining 19 patients, hyperventilation neither induced the nystagmus nor affected the spontaneous nystagmus.

In contrast, hyperventilation induced nystagmus or affected the spontaneous nystagmus in only 21 (21%) of 98 patients with chronic UPV. The HIN was contralesional in 20 (20%) (figure 2C) and ipsilesional in only 1 patient (1%). In the chronic UPV group, the incidences of HIN and i-HIN were lower than those in acute UPV group (21 vs 60%, $p < 0.01$ and 1 vs 21%, $p < 0.01$).

Hyperventilation-induced nystagmus in peripheral vestibulopathy and cerebellopontine angle tumor

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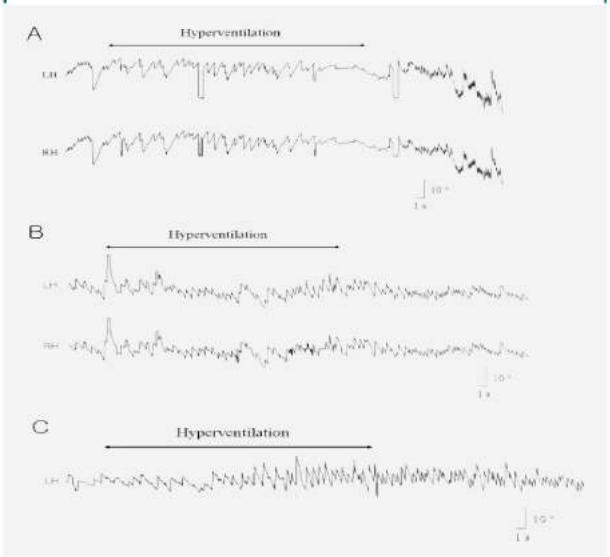
Table 3 HIN in patients with CPA tumors, acute and chronic UPV

	CPA tumor (n = 33)	Acute UPV (n = 47)	Chronic UPV (n = 98)
HIN			
Incidence, %	82	60	21
Mean velocity, °/s	6.6 ± 8.0	18.9 ± 19.2	5.7 ± 2.0
i-HIN			
Incidence, %	52	21	1
Mean velocity, °/s	6.9 ± 3.7	32.3 ± 25.7	6

HIN = hyperventilation-induced nystagmus; CPA = cerebellopontine angle; UPV = unilateral peripheral vestibulopathy; i-HIN = ipsilesional HIN.

Figure 2 Hyperventilation-induced nystagmus in patients with unilateral peripheral vestibulopathy



(A) After hyperventilation, contralesionally beating spontaneous nystagmus reverses its direction in a patient with acute right vestibular neuritis. (B) Hyperventilation augments spontaneous right beating nystagmus in a patient with acute left vestibular neuritis. (C) In a patient with chronic left peripheral vestibulopathy, hyperventilation markedly enhances contralesionally beating spontaneous nystagmus. LH = horizontal position of the left eye; RH = horizontal position of the right eye.

Inversione del nistagmo con Iperventilazione in un DVA destro

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Hyperventilation-Induced Nystagmus in Patients with Vestibular Neuritis in the Acute and Follow-Up Stages

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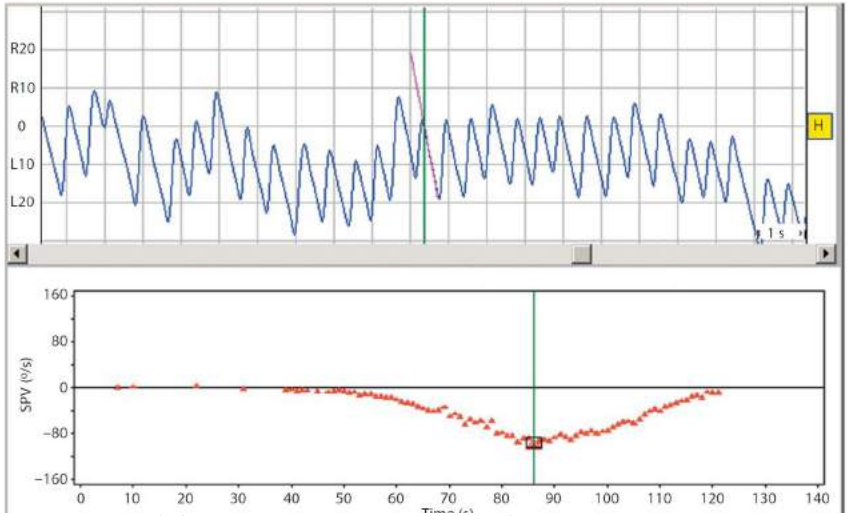
Bo Ra Na[a]

In the acute stage, 31 of 35 patients had spontaneous nystagmus (SPV of 6 ± 6°/s, 0–29°/s) in darkness. HVIN was observed in 18 (51%) of 35 patients with acute VN (table 1). HVIN beat toward the side of the lesion (ipsilesional HVIN) occurred in 8 (23%) of 35 patients. In 10 (28%) of 35 patients, HVIN beat away from the lesion (contralesional HVIN).

In the follow-up stage, 4 of 19 patients (21%) had normalized caloric responses and 13 patients had spontaneous nystagmus (SPV of 2 ± 2°/s; range = 0–8) in darkness, which was significantly lower than in the acute stage ($p < 0.001$). HVIN was observed in 4 of 19 patients (21%), which had a significantly lower incidence compared to the incidence (51%) in the acute VN ($p = 0.04$, Fisher's exact test; table 1).



Inversione del nistagmo con Iperventilazione in un DVA sinistro

Stages	HVIN (+)	HVIN (–)	Total
Acute	18 (51) ipsilesional HVIN = 8 (23) contralesional HVIN = 10 (28)	17 (49)	35 (100)
Follow-up	4 (21) ipsilesional HVIN = 0 (0) contralesional HVIN = 4 (21)	15 (79)	19 (100)

Whether a patient develops ipsilesional- or contralesional-beating HVIN will depend on the amount of vestibular nerve conductance block reversal, which will cause ipsilesional-beating HVIN, relative to the loss of central compensation, which will cause contralesional-beating HVIN [Bance et al., 1998]. These mechanisms account for our findings. In this study, nystagmus in the acute stage beat toward the ipsilesional (23%) or contralesional side (28%), whereas in the follow-up stage, nystagmus always beat toward the contralesional side (21%). Demyelination can cause ipsilesional HVIN in the acute stage, but not in the follow-up stage, which suggests that the demyelination recovered in the follow-up stage and only contralesional HVIN can occur in this stage. Our results also showed that HVIN is more common in the acute stage (51%) than the follow-up stage (21%), which is similar to a previous report [Choi et al., 2007]. Contralesional HVIN is caused by the relatively large loss of central compensation compared to the amount of vestibular nerve conductance block reversal by hyperventilation. These contralesional HVINs were observed in some patients in the acute stage and all of the patients in the follow-up stage.

Vibration- and Hyperventilation-Induced Nystagmus in Patients with Ramsay Hunt Syndrome with Vertigo

Chang-Hee Kim, MD, PhD[1], Kyung-Hwa Jeong, MD[1], Sung Hwan Ahn, MD[1], Dong Hyuk Shin, MD[1], Yong Won Kim, MD[1], and Jung Eun Shin, MD, PhD[1]

Objectives. The aims of this study were to compare vibration-induced nystagmus (VIN) and hyperventilation-induced nystagmus (HVIN) findings in patients with Ramsay Hunt syndrome with vertigo (RHS-V), sudden sensorineural hearing loss with vertigo (SSNHL-V), and vestibular neuritis (VN) during the acute stage and to address the possible lesion sites of vestibular deficit in RHS-V.

Results. An abnormal VIN was observed in 91% of the patients with VN, 89% of those with SSNHL-V, and 94% of those with RHS-V, and the prevalence of abnormal VIN was not significantly different (P = .436). An abnormal HVIN was observed in 51% of the patients with VN, 22% of those with SSNHL-V, and 59% of those with RHS-V. While the prevalence of an abnormal HVIN was significantly different between SSNHL-V and VN groups (P = .007) and between SSNHL-V and RHS-V groups (P = .014), that between VN and RHS-V groups did not show a significant difference (P = .547).

Conclusion. Since the results of HVIN in RHS-V patients were more similar to those in VN patients than those in SSNHL-V patients, a lesioned site may be more likely within the vestibular nerve than the inner ear as a cause for vestibular deficit in patients with RHS-V who show caloric canal paresis of 25% or greater.

Vestibular assessment in patients with vestibular schwannomas: what really matters?

Valutazione della funzione vestibolare nei pazienti affetti da Schwannomi vestibolari: cosa conta realmente?

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Table III. Cochlear and vestibular data.

	Vertigo	Imbalance	Mean PTA in decibel	Class of Hearing function	Class of vestibular function	UW%	Hyperveni
Koos 1 (n = 11)	5 (45%)	5 (45%)	49 ± 27	A = 4 B = 1 C = 4 D = 2	A = 1 B = 3 C = 6 D = 1	52 ± 21	9 (82%)
Koos 2 (n = 8)	3 (37%)	4 (50%)	25 ± 13	A = 5 B = 2 C = 1	A = 4 B = 4	29 ± 14	4 (50%)
Koos 3A (n =15)	4 (27%)	3 (20%)	60 ± 30	A = 3 B = 6 C = 3 D = 3	A = 2 B = 5 C = 4 D = 4	46 ± 27	11 (73%)
Koos 3B (n = 20)	6 (30%)	9 (45%)	57 ± 35	A = 4 B = 6 C = 6 D = 4	A = 2 B = 9 C = 5 D = 4	49 ± 25	16 (80%)
Koos 4A (n = 10)	1 (10%)	5 (50%)	55 ± 34	A = 2 B = 3 C = 3 D = 2	A = 1 B = 6 C = 1 D = 2	47 ± 30	7 (70%)

PTA = Pure tone average, UW = unilateral weakness.

Tinnitus, Oscillopsia, and Hyperventilation-Induced Nystagmus: Vestibular Paroxysmia

Bryan K Ward, MD[1,*] and **Daniel R Gold, DO**[1,2,3,4]

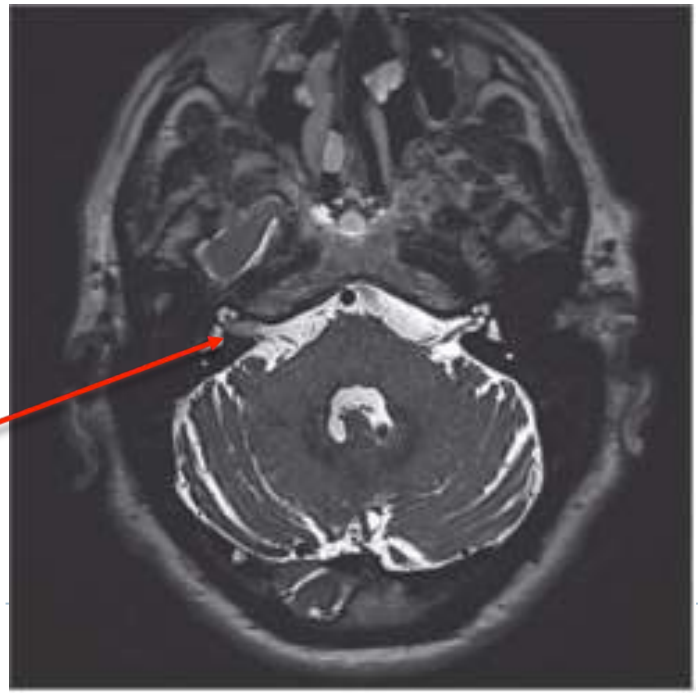
The above case is an example of vestibular paroxysmia. Vestibular paroxysmia is currently a diagnosis of exclusion, but common features include: brief attacks of vertigo or oscillopsia *(i.e.* the false sense that the visual surround is oscillating [5], presumably due to spontaneous nystagmus from transient vestibulocochlear nerve irritation) lasting seconds to minutes, with associated tinnitus, hearing changes or gait disturbance during attacks, measurable auditory or vestibular deficits, and efficacy of anti-epileptics [6,7]. Many patients develop nystagmus with hyperventilation [7], thought to be due to transient changes in conductivity across the demyelinated portion of the nerve during hyperventilation, causing excitatory or inhibitory patterns of nystagmus. Consequently, the symptoms of vestibular paroxysmia can be exercise-induced. Head movements or different head positions can trigger symptoms in some patients, presumably by increased neurovascular contact during certain positions. Finally, these patients may have prolonged wave latency on ABR [8], as was identified in this case, perhaps due to demyelination.

OBSERVATION

Exercise-Induced Vertigo in Vestibular Schwannoma

Emma Foster, MBBS
Luke Chen, MBBS, PhD

Exercise or hyperventilation induced nystagmus (HIN) may be the first sign of vestibular nerve dysfunction, and differentials beyond SCD need to be considered. These include tumor, multiple sclerosis, and cerebellar dysfunction.[1] In this case, the pattern of HIN indicates horizontal canal or superior vestibular nerve involvement



Neurinoma intracanalare a destra

ACTA OTORHINOLARYNGOLOGICA ITALICA 2011;31:17-26

Hyperventilation-induced nystagmus in a large series of vestibular patients

Nistagmo evocato dall'iperventilazione in un'ampia popolazione di pazienti vestibolari

L. CALIFANO, M.G. MELILLO, A. VASSALLO, S. MAZZONE

Otology & Neurotology
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Hyperventilation-Induced Nystagmus in Patients With Vestibular Schwannoma

Luigi Califano, Giuseppina Iorio, Francesca Salafia, Salvatore Mazzone, and Maria Califano

ACTA OTORHINOLARYNGOLOGICA ITALICA 2017;37:336-340

Sensitivity and specificity of vestibular bed-side examination in detecting VIII cranial nerve schwannoma with sensorineural sudden unilateral hearing loss as presenting symptom

Sensibilità e specificità della vestibular bed-side examination nell'individuare lo schwannoma dell'VIII nervo cranico con ipoacusia improvvisa come sintomo di esordio

L. CALIFANO, F. SALAFIA, M.G. MELILLO, S. MAZZONE

Utility of the hyperventilation test in the evaluation of the dizzy patient

Luigi Califano, Salvatore Mazzone, and Francesca Salafia

IL NISTAGMO DA IPERVENTILAZIONE
L. Califano, S. Mazzone, F. Salafia

LA DIAGNOSI BEDSIDE DELLA VERTIGINE ACUTA
Bedside examination of acute vertigo

Il nistagmo evocato da iperventilazione

Luigi Califano

Vestibologia Clinica

ACTA OTORHINOLARYNGOLOGICA ITALICA 2011;31:17-26

Hyperventilation-induced nystagmus in a large series of vestibular patients

Nistagmo evocato dall'iperventilazione in un'ampia popolazione di pazienti vestibolari

L. CALIFANO, M.G. MELILLO, A. VASSALLO, S. MAZZONE

1202 patients (683 female, 519 male) were included in the study; mean age 54.3 years (range 10-88 years).

HVT was performed in a sitting position through quick and deep respiratory cycles for 70 seconds: this duration might be enough to cause the metabolic and neurophysiological effects of the hyperventilation. HVIN was present if its slow-phase velocity (SPV) was of at least 5°/sec. within one minute after the end of hyperventilation, with a duration of at least 5 seconds.

If spontaneous nystagmus was already present, HVIN was present if it increased or decreased SPV of the spontaneous nystagmus of at least 5°/sec., for at least 5 seconds.

Normative data were obtained following an analysis of the oculo-motor responses evoked by HVT in the Control group (outlined below).

Table I. HVIN in vestibular disorders sorted by incidence.

Diagnosis	HVIN+
Pre-surgery acoustic neuroma	11/12 (91.7%)
Post-surgery acoustic neuroma	7/9 (77.8%)
Multiple sclerosis	9/12 (75%)
Cerebellar diseases	8 /11 (72.7%)
Acute vestibular neuritis	39/54 (72.2%)
Neurovascular compression	3/5 (60%)
Labyrinthine Fistulas and Superior canal dehiscence Syndrome	11/20 (55%)
Unilateral Menière's disease	35/93 (37.6%)
Compensated vestibular neuritis	33/89 (37.1%)
Bilateral vestibular loss of function	1/5 (20%)
Migraine-related vertigo	36/188 (19.1%)
Vascular vertigo	27/152 (17.8%)
BPPV	24/455 (5.3%)
Chronic subjective dizziness	0/23
Undiagnosed vertigo	19/74 (25.7%)
Overall	263/1202 (21.9%)

Table V. Prevalence of HVIN in each vestibular disorder and logistic regression analysis evaluating the HVIN odds ratio in each of them compared to healthy controls and adjusted for age and sex.

Vestibular disorders	N	HVIN	%	β	SE	p	Adjusted OR (95%CI)
BPPV	455	24	5.3	0.147	0.077	0.496	2.729 (0.361-20.612)
Menière's disease	93	35	37.6	0.378	0.107	< 0.001	29.569 (3.908-223.753)
Acute vestibular neuritis	54	39	72.2	0.708	0.117	< 0.001	127.400 (16.117-1007.076)
Compensated vestibular neuritis	89	33	37.1	0.357	0.169	< 0.001	28.875 (3.807-218.989)
Labyrinthine fistulas	20	11	55	0.586	0.117	< 0.001	59.889 (6.858-522.955)
Pre-surgery acoustic neuroma	12	11	91.7	0.754	0.085	< 0.001	539.001 (31.244-9298.573)
Post-surgery acoustic neuroma	9	7	77.8	0.363	0.185	< 0.001	171.501 (13.693-2148.022)
Bilateral vestibular areflexia	5	1	20	0.207	0.107	0.175	12.250 (0.639-234.810)
Neurovascular compression	5	3	60	0.278	0.087	0.001	73.500 (5.098-1059.783)
Migraine-related vertigo	188	36	19.1	0.301	0.148	0.002	11.605 (1.550-86.867)
Multiple sclerosis	12	9	75	0.423	0.109	< 0.001	147.000 (13.712-1575.926)
Cerebellar diseases	11	8	72.7	0.486	0.111	< 0.001	130.667 (12.052-1416.28)
Vascular vertigo	152	27	17.8	0.358	0.107	0.004	10.584 (1.400-80.032)
Chronic subjective dizziness	23	0	0	-0.275	0.148	0.827	0.980 (0.942-1.020)
Undiagnosed vertigo	74	19	25.7	0.421	0.205	< 0.001	16.927 (2.185-131.149)

SE: standard error; OR: Odds Ratio; 95% CI: 95% confidence intervals; a p value < 0.003 was considered significant according to Bonferroni correction.

Table VII. Prevalence of p-HVIN and e-HVIN in vestibular neuritis and acoustic neuroma.

Vestibular disorders	p-HVIN	%	e-HVIN	%
Acute vestibular neuritis	32	82.05	7	17.95
Compensated vestibular neuritis	32	96.96	1	3.04
Pre-surgery acoustic neuroma	4	36.37	7	63.63
Post-surgery acoustic neuroma	7	100	0	0

p = 0.006; adjusted Odds Ratio 8.000; 95% confidence intervals (1.829-34.996).

Conclusions

HVT is a simple test.
Could it be useful in the bedside examination of a vestibular patient? We think so.
However, demonstrating how hyperventilation affects the vestibular system is a difficult task since several mechanisms can be hypothesized, which act either at a central level (cerebellum or other control sites for the compensation of a vestibular asymmetry) or on the vestibular nerve (transient improvement of the neural conduction), or on the labyrinthine periphery, acting by direct stimulation of hair-cells and/or by increasing neural excitability determined by hyperventilation-induced metabolic modifications.

Nonetheless, HVT is "the only test that unmasks unilateral vestibular disease without testing the dynamic properties of the vestibule-ocular reflex" [5].

HVT can provide patterns of oculo-motor responses that justify further evaluations through Gadolinium-enhanced MRI, in the search of an acoustic neuroma or of central neurological diseases: this is the case of the e-HVIN detected in unilateral acute or compensated vestibular loss of function (neuritis or neuroma) with an opposite oculomotor response to HST and caloric tests as well as the case of central-type HVIN (upbeat or downbeat nystagmus; rotatory nystagmus) and the case of inhibition of a central-type nystagmus, as in Multiple sclerosis.

HVT has proved to be accepted with excellent tolerability and to offer good clinical validity, although it is advisable to consider results of the investigation within the wider context of a general evaluation of auditory and vestibular functions.

Test di Iperventilazione

- Utilizzata 60 sec di Iperventilazione profonda con paziente seduto
- Osservazione in Videonistagmoscopia e Videonistagmografia
  - Registrazione ed ottimizzazione video

PATOLOGIA PERIFERICA E NEURALE

Il nistagmo da iperventilazione nel Deficit vestibolare acuto monolaterale

Il nistagmo da iperventilazione nel Neurinoma dell' VIII n.c.

Terminologia

- Nistagmo evocato da iperventilazione (HVIN) "ipsilaterale eccitatorio"
  - Fasi rapide verso il lato leso
    - Nistagmo ex-novo
    - Riduzione/inibizione del nistagmo spontaneo ("ipsilaterale eccitatorio")
    - Inversione del nistagmo spontaneo ("ipsilaterale fortemente eccitatorio")

Terminologia

- Nistagmo evocato da iperventilazione (HVIN) "controlaterale inibitorio"
  - Fasi rapide verso il lato sano
    - Nistagmo ex-novo
    - Rinforzo del nistagmo spontaneo

Terminologia

La variazione di intensità della nistagmo spontaneo deve essere di almeno 3°/sec di SPV per almeno 5” se si usa una registrazione videonistagmografica

Vestibulopatia deficitaria acuta monolaterale

- **Elaborazione su 300 casi**
  - **Osservazione tra 3 ore e cinque giorni dall' esordio dei sintomi**

HVIN orizzontale:242/300 casi (80.7%)

Vestibulopatia deficitaria acuta monolaterale

I patterns del nistagmo da iperventilazione nel DVA monolaterale

Vestibulopatia deficitaria acuta monolaterale

HVIN -	58/300 (19.3%)
HVIN inibit	149/300 (49.7%)
HVIN ecc.	40/300 (13.3%)
HVIN fort. eccitatorio	53/300 (17.7%)
HVIN +	242/300 (80.7%)

Vestibulopatia deficitaria acuta monolaterale

- I patterns "eccitatorio" e "fortemente eccitatorio" sono propri delle fasi molto iniziali della patologia
- Nell'evoluzione: rapida reversione del Nistagmo eccitatorio
  - Dati personali: in 7-10 giorni
  - Choi: in 1-8 mesi
  - Fisiopatologia:
    - da demielinizzazione (fase eccitatoria) a degenerazione assonale (fase inibitoria) (Kuwabara, 2006); minore pressione sul nervo determinata dalla diminuzione di pressione liquorale

Vestibulopatia deficitaria acuta monolaterale

- **Buona sensibilità del test (*inferiore all'HST ed al VT*)**
- **Test utile ed immediato per evidenziare eventuali segni da demielinizzazione *(nistagmo eccitatorio),* il cui riscontro *obbligherebbe* alla diagnostica per immagini (Minor) (?)**
- **Possibili falsi (??) positivi: i casi con pattern eccitatorio/fortemente eccitatorio da microdemielinizzazione per l'edema (Matsuo, Califano) non sono indicativi del lato leso**

Vestibulopatia deficitaria acuta monolaterale

La presenza dei pattern inibitorio rispetto ai pattern eccitatori sembra ben correlare con l'età dei pazienti e con il grado di rischio vascolare

- I pattern eccitatori sono più frequenti nella popolazione più giovane e/o con rischio vascolare basso
- Il pattern inibitorio è più frequente nella popolazione più anziana e/o con rischio vascolare alto

I pattern della iperventilazione possono essere indicatori della possibile etiologia infiammatoria vs. vascolare della vestibulopatia deficitaria acuta monolaterale?

L. Califano et al.: IN PRESS

Vertigine acuta

Protocollo HINT
Plus
Ice test simultaneo
(In PS: STANDING)

Vertigine periferica

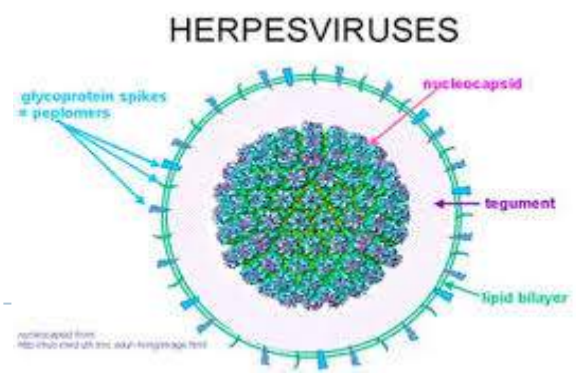
Vertigine centrale

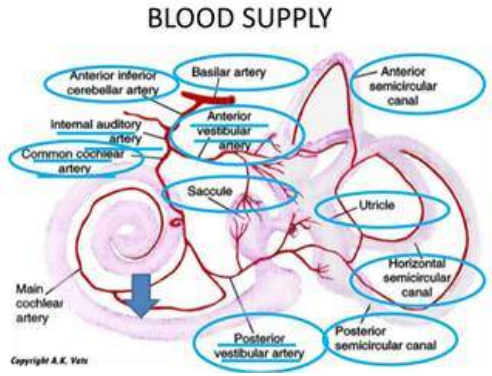
Vertigine acuta

Vertigine periferica tipo
Vestibulopatia acuta
deficitaria monolaterale

Neurite vestibolare

Ischemia labirintica





Non esistono dati semeiologici conclamati per porre diagnosi differenziale che è sostanzialmente di tipo anamnestico-probabilistico

Età

Modalità di insorgenza

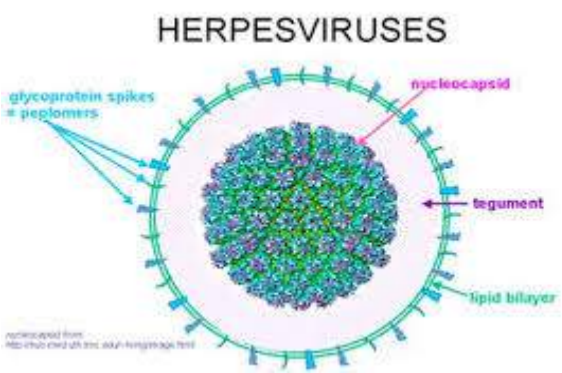
Associazione ad ipoacusia acuta monolaterale

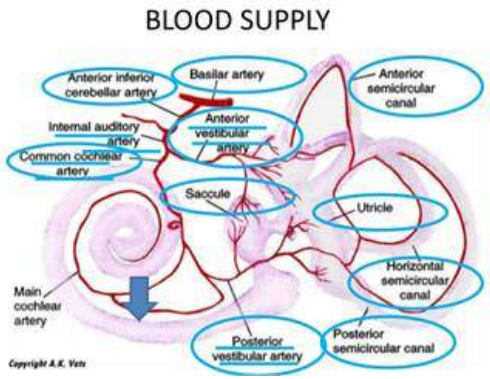
Presenza o meno di fattori di rischio vascolari

Flogosi recente a carico delle vie aeree….

Il test di iperventilazione fornisce dati in fase acuta di deficit vestibolare monolaterale?

Può il test di iperventilazione contribuire alla diagnosi differenziale tra forme nevritiche e forme vascolari?





I pattern del nistagmo da iperventilazione in corso di vestibulopatia deficitaria acuta monolaterale

HVIN negativo
- Assenza di modifiche del nistagmo spontaneo

HVIN inibitorio
- Rinforzo del nistagmo spontaneo

HVIN eccitatorio
- Inibizione del nistagmo spontaneo (fino alla sua scomparsa)

HVIN fortemente eccitatorio
- Inversione del nistagmo spontaneo

Table III. HVIN in Acute Vestibular Neuritis.

HVIN	15/54 (27.8%)
Paretic HVIN	32/54 (59.2%)
Excitatory HVIN	3/54 (5.6%)
Strongly excitatory HVIN	4/54 (7.4%)

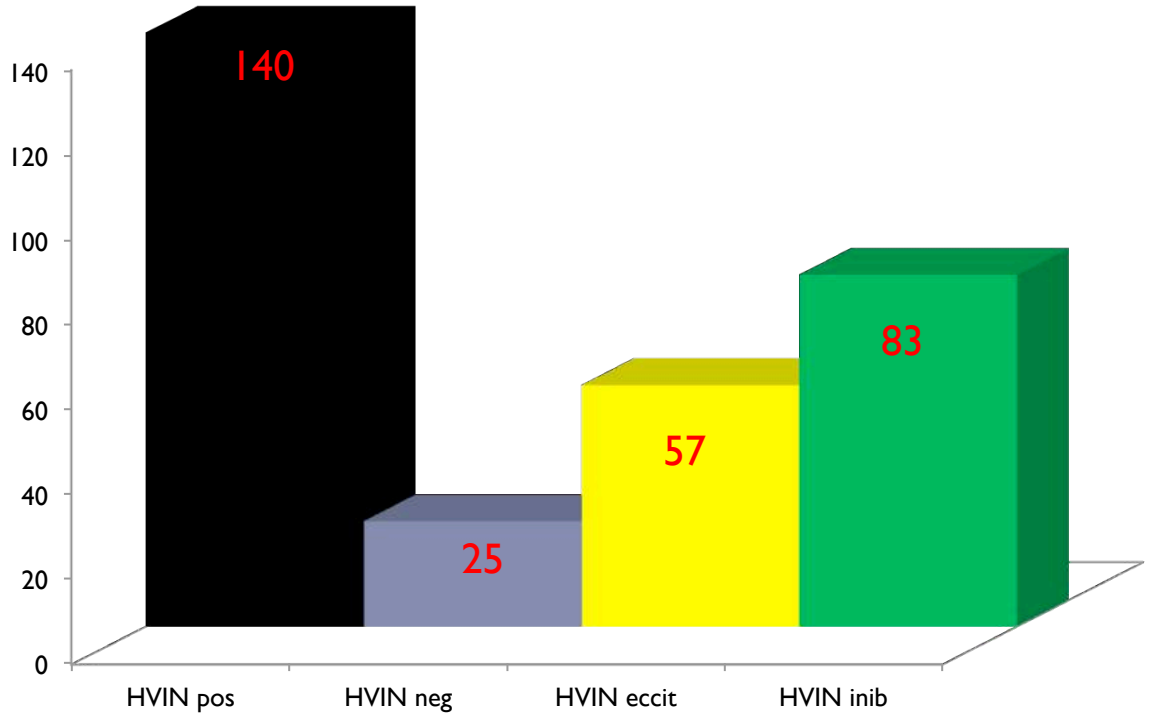
I patterns del nistagmo da iperventilazione nel DVA monolaterale

I patterns del nistagmo da iperventilazione nel DVA monolaterale

Ai fini delle valutazioni seguenti, è stata misurato con capnografia "side stream" il valore di P_{CO2} almeno fino al raggiungimento del livello da noi precedentemente dimostrato essere efficace di evocare o modificare il nistagmo, sia in modo eccitatorio che inibitorio, in caso di varie patologie dell'apparato vestibolare, e cioè 16 mm Hg.

Ciò al fine di non imputare le differenze, soprattutto di età, ad una scorretta esecuzione del test

HVIN pos	140/165 (84.8%)
HVIN neg	25/165 (15.2%)



HVIN ecc	57/140 (41.7%)
HVIN inib	83/140 (59.3%)

Esiste una correlazione tra presenza/ assenza di nistagmo evocato da iperventilazione e tra evocabilità dei patterns inibitorio o eccitatorio ed età e rischio cardiovascolare?

Il rischio cardiovascolare globale è stato calcolato usando il "Calcolatore del Rischio cardiovascolare globale" del progetto CUORE dell'Istituto Superiore di Sanità

Sesso
Età
Ipertensione
Colesterolemia
HDL Colesterolemia
Diabete mellito
Abitudine al fumo
Anamnesi di eventi vascolari maggiori

Classe rischio		
VI	> 30%	Molto alto
V	20-29%	Alto
IV	15-19%	Moderato
III	11-14%	Medio-basso
II	5-10%	Basso
I	<5%	Molto basso

Popolazione complessiva

	Età media
HVIN pos	58.89÷
HVIN neg	7(

	Età media
	46.57 ±11.76
	1÷12.35

Età media Classi I-II-III
56.34 ÷13.01 anni

Età media Classi IV-V-VI
72.45 ÷11.84 anni

	Basso rischio (Classi I-III)	
HVIN pos	95/104 (91.3%)	45/61 (
HVIN neg	9/104 (8.7%)	16/61 (26.2%)

	I-III)	Alto rischio (Classi IV-VI)
	48/95 (50.5%)	9/45 (20%)
HVIN inib	47/95 (49.5%)	36/45 (80%)

Senectus Ipsa est Morbus

P. Terenzio Afro, *Phormio, Atto IV, Scena I*

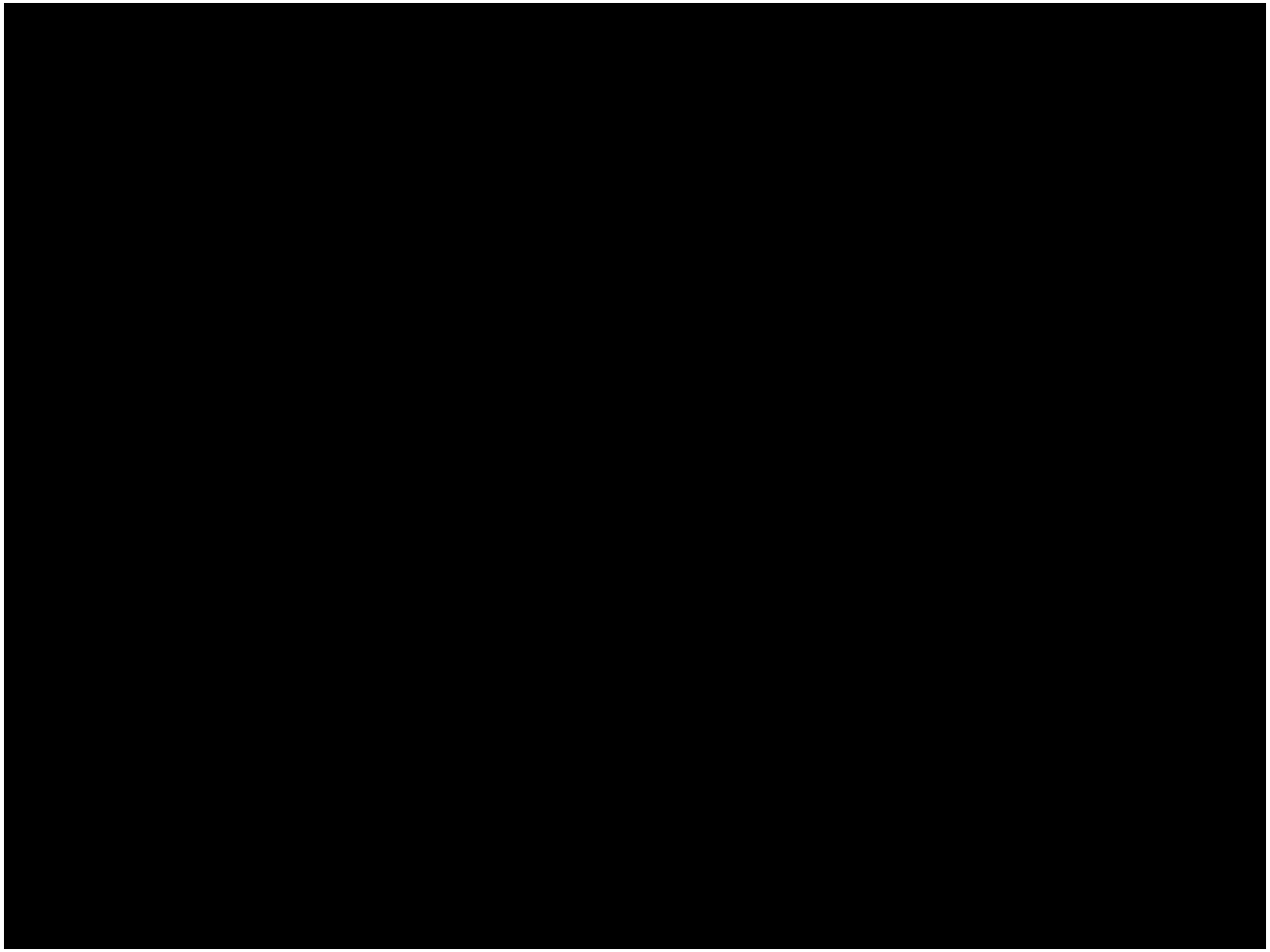
L’ HVIN è evocato più frequentemente nei soggetti più giovani ed in quelli con rischio vascolare minore

Il pattern eccitatorio è più frequente nei soggetti più giovani e con rischio vascolare minore

Il pattern inibitorio è più frequente in quelli più anziani e con rischio vascolare maggiore

La variabile "durata del nistagmo"

140 Pazienti con nistagmo spontaneo a durata maggiore di 72 h	25 Pazienti con nistagmo spontaneo a durata minore di 72 h
Età media (range 8-78 aa)	**Età media (range 65-89 aa)**
55.62÷14.65	75.56÷8.71
Età > 65 anni	25/25> 60 anni
47/140 (33.6%)	



DVA monolaterale a rapida evoluzione con assenza di nistagmo da iperventilazione

La variabile "durata del nistagmo"

140 Pazienti con nistagmo spontaneo a durata maggiore di 72 h

HVIN pos	126/140 (90%)
HVIN neg	14/140 (10%)

HVIN ecc	57/140 (40.7%)
HVIN inib	69/140 (49.3%)

25 Pazienti con nistagmo spontaneo a durata minore di 72 h

HVIN pos	14/25 (56%)
HVIN neg	11/25 (44%)

HVIN ecc	0
HVIN inib	14/25 (56%)

La variabile "durata del nistagmo"

140 Pazienti con nistagmo spontaneo a durata maggiore di 72 h

	Basso rischio (Classi I-III)	Alto rischio (Classi IV-VI)
HVIN pos	95/140 (67.9%)	31/140 (22.1%)
HVIN neg	12/140 (8.7%)	2/140 (1.5%)

	Basso rischio (Classi I-III)	Alto rischio (Classi IV-VI)
HVIN ecc	48/140 (34,3%)	9/140 (6.4%)
HVIN inib	47/140 (33.6%)	22/140 (15.7%)

25 Pazienti con nistagmo spontaneo a durata minore di 72 h

	Basso rischio (Classi I-III)	Alto rischio (Classi IV-VI)
HVIN pos	0/25	14/25 (56%)
HVIN neg	2/25 (8%)	9/25 (36%)

	Basso rischio (Classi I-III)	Alto rischio (Classi IV-VI)
HVIN ecc	0	0
HVIN inib	0	14/25 /56%)

L’HVIN è evocato più frequentemente nei soggetti con durata del nistagmo >72 ore

Il pattern eccitatorio è stato riscontrato unicamente nel gruppo con nistagmo di durata > 72 ore

Il Nistagmo evocato da iperventilazione nella fase iniziale di un deficit vestibolare acuto appare più frequentemente riscontrabile nei soggetti più giovani, con rischio vascolare minore e con durata del nistagmo > 72 ore

La presenza del pattern eccitatorio esclusivamente nel gruppo con durata del nistagmo > 72 ore, di età minore e con minore rischio vascolare potrebbe suggerire in questi casi una origine infiammatoria "neuritica" del danno vestibolare

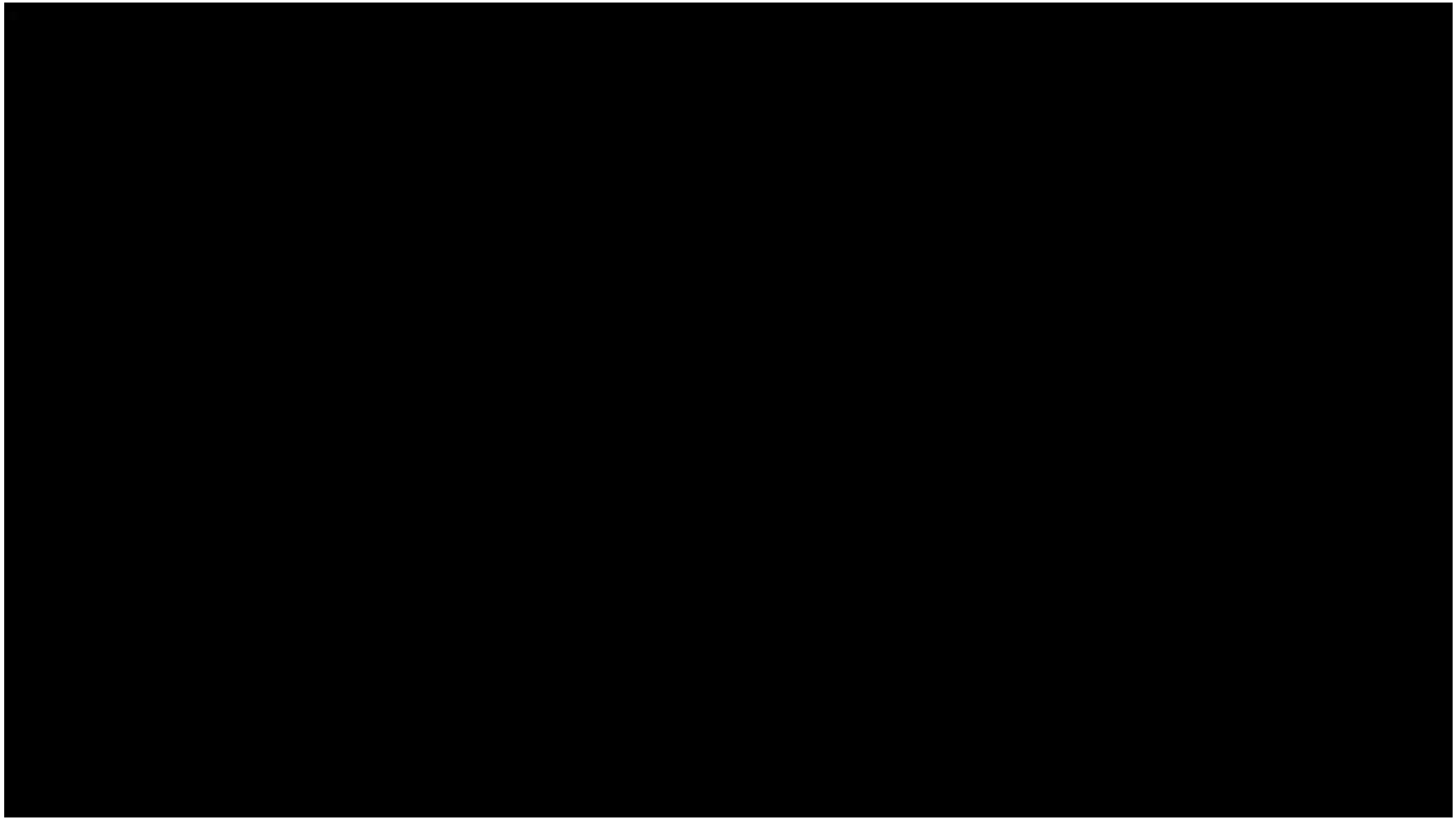
La presenza prevalente del pattern inibitorio nei pazienti più anziani, con maggiore rischio vascolare e nel gruppo con nistagmo < 72 ore potrebbe suggerire un' origine microvascolare labirintica acuta del danno vestibolare

«Terra di nessuno» è da considerare il gruppo con nistagmo >72 ore e pattern inibitorio, in cui la valutazione rimane probabilisticamente legata alla valutazione anamnestica e del rischio vascolare globale

Ma anche in forma acute centrali

Inibizione di nistagmo centrale con Iperventilazione

Prima poussée acuta di Sclerosi a placche
Meccanismo fisiopatologico di inibizione del nistagmo attraverso un miglioramento della conduzione lungo vie demielinizzate



Inibizione di nistagmo centrale con Iperventilazione

Prima poussée acuta di Sclerosi a placche
Meccanismo fisiopatologico di inibizione del nistagmo attraverso un miglioramento della conduzione lungo vie demielinizzate

Ragazzo di 16 anni, vertigine acuta «posizionale»

Prima poussée acuta di Sclerosi a placche
Slatentizzazione del nistagmo per verosimile azione metabolica centrale della Iperventilazione

The effect of hyperventilation on downbeat nystagmus in cerebellar disorders

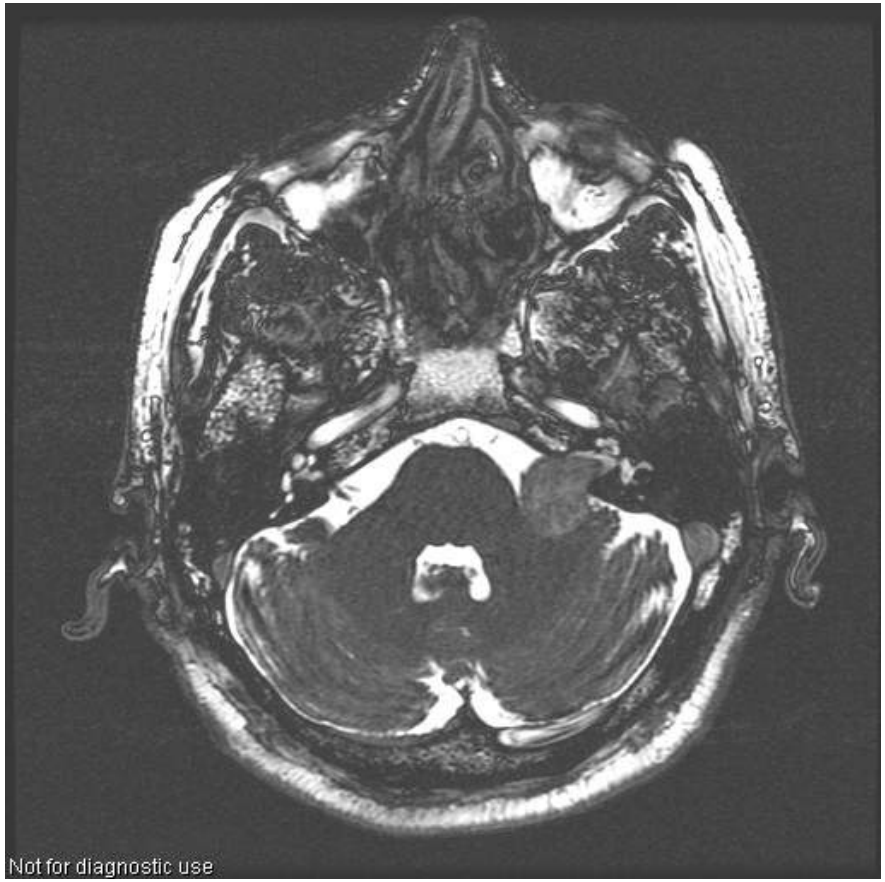
Article abstract—Hyperventilation can affect nystagmus in patients with vestibular disorders. However, the effects on nystagmus in patients with cerebellar disease have not been systematically studied. Using the magnetic field search coil technique, we studied the effects of hyperventilation on nystagmus in a series of cerebellar patients. In four of eight patients, hyperventilation produced an increase in the slow-phase velocity of downbeat nystagmus. We speculate that this effect may be mediated through metabolic effects on cerebellar calcium channels. **Key words:** Hyperventilation—Nystagmus—Downbeat nystagmus—Cerebellum.

NEUROLOGY 1999;53:1576–1579

Mark F. Walker, MD; and David S. Zee, MD

Downbeat nystagmus in cerebellar patients may be modified by a number of factors, including orbital position, head position and movement,[5] head shaking,[8] and caloric stimulation.[5] We have now shown that downbeat nystagmus may also be increased by hyperventilation.

Neurinoma dell' VIII



Neurinoma dell' VIII
I patterns dell' HVIN

TABLE 2. *Distribution of paretic and excitatory HVIN according to Sanna's classification of vestibular schwannomas*

	Grades 0–1 (≤10 mm)	Grade 2 (11–20 mm)	Grade 3 (21–30 mm)	Grade 4 (31–40 mm)	Grade 5 (>40 mm)	Overall
Paretic HVIN	5	6	3	1	1	16
Excitatory HVIN	4	14	2	3	1	24
	9	20	5	4	2	40

Differences are not significant ($p = 0.55$)

HVIN presente in 40/45 casi (88.8%)

e-HVIN 24 casi i-HVIN 16 casi HVIN- 5 casi

Neurinoma sinistro 3 cm: HVIN eccitatorio (o ipsilaterale)

Neurinoma sinistro 3 cm: HVIN inibitorio (o controlaterale)

Neurinoma dell' VIII
I patterns dell' HVIN

Non abbiamo evidenziato correlazione tra pattern dell' HVIN e la dimensione del nuerinoma, né usando le categorie di Sanna, né con cut-off a 10, 15, 20 mm

Neurinoma dell' VIII
I patterns dell' HVIN

Alcuni Autori (Bance,1998; Choi, 2007; Nuti,2012) segnalano la presenza di

- **Nistagmo eccitatorio nei tumori più piccoli**
  (Bance: media 10 mm; Choi <10 mm; Nuti < 20 mm)

- **Nistagmo inibitorio nei tumori più grandi**
  (Bance: media 15 mm; Choi > 10 mm; Nuti > 20mm)

Neurinoma dell' VIII
I patterns dell' HVIN

La nostra Casistica conferma alcuni dati di Letteratura *(Minor, Kwang-Dong), ma non altri (Bance, Choi, Nuti)*

La direzione (deficitaria- irritativa) del Nistagmo è funzione della grandezza del neurinoma, ma non in modo univoco

Neurinoma dell' VIII
I patterns dell' HVIN

Il Nistagmo eccitatorio *è spiegabile con la presenza di focolai di demielinizzazione sul nervo, ove la conduzione nervosa è fasicamente migliorata dalle alterazioni metaboliche ed acido-base indotte dalla iperventilazione con conseguente transitoria up-regulation dei meccanismi centrali del compenso*

Il Nistagmo eccitatorio scompare sempre dopo l'asportazione chirurgica del neurinoma (Minor, Choi, Nuti,Califano), sempre sostituito da Nistagmo inibitorio

Neurinoma dell' VIII
I patterns dell' HVIN

Il Nistagmo inibitorio dipenderebbe dal danno neuronale non migliorabile nella conduzione per

- Discontinuità funzionale delle fibre: tumori più grandi
- assenza di demielinizzazione: tumori più piccoli

con rottura dei meccanismi di compenso centrali indotta dalla HV

Iperventilazione e patologia vestibolare: neuriti e neurinomi

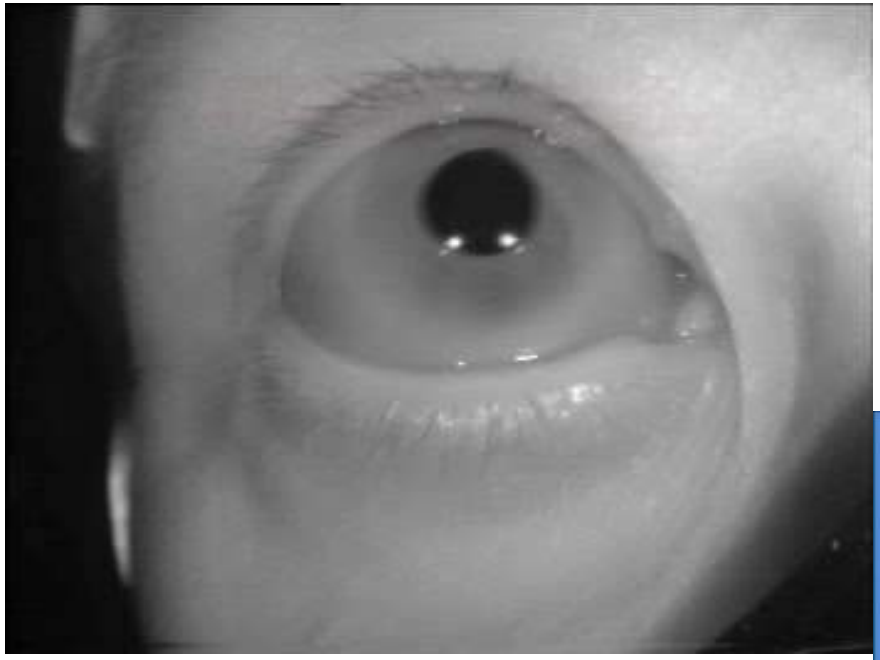
- **Il Nistagmo deficitario prevale nelle forme acute e negli esiti a distanza**
- **Il Nistagmo irritativo prevale nelle forme tumorali, relazionandosi in modo solo parziale alla grandezza del tumore**
- **Il Nistagmo irritativo non è specifico della patologia tumorale, così come il Ny deficitario non lo è della patologia deficitaria acuta**

Iperventilazione e patologia vestibolare: Nistagmo irritativo in neuriti e neurinomi

La vera questione che fa la specificità è la contestualizzazione clinica del segno

1. Nelle NVA il Nistagmo irritativo si sovrascrive, annullandolo o invertendolo, ad un Nistagmo spontaneo (senza escludere a priori che una vertigine da deficit acuta possa avere alla base un neurinoma)
2. Nei neurinomi l'HV crea «ex-novo» un Nistagmo irritativo
3. Nelle NVA mancano di solito segni di sofferenza uditiva monolaterale
4. Se presenti, sono anch'essi ad insorgenza acuta
5. Nelle NVA il Nistagmo irritativo è tempo-dipendente: scompare tra 3 e 10 giorni dall'insorgenza
6. Negli esiti di NVA è presente il dato anamnestico e spesso la documentazione (RM compresa) del pregresso episodio acuto

In appendice….



- Fistola csl, pressione ed iperventilazione

Il test di iperventilazione è l'unico test che può svelare malattie vestibolari unilaterali, anche basalmente compensate, senza testare le proprietà dinamiche del riflesso vestibulo-oculomotorio

Manohar L. Bance

KEY POINTS

- The hyperventilation test is easily performed in vestibular bedside examination.
- It is a good indicator of a latent vestibular asymmetry.
- It works through metabolic mechanisms, either in peripheral or in central vestibular diseases.
- It is one of the most efficient tests in the diagnosis of schwannoma of eighth cranial nerve.
- It can provide patterns of oculomotor response, which justify resorting to cerebral MRI.

CURRENT OPINION

Utility of the hyperventilation test in the evaluation of the dizzy patient

Luigi Califano, Salvatore Mazzone, and Francesca Salafia

Come tutti i test, va inserito nel contesto di un più complesso protocollo di valutazione del paziente vertiginoso e contestualizzato alla situazione clinica del paziente


CINEMA
MIGNON