

Conclusioni

- La vertigine in PS, seppur raramente, può essere l'espressione di una condizione di rischio grave ma potenzialmente trattabile
- Sindrome vestibolare acuta: neurite vestibolare vs stroke
- La valutazione clinica (o con un minimo supporto strumentale) è meglio della RM
- La TC encefalo solo molto raramente è utile

Diagnosis		Patients No (%)
Peripheral neurologic	↑↑ cardio (6 mo) and cerebro (30 days) risk (Kim SA et al 2011, Lee CC et al 2012).	294 (32)
1. Peripheral vertigo		185 (20)
2. BPPV		78 (9)
3. Vestibular neuritis		27 (3)
4. Meniere disease		4 (<1)
Serious neurologic disease		49 (5)
1. Ischemic stroke		24 (3)
2. TIA		8 (1)
3. Brain neoplasm		6 (1)
4. Intracerebral hemorrhage		5 (1)
5. Seizure		4 (<1)
6. Demyelinating disease		2 (<1)
Other neurologic disease		388 (43)
1. Dizziness NOS		199 (22)
2. Orthostasis/near syncope		121 (13)
3. Migraine		37 (4)
4. Syncope		20 (2)
5. Concussion		11 (1)
Psychiatric conditions		22 (2)
Serious cardiac disease		35 (4)
1. Arrhythmia		22 (2)
2. Hypertensive emergency		10 (1)
3. Acute coronary syndrome		2 (<1)
4. Heart failure		1 (<1)
Other medical condition		119 (13)
1. Drug/substance ingestion/withdrawal		46 (5)
2. Systemic infection		34 (4)
3. Electrolyte disorder		14 (2)
4. Anemia		10 (1)
5. Hypoglycemia		4 (<1)
6. Other		11 (1)

Dizziness without neurological signs or symptoms: 0.7% has a vascular cause

Target

- Acute vestibular syndrome
 - rapid onset (seconds to hours) of vertigo, nausea/vomiting, and gait unsteadiness in association with head motion intolerance, and nystagmus, lasting days to weeks

High Risks

- 25% isolated AVS have a vascular origin, and AVS is the condition that most frequently leads to a misdiagnosed stroke (Tehrani A. et al, 2014).
- Mortality is about 40% in ED dizzy patients with a misdiagnosed cerebellar stroke

Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome

Alexander A. Tarnutzer MD, Aaron L. Berkowitz MD PhD, Karen A. Robinson PhD, Yu-Hsiang Hsieh PhD, David E. Newman-Toker MD PhD

CMAJ 2011. DOI:10.1503/
cmaj.100174

KEY POINTS

- The most common causes of acute vestibular syndrome are vestibular neuritis (often called labyrinthitis) and ischemic stroke in the brainstem or cerebellum.
- Vertebrobasilar ischemic stroke may closely mimic peripheral vestibular disorders, with obvious focal neurologic signs absent in more than half of people presenting with acute vestibular syndrome due to stroke.
- Computed tomography has poor sensitivity in acute stroke, and diffusion-weighted magnetic resonance imaging (MRI) misses up to one in five strokes in the posterior fossa in the first 24–48 hours.
- Expert opinion suggests a combination of focused history and physical examination as the initial approach to evaluating whether acute vestibular syndrome is due to stroke.
- A three-component bedside oculomotor examination — HINTS (horizontal head impulse test, nystagmus and test of skew) — identifies stroke with high sensitivity and specificity in patients with acute vestibular syndrome and rules out stroke more effectively than early diffusion-weighted MRI.

Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION



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, Arun V. Talkad, David Z. Wang, Yu-Hsiang Hsieh and David E. Newman-Toker

Stroke. 2009;40:3504-3510; originally published online September 17, 2009;
doi: 10.1161/STROKEAHA.109.551234

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HINTS:	sensitivity 100%	specificity 96%
Early MRI with DWI:	sensitivity 72%	specificity 100%

HINTS to INFARCT: Impulse (test) Normal, Fast (phase) Alternating, Refixation on Cover Test

DIAGNOSIS

H(ead)I(mpulse)N(ystagmus)T(est for)S(kew)

- Nystagmus
 - Peripheral = Horizontal(-torsional), unidirectional, multipositional
 - **CNS** = horizontal, direction changing (bidirectional, pluripositional) → gaze evoked nystagmus (*+ vertical or torsional*)
- Head impulse test (**normal / untestable**)
- Cover test per skew deviation (**refixation movement / untestable**)

HINTS - NYSTAGMUS

The basics of vestibular system physiology

- The vestibular receptors have a tonic activity, i.e they are active even when the head is steady
- The CNS uses the signals from both sides to understand if the head is moving
- The tonic discharge is the same in the two sides
- In terms of vestibular signals, the head is still not when there is no activity but when the activity is the same in the two sides.

The basics of vestibular system physiology

- Leftward head rotation (acceleration) unbalances the activity of the two hemi-systems :
 - ↑ left (excitation)
 - ↓ right (inhibition)
- CNS
 - Unbalanced activity = rotation
 - Direction = excited side

Vestibulo-Ocular-Reflex

VOR

- VOR = gaze stabilization in space = the eyes move in the opposite direction with respect to head motion

Vestibular ocular motor connections

	Normal		Left Out	
	right	left	right	left
Lateral	→	←	→	
Anterior	↑ ∩ ▼	↑ ▼ ∩	↑ ∩ ▼	
Posterior	↓ ∩ ▼	↓ ▼ ∩	↓ ∩ ▼	
L + A + P	→ ∩ ▼	← ▼ ∩	→ ∩ ▼	
Right + Left	0		→ ∩ ▼	

Vestibular ocular motor connections

	Normal		Posteriors Out	
	right	left	right	left
Lateral	→	←	→	←
Anterior	↑ ∩ ▼	↑ ▼ ∩	↑ ∩ ▼	↑ ▼ ∩
Posterior	↓ ∩ ▼	↓ ▼ ∩		
L + A + P	→ ∩ ▼	← ▼ ∩	↑	↑
Right + Left	0		↑↑	

Peripheral spontaneous nystagmus

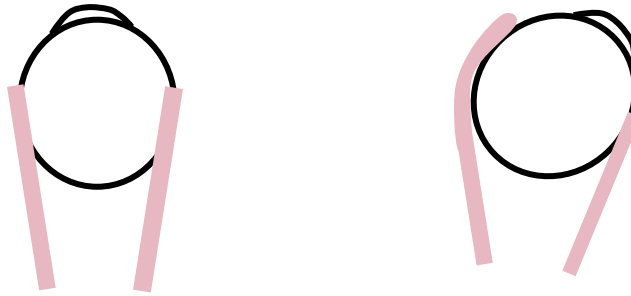


Peripheral Spontaneous Nystagmus

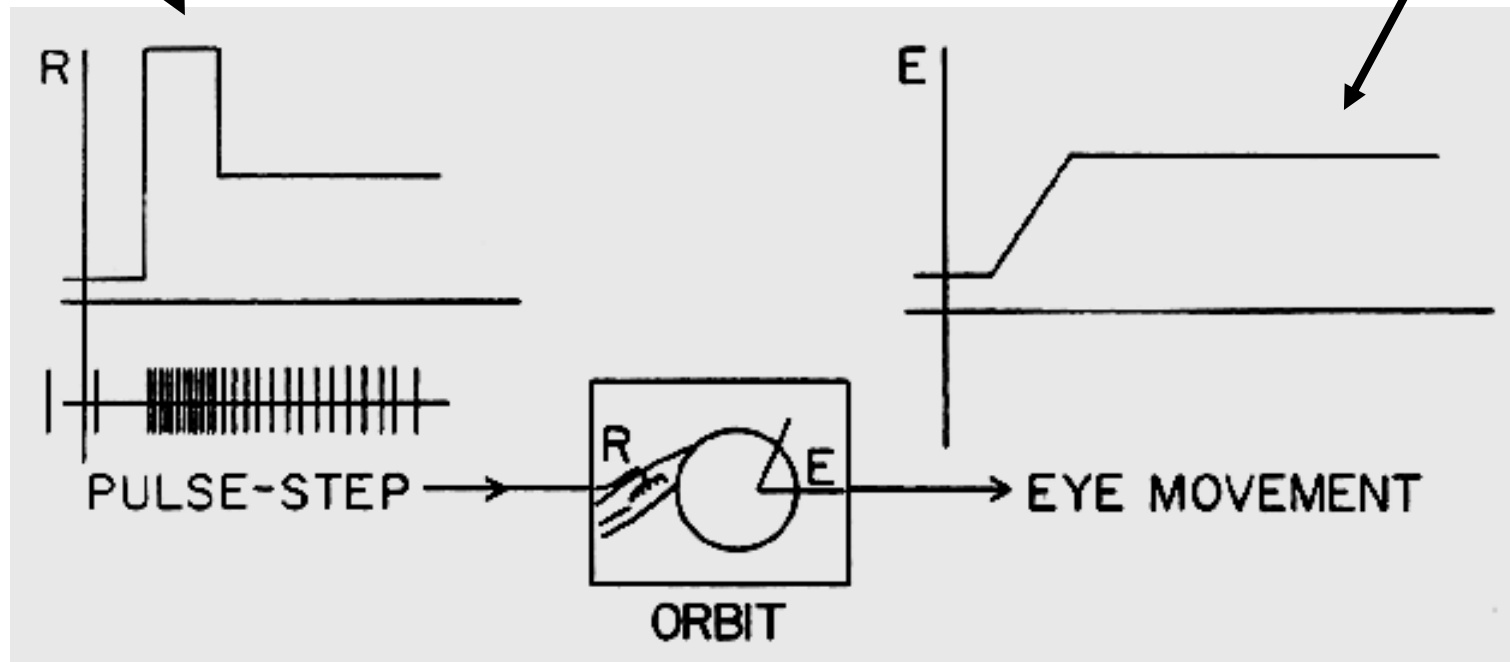
- Horizontal torsional
- Unidirectional: direction does not change depending on eye position
- Reduced by visual fixation, enhanced when visual fixation is removed (pen-light test)

What drives the saccade: Pulse-Step of innervation:
PULSE moves the eye rapidly during the saccade and
STEP holds the eye in position at end of the saccade

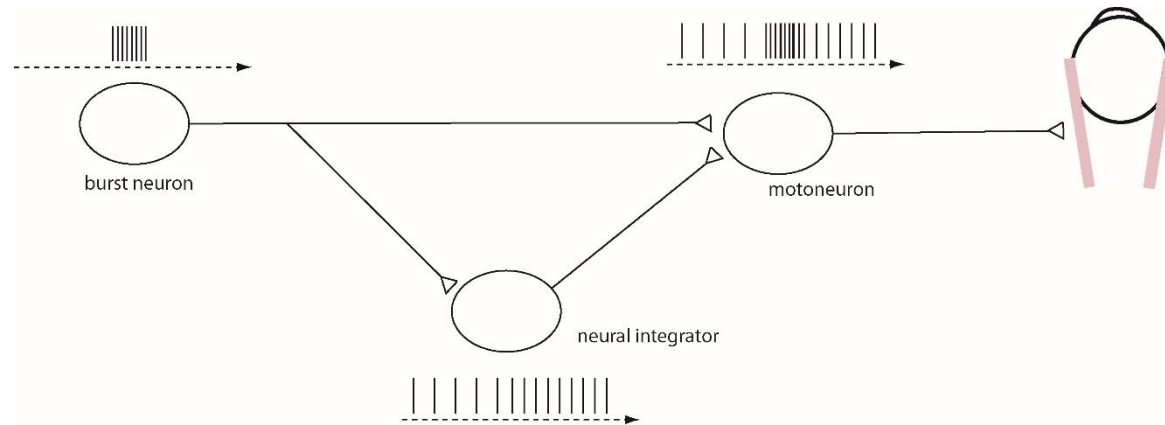
Pulse



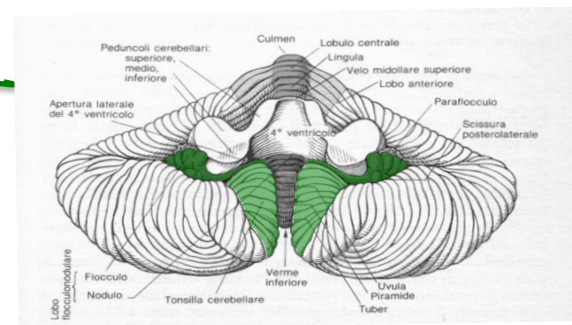
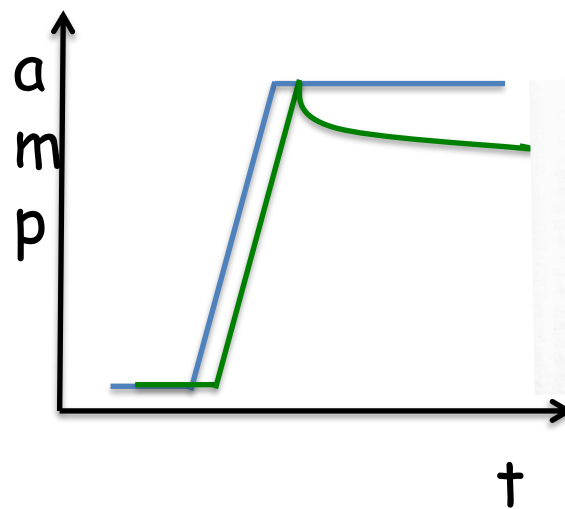
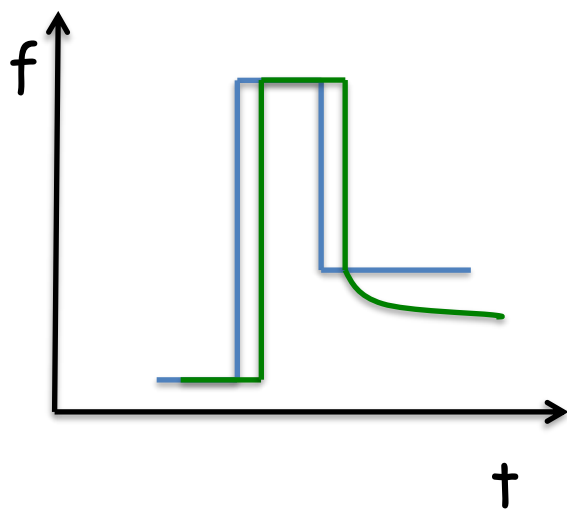
Step



Brainstem Saccade Generator



	Horizontal (pons)	Vertical (midbrain)
Burst neurons	Paramedian pontine reticular formation (PPRF)	Rostral interstitial nucleus of the MLF (riMLF)
Neural integrator	Medial vestibular nucleus/nucleus prepositus hypoglossi	Interstitial nucleus of Cajal (INC)

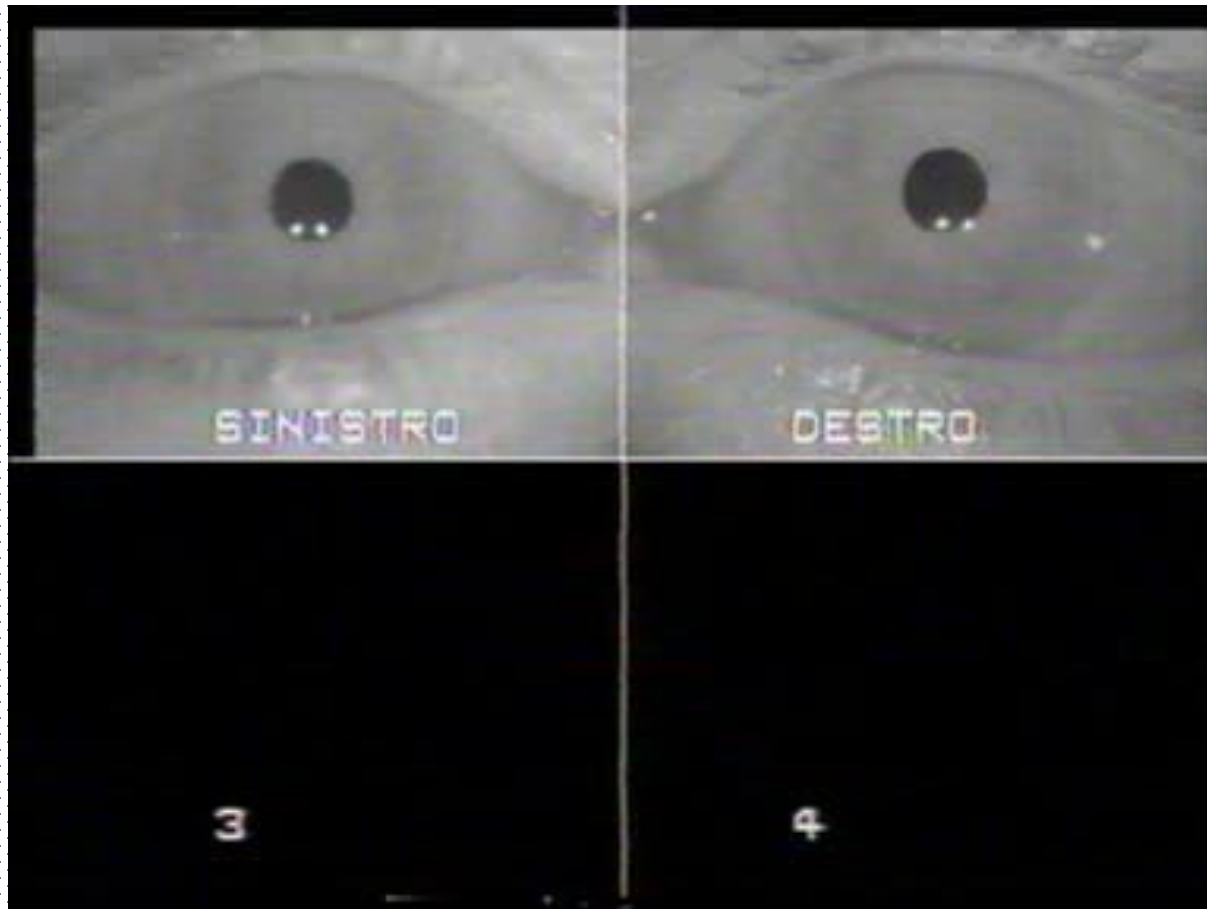


Gaze-Evoked Nystagmus

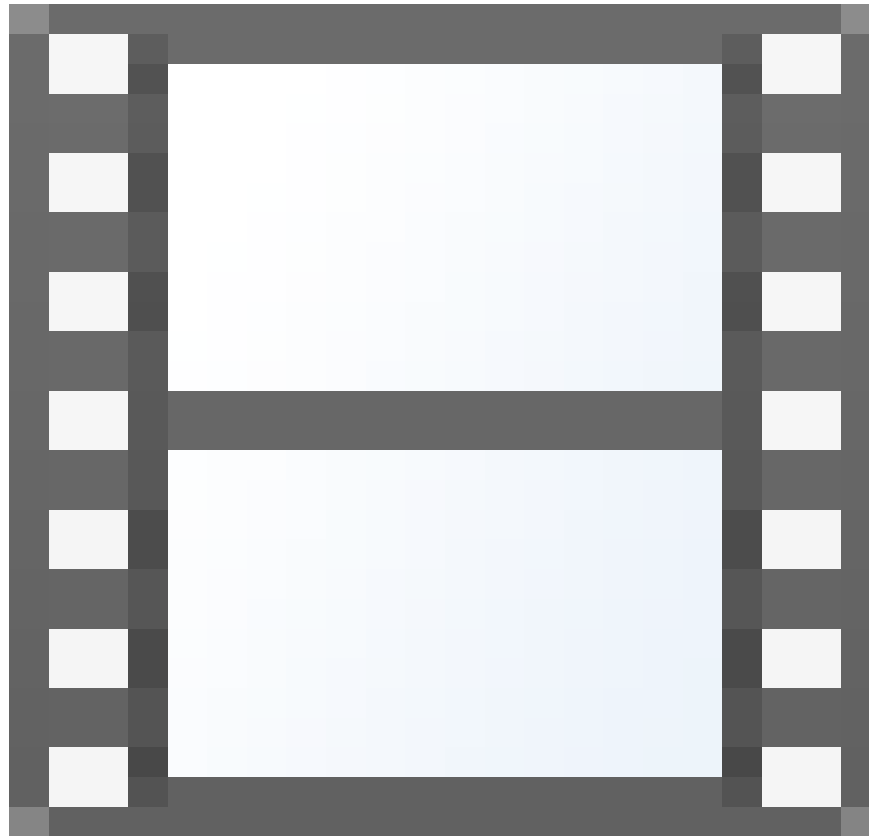
- nystagmus in lateral and/or upward and/or downward gaze beating toward gaze direction
- not influenced by visual fixation

lesion: flocculus

Gaze-evoked and rebound nystagmus



Gaze-evoked and rebound nystagmus



(physiological) end-point nystagmus

- in far-lateral gaze only
- small amplitude
- influenced (usually reduced) by visual fixation
- not associated to other floccular or cerebellar signs

Nystagmus too neurological to be considered by HINTS

- Down-beating Ny
- Up-beating Ny
- Torsional Ny
- Internuclear ophthalmoplegia
- Pendular Ny

HINTS – HEAD IMPULSE TEST

Head impulse test

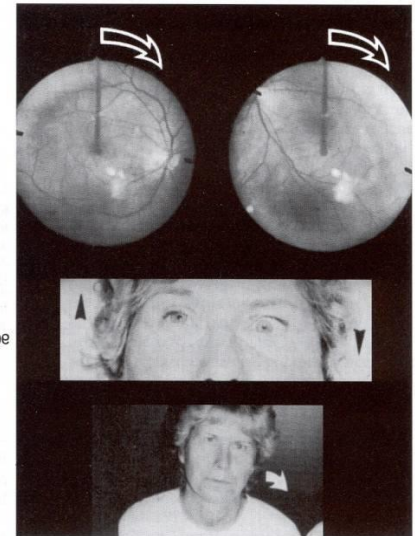
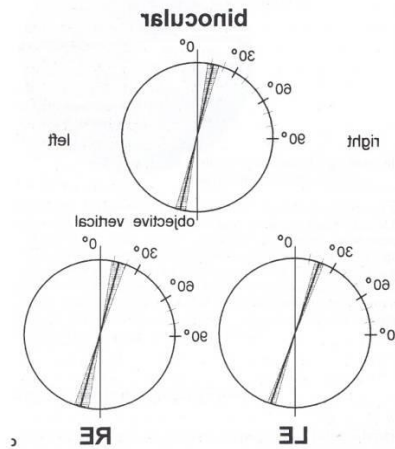
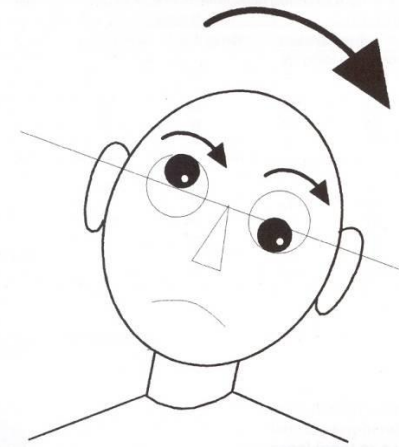
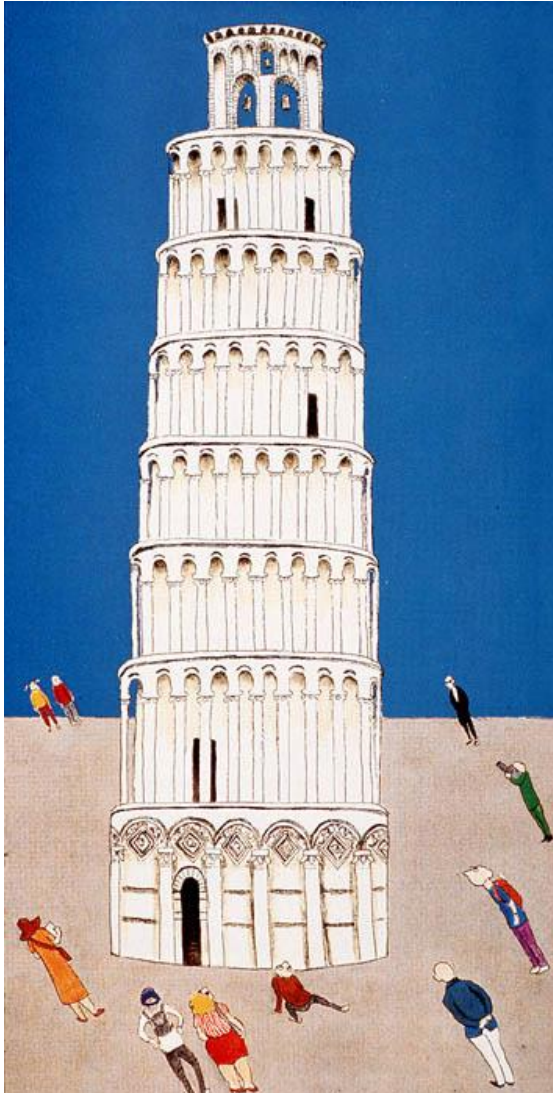
- Brisk head turn while looking at a target (the examiner's nose)
- When the head is stopped, the examinee looks at the subject's eyes
 - The VOR is normal:
 - No eye movement
 - The VOR is defective (the eyes move less than the head)
 - A saccade in the opposite direction with respect to head turn
 - The VOR is hyperactive (the eyes move more than the head)
 - A saccade in the same direction with respect to head turn

Head impulse test



HINTS – TEST OF SKEW

OCULAR TILT REACTION

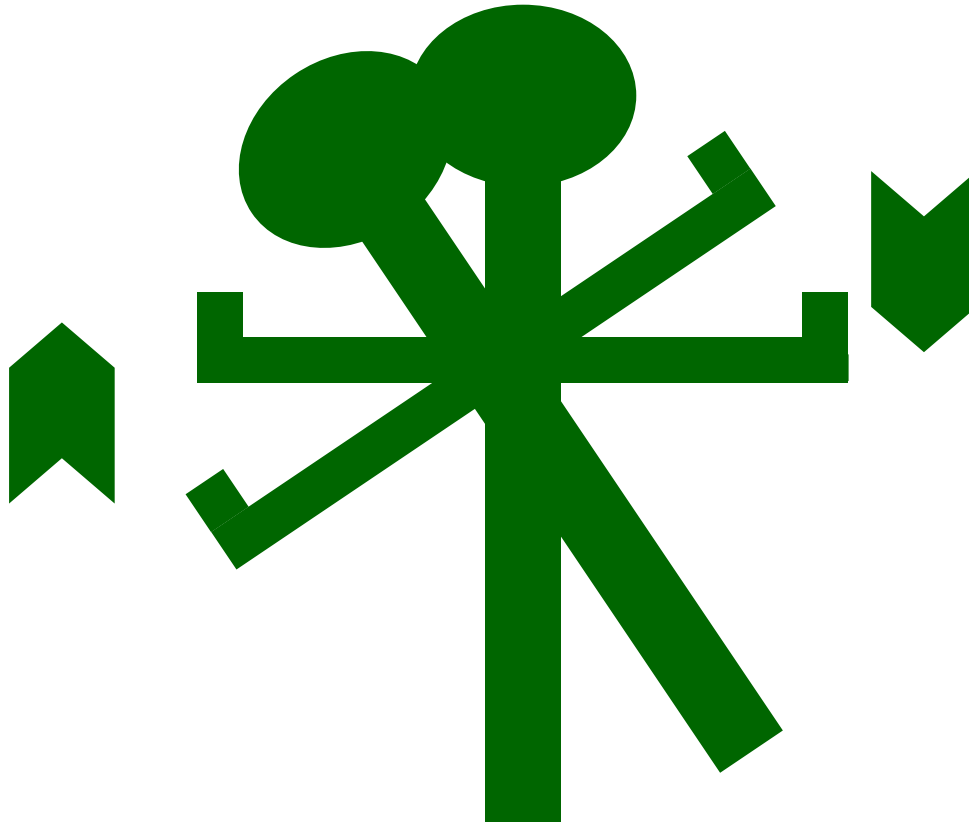


OTR – SKEW DEVIATION

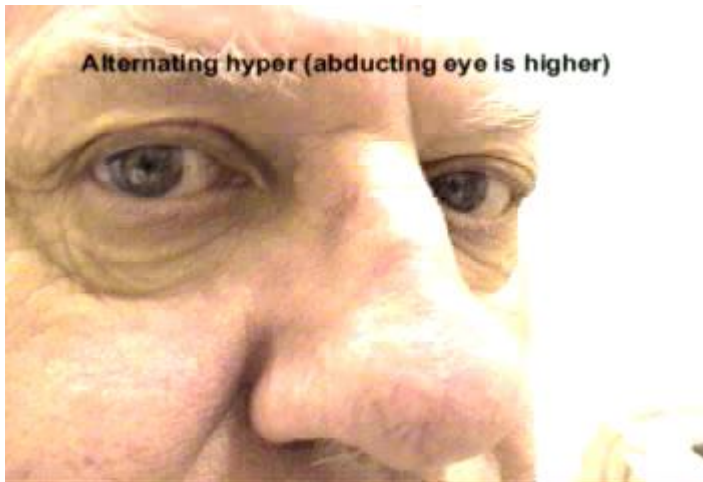
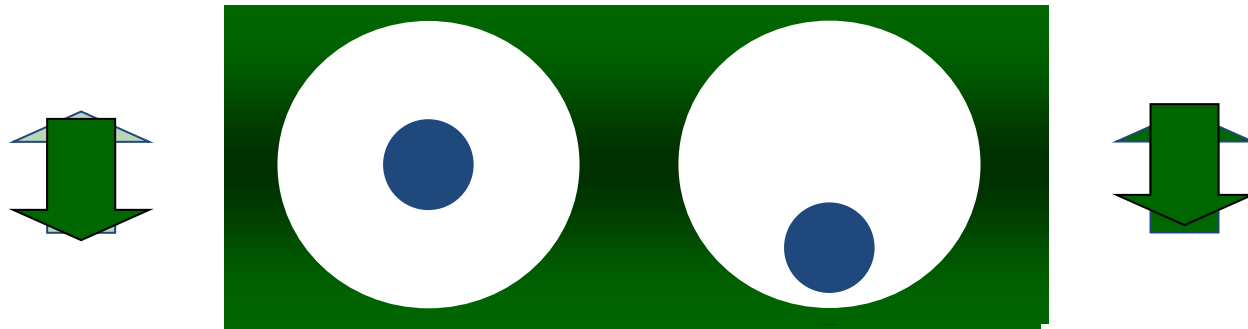
Acquired vertical comitant misalignment

Ocular misalignment = diplopia

OTR



Skew deviation: cover test



DIAGNOSIS

H(ead)I(mpulse)N(ystagmus)T(est for)S(kew)

- Vestibular Neuritis
 - *Peripheral* nystagmus **AND** *Abnormal* HIT **AND** *Normal* Cover test
- Stroke
 - *GE* nystagmus **OR** *Normal* HIT **OR** *Refixation on* Cover test

Risk evaluation

ABCD2 Score and TIA

Predictors	Point
Age	A >60 years = 1
Blood pressure	B systolic ≥ 140 mmHg, diastolic ≥ 90 mmHg = 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

Score	2-day risk
0-1	0%
2-3	1.3%
4-5	4.1%
6-7	8.1%

	Sensitivity	Specificity	NLR central cause (95% CI)
ABCD2>4	58%	61%	0.69 (0.52-0.92)
HIT	91%	100%	0.09 (0.05-0.16)
HINTS	97%	98%	0.03 (0.01-0.09)
HINTS plus	99%	97%	0.01 (0.0-0.06)

more than HINTS

- Age > 60 years
- ABCD2 score > 4
- Headache
- “Subtle” neurological (*ocular motor*) signs
- Truncal and gait ataxia
- Unable to keep upright position unassisted (STANDING)
- Positive tandem test

Table 2. Pretest and Post-Test Probabilities of Stroke Using Different Tests to Rule Out Stroke in the Spontaneous Acute Vestibular Syndrome

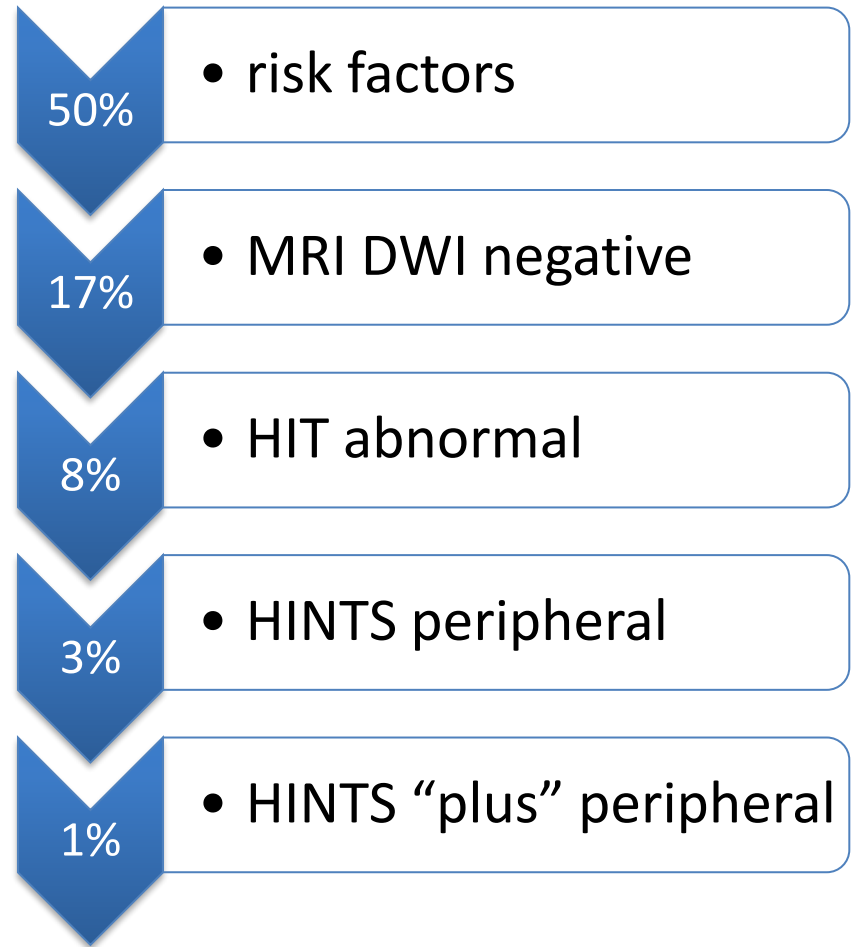
	Post-Test Probability of Stroke Following a Negative Test Obtained in First 24 h			
Pretest probability of stroke (vascular risk profile)	General neuro examination (Sn, 19% ⁵⁶ ; Sp, 95%; NLR, 0.85)	CT brain (Sn, 16% ¹³ ; Sp, 98% ¹³ ; NLR, 0.86)	MRI-DWI brain (Sn, 80% ⁵⁵ ; Sp, 96% ¹³ ; NLR, 0.21)	HINTS+Battery (Sn, 99% ⁵⁸ ; Sp, 97% ⁵⁸ ; NLR, 0.01)
10% (low)	8.7%	8.7%	2.3%	0.1%
25% (average ⁵⁵)	22.2%	22.3%	6.5%	0.3%
50% (high)	46.1%	46.2%	17.2%	0.8%
75% (very high)	71.9%	72.1%	38.5%	2.4%

CT indicates computed tomography; HINTS, head impulse, nystagmus, test of skew, plus hearing; MRI-DWI, magnetic resonance imaging with diffusion-weighted imaging; NLR, negative likelihood ratio; Sn, sensitivity; and Sp, specificity.

Adapted from Newman-Toker et al²³ with permission. Copyright ©2015, Georg Thieme Verlag KG.

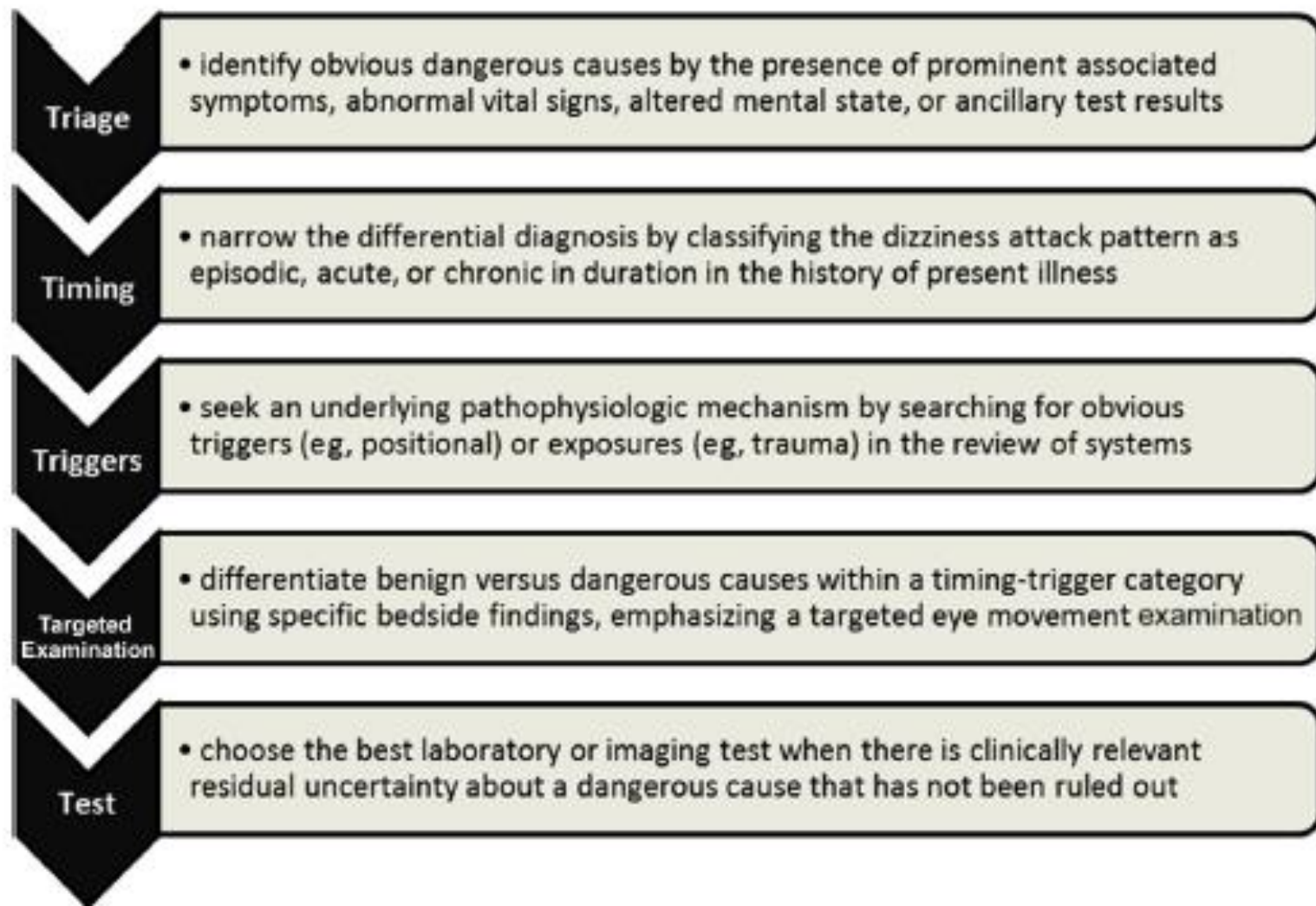
Neuroimaging

- Stroke lateral brainstem / cerebellum
- acute stage (48 hours)
 - sensibility TC: 7% (Ozono et al. 2014)
 - sensibility MRI DWI: 80
 - 90% (Tarnutzer et al., 2011; Kim et al., 2013)



TiTrATE

A Novel, Evidence-Based Approach to Diagnosing Acute Dizziness and Vertigo

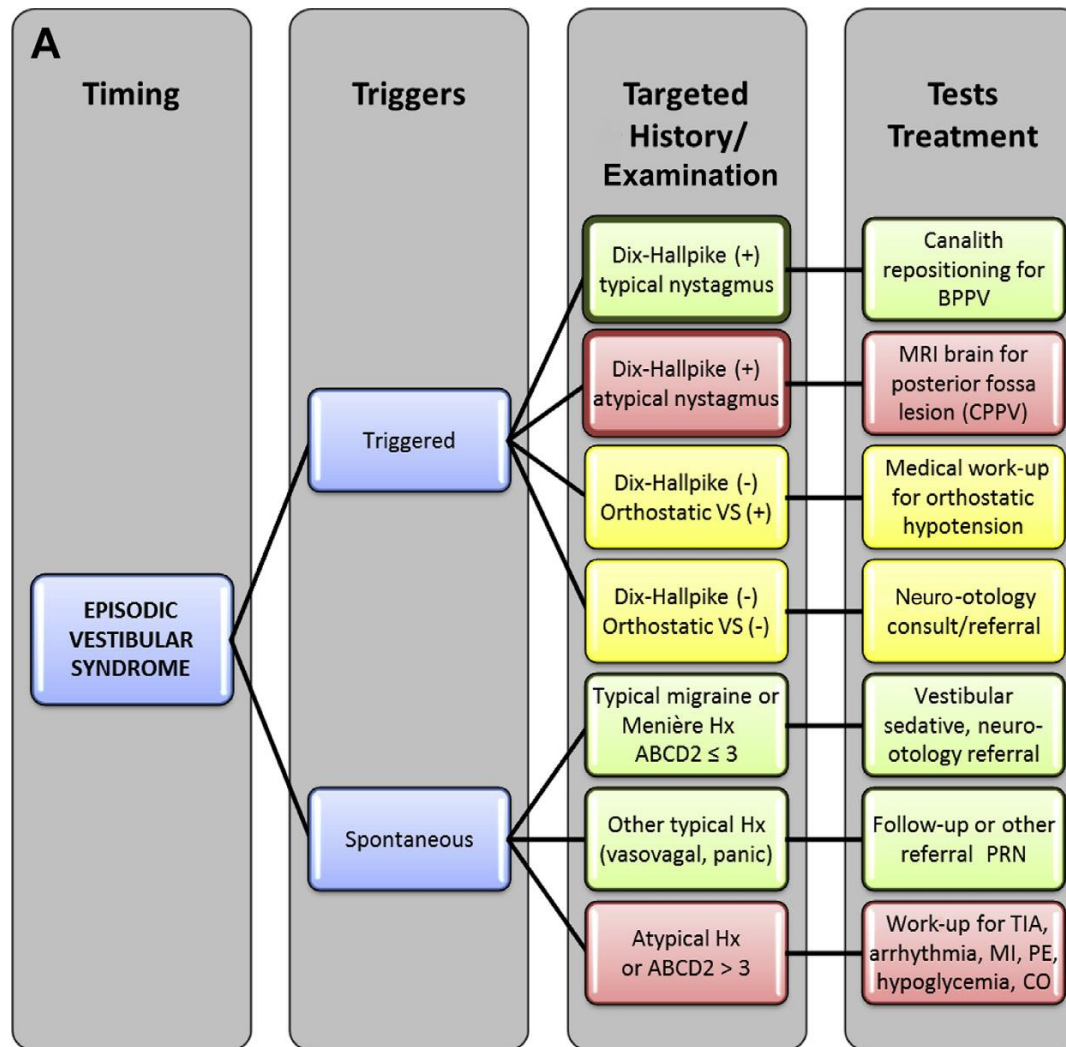


TiTrATE

A Novel, Evidence-Based Approach to Diagnosing Acute Dizziness and Vertigo

David E. Newman-Toker, MD, PhD^{a,*}, Jonathan A. Edlow, MD^b

Neurol Clin 33 (2015) 577–599

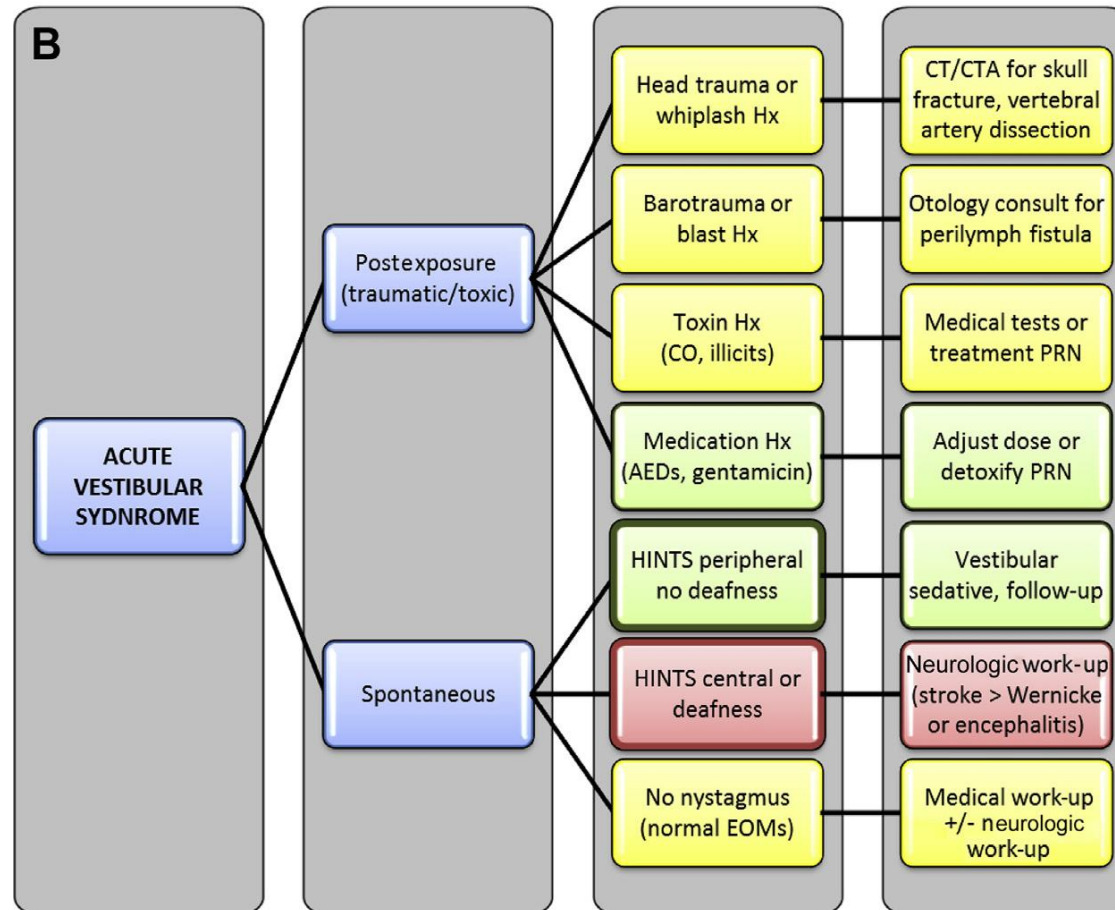


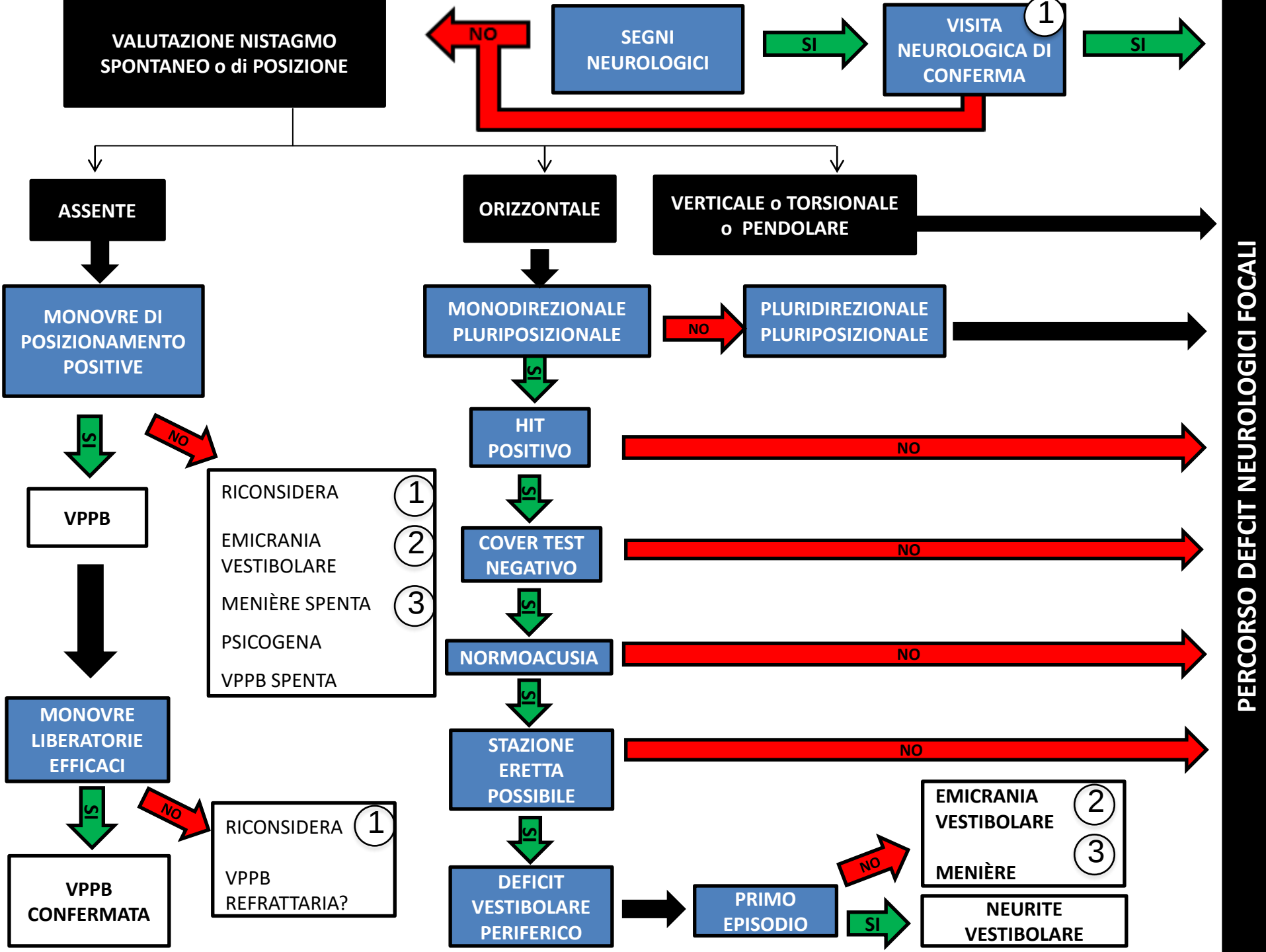
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IL NOSTRO STUDIO

- ✓ Iter diagnostico eseguito
- ✓ In quanti pazienti è stato applicato HINTS?
- ✓ In quanti pazienti è stato applicato HINTS-Plus?

1,2 pazienti al
giorno

201
pazienti

1 Gennaio 2019

13 Giugno 2019



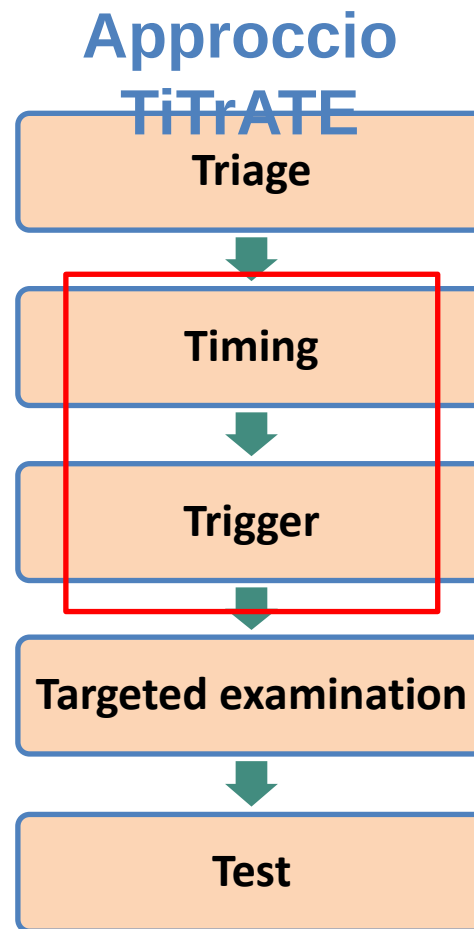
DATI RACCOLTI

- Età e sesso
- Codice priorità
- Anamnesi patologica remota e terapia domiciliare
- Anamnesi patologica prossima
- Esame obiettivo e targeted examination
- Consulenze richieste
- Esami di imaging e strumentali
- Terapia in PS
- Tempo medio di permanenza in PS
- Diagnosi di

RISULTATI: caratteristiche vertigine

Andamento temporale	N. (%)
N. di pazienti in cui è stato valutato (%), di cui:	169 (84,1%)
- Episodio unico	101 (59,8%)
- Crisi ricorrenti	25 (14,8%)
- Crisi a grappolo	42 (24,8%)
- Vertigine persistente	1 (0,6%)

Fattori scatenanti	N. (%)
N. di pazienti in cui è stato valutato (%), di cui:	195 (97%)
- Vertigine spontanea	115 (59%)
- Rotazione del capo	31 (15,9%)
- Cambi posturali	49 (25,1%)



RISULTATI: targeted

examination

	N. (%)		
Pazienti a cui non è stata richiesta nessuna consulenza	50 (24,8%)		
Pazienti a cui è stata richiesta una o più consulenze	151 (75,1%)		
Numero di consulenze richieste, di cui:	192		
- Audiovestibologia	134 (69,8%)		
- Neurologia	53 (27,6%)		
- Otorinolaringoiatria	5 (2,6%)		
Bedside test	N. pz in cui è stato valutato	Negativo	Positivo
Head shaking test	63 (31,3%)	38 (60,3%)	25 (39,7%)
Head impulse test	49 (24,4%)	44 (89,8%)	5 (10,2%)
Video HIT	21 (10,4%)	20 (95,2%)	1 (4,8%)
Skew deviation	4 (2%)	4 (100%)	0 (0%)
Manovra di Dix-Hallpike	56 (27,9%)	34 (60,7%)	22 (39,3%)
Manovra di Pagnini-McClure	18 (9%)	16 (88,9%)	2 (11,2%)
Manovra di Semont	3 (1,5%)	3 (100%)	0 (0%)
	N. (%)		
Pazienti con trigger: rotazione del capo/cambi posturali	80 (41%)		
Manovre posizionali eseguite in questi pazienti	43 (53,8%)		

Nistagmo	N. pz in cui è stato valutato	Negativo	Positivo
Nistagmo:	178 (88,6%)	108 (60,7%)	70 (39,3%)
- Spontaneo	176 (98,9%)	124 (70,5%)	52 (29,5%)
- Posizionale	10 (5,6%)	3 (30%)	7 (70%)
- Provocato	17 (9,6%)	3 (17,6%)	14 (82,4%)
Piano:	53 (29,8%)		
- Orizzonto-torsionale			4 (7,5%)
- Vertico-torsionale			3 (5,7%)
- Puro orizzontale			41 (77,4%)
- Puro verticale			4 (7,5%)
- Puro rotatorio			1 (1,9%)
Direzione:	28 (15,7%)		
- Monodirezionale			26 (92,9%)
- Pluridirezionale			2 (7,1%)
Fissazione:	1 (0,56%)		
- Inibito			1 (100%)
- Non inibito			

**SOLO NEL 2% (4) PAZIENTI
È STATO ESEGUITO IL
PROTOCOLLO HINTS
COMPLETO**

RISULTATI: indagini strumentali

Esami strumentali	N. pz in cui è stato effettuato	Negativo	Positivo
Audiometria	117 (58,2%)	58 (49,6%)	Monolaterale: 18 (15,4%) Bilaterale: 41 (35%)
Stabilometria	65 (32,3%)	49 (77,8%)	Monodirezionale: 6 (9,2%) Pluridirezionale: 10 (15,4%)
TC senza mdc	138 (68,6%)	136 (98,6%)	2 (1,4%)
Angio TC	11 (5,5%)	11 (100%)	
RMN	0 (0%)		
ECD TSA	2 (1%)	2 (100%)	

- Scarsa disponibilità in acuto
- Falsi negativi prime 48 h

- In nessun caso ha identificato la causa sottostante
- Scarsa sensibilità (7-16%) per lesioni ischemiche

RISULTATI: diagnosi di dimissione

Diagnosi dimissione	N. (%)
Vertigini	104 (51,7%)
Instabilità	3 (1,5%)
VPPB	39 (19,4%)
Neurolabirintopatia acuta/deficit vestibolare periferico	39 (19,4%)
Malattia di Ménière	4 (2%)
Emicrania vestibolare	4 (2%)
Vertigine psicogena	3 (1,5%)
Neurinoma acustico	1 (0,2%)
TIA	2 (1%)
Ictus	0 (0%)
Vertigine cervicogenica	1 (0,2%)
Vertigine iatrogena	1 (0,2%)

- Nel 53,2% diagnosi dimissione = a diagnosi d'ingresso
- Migliorare accuratezza diagnostica

Diagnosing Stroke in Acute Dizziness and Vertigo Pitfalls and Pearls

Ali S. Saber Tehrani, MD; Jorge C. Kattah, MD; Kevin A. Kerber, MD, MS;
Daniel R. Gold, DO; David S. Zee, MD; Victor C. Urrutia, MD; David E. Newman-Toker, MD, PhD

- In letteratura 3-5% delle cause
- Sottostimati?

Malattie cerebrovascolari

Prevalenza ictus cerebrali
1,6 - 6,5% nella diverse
fasce d'età

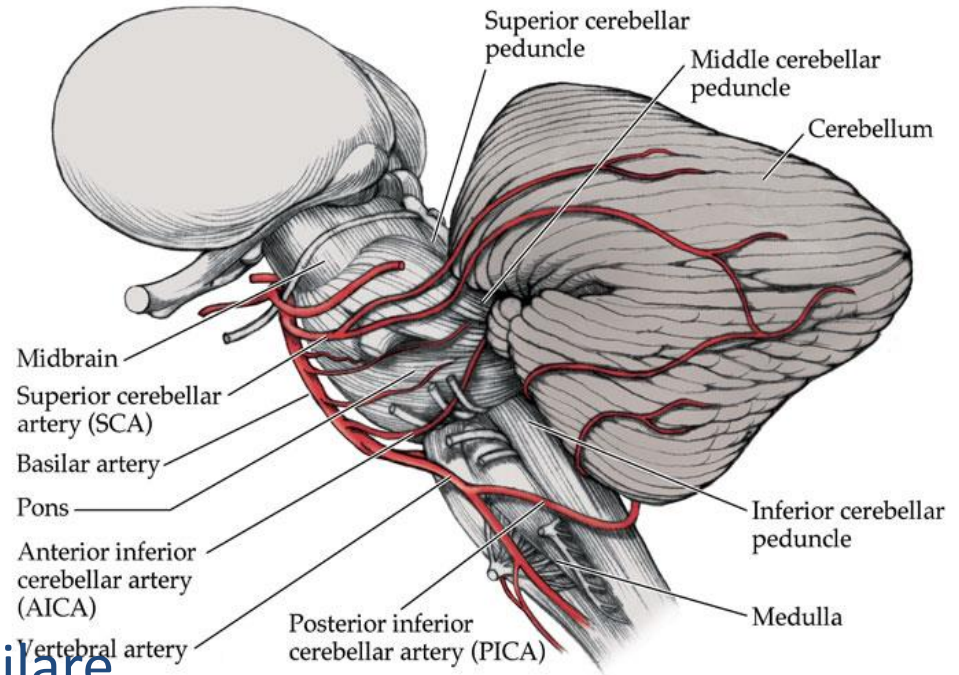
Incidenza di 4 sottotipi
di ictus cerebrali in base alla sede
(537 soggetti):

17% circolo anteriore
(completo)

34% circolo anteriore
(parziale)

24% territorio vertebrobasilare

25% arterie perforanti



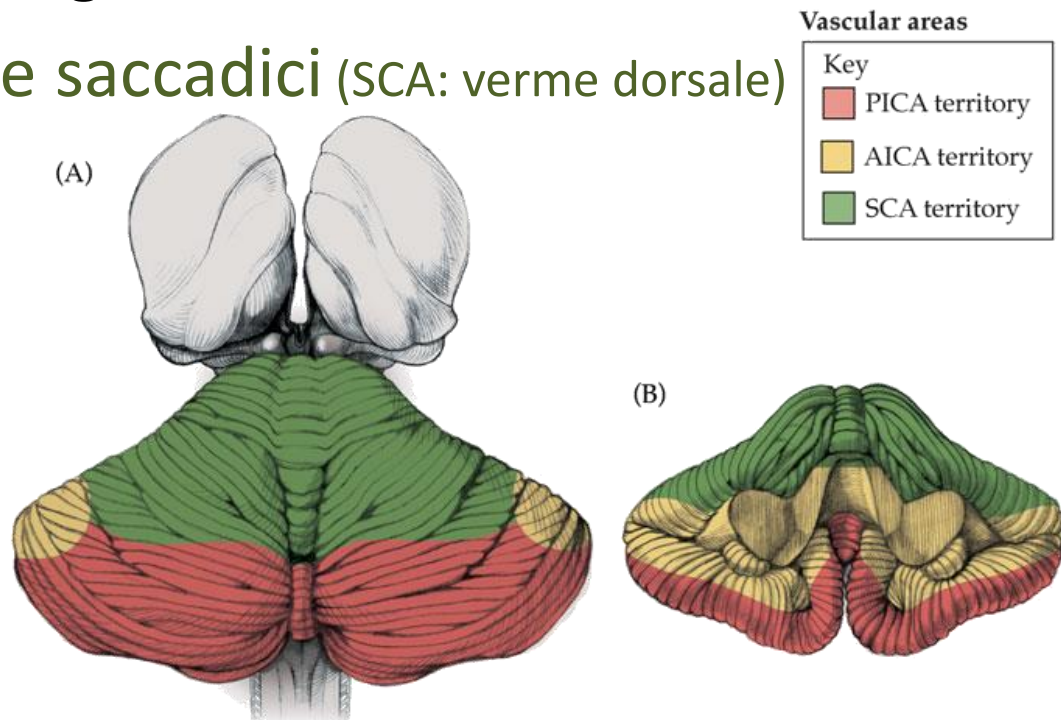
© 2002 Sinauer Associates, Inc.

Bamford J et al. **Classification and natural history of clinically identifiable subtypes of cerebral infarction.** Lancet. 1991 Jun 22;337(8756):1521-6.

Malattie cerebrovascolari

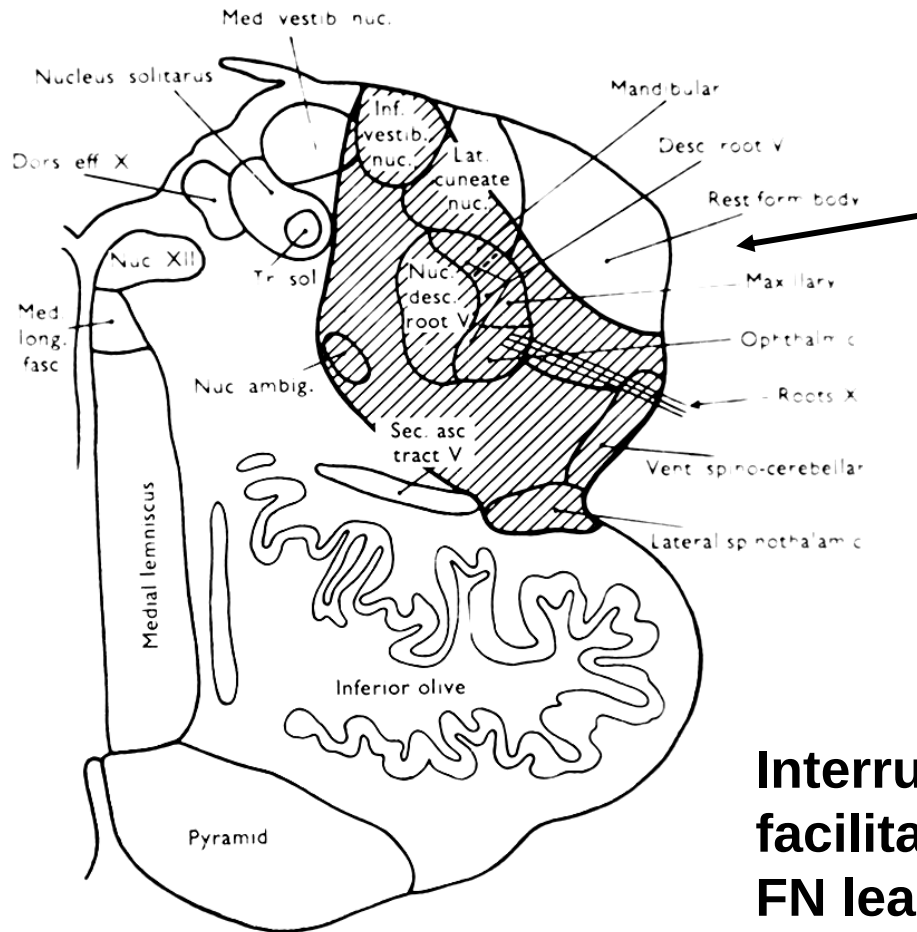
Quadri sindromici determinati dal territorio vascolare interessato:

- **Sindrome di Wallenberg** (PICA: porzione laterale bulbo, peduncolo cerebellare inferiore, nodulo ed uvula)
- **Downbeat nystagmus** (AICA: nuclei vestibolari, flocculo)
- **Contrapulsione saccadici** (SCA: verme dorsale)

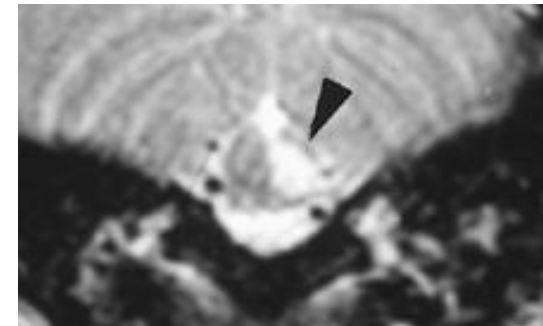


SINDROME DI WALLENBERG

Wallenberg's Syndrome – PICA distribution infarct involving the dorsolateral medulla

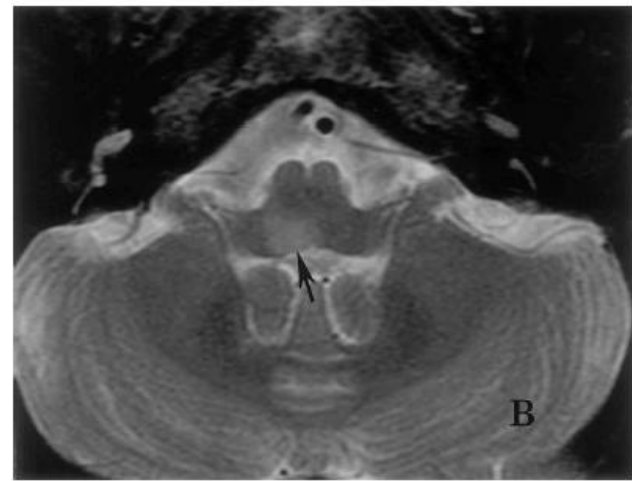


Restiform body (ICP)



Interruption of climbing fibers facilitates Purkinje Cell inhibition of FN leading to a *functional* lesion of the FN and produces saccade lateropulsion

Sindrome di Wallenberg



• Lesione

- Tratto discendente e nucleo del V
- Fibre del IX e X
- Peduncolo cerebellare?
- Vie simpatiche pupillari
- Nuclei gracile e cuneato
- Nucleo e tratto solitario
- ?
- Tratto spino-talamico

• Segni ipsilaterali

- Ipoestesia termo-dolorifica emivolto
- Disartria e disfagia
- Atassia segmentaria.
- Sd. di Horner ipsi
 - Ptosi + Miosi + Enoftalmo
- Ipoestesia tattile discr.emisoma
- Anageusia
- Singhiozzo

• Segni contralaterali

- Ipoestesia termo-dolorifica emisoma

Sindrome di Wallenberg – segni vestibolari ed oculomotori

- Lesione

- Nuclei vestibolari + vie otolitiche + fibre olivo-cerebellari (= ipofunzione nucleo del fastigio)

- Sintomi

- Vertigine e lateropulsione verso il lato leso

Sindrome di Wallenberg – segni vestibolari ed oculomotori

- Al buio occhi deviano verso il lato lesa (“lateropulsion”)
- Ny spontaneo orizzonto-rotatorio (con possibile componente verticale)
- Ocular Tilt Reaction (inclinazione della testa ipsi + skew deviation con ipotrofia ipsi + ciclorsione ipsi + inclinazione della verticale visiva soggettiva ipsi)
- Saccadici orizzontali ipermetrici verso il lato lesa e ipometrici verso il lato sano (“ipsipulsion”)
- Saccadici verticali→obliqui (hanno una componente orizzontale verso il lato lesa)
- Inseguimento lento deficitario verso il lato sano
- VOR : preponderanza direzionale verso il lato lesa

VERTIGINI E SCLEROSI MULTIPLA

MS – the essentials

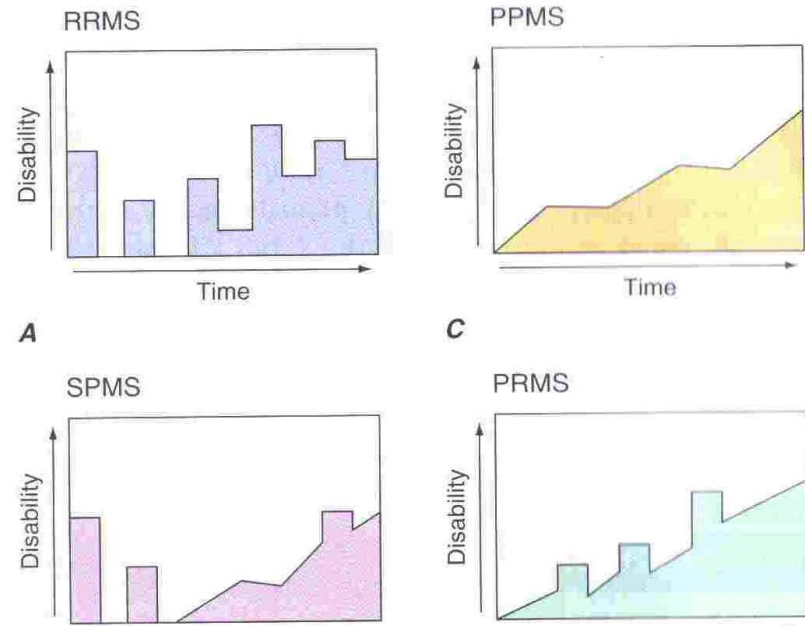
- MS is a chronic disease characterized by inflammation, demyelination (*central not peripheral, oligodendrocytes not Schwann cells*), gliosis and neuronal loss

MS – the essentials

- The more typical disease course is Relapsing Remitting
- Relapse = discrete attack that evolves over days to week

MS – the essentials

- Disease courses
 - Relapsing-remitting (RR) – 85%
 - Secondary-progressive (SP)
 - Primary-progressive (PP) – 15%
 - Progressive-relapsing (PR) = a mix of PP and SP



- **Relapse means inflammation, progression means degeneration**
- From RR to SP: about 2% each year
- In 15 years about 75% develop a secondary progressive course

MS – the essentials

- Diagnosis:
 - there are several diagnostic criteria BUT the diagnosis is clinical (history + examination) and needs (RR) dissemination in space and time = two different sites/relapses (symptoms > 24 h) at different time (interval > 1 month)

Sintomi iniziali (%)

Età di esordio	Neurite ottica	Diplopia /vertigine	Motorio	Atassia	Sensitivo
<20	23	18	10	14	46
20-29	23	12	13	11	52
30-39	13	11	21	15	44
40-49	9	17	34	13	33
>=50	6	13	51	11	32

Modificata da Weinshenker et al., 1989

Sindrome clinicamente isolata

- CIS: Clinically Isolated Syndrome

- Nell'85% dei pazienti che svilupperanno MS l'esordio è dato dalla comparsa acuta o subacuta di sintomi neurologici dovuti ad una singola lesione della sostanza bianca; tale presentazione è nota come CIS.

- Dopo 14 anni il 68% delle CIS sviluppa una MS.

- Presentazione delle CIS:

- 21% NORB

- 46% sintomi e segni di sofferenza delle vie lunghe

- 10% sindrome troncoencefalica

- 23% sintomi multifocali

- La disseminazione spaziale manca nel 77% delle CIS
 - E' sempre assente la disseminazione temporale

Sintomi troncoencefalici / cerebellari (%)

Sintomo	All'esordio	Nel corso della malattia
vertigine	4.3	36
diplopia	8	51
atassia	11	82
disartria	13	44
Ipoacusia	0.6	17
Disfagia	0.3	13

La causa più frequente di vertigine nella SM? VPPB

Vertigo in MS: Utility of positional and particle repositioning maneuvers

Article abstract—A 4-year experience with new-onset vertigo in a university-based MS population was retrospectively reviewed. Of 1,153 patients with MS, 25 could be clinically evaluated during the vertiginous episode. Of these, 13 (52%) were diagnosed with benign paroxysmal positioning vertigo and eight (32%) had acute MS exacerbations with corresponding lesions within the brainstem. All patients diagnosed with benign paroxysmal positioning vertigo were treated successfully with particle repositioning maneuvers.

NEUROLOGY 2000;55:1566–1568

E.M. Frohman, MD, PhD; H. Zhang, MD, PhD; R.B. Dewey, MD; K.S. Hawker, MD; M.K. Racke, MD;
and T.C. Frohman, BA



Positional nystagmus and vertigo due to a solitary brachium conjunctivum plaque

E Anagnostou, D Mandellos, G Limbitaki, A Papadimitriou and D Anastasopoulos

J. Neurol. Neurosurg. Psychiatry 2006;77:790-792

doi:10.1136/jnnp.2005.084624



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Short communication

A minute demyelinating lesion causing acute positional vertigo

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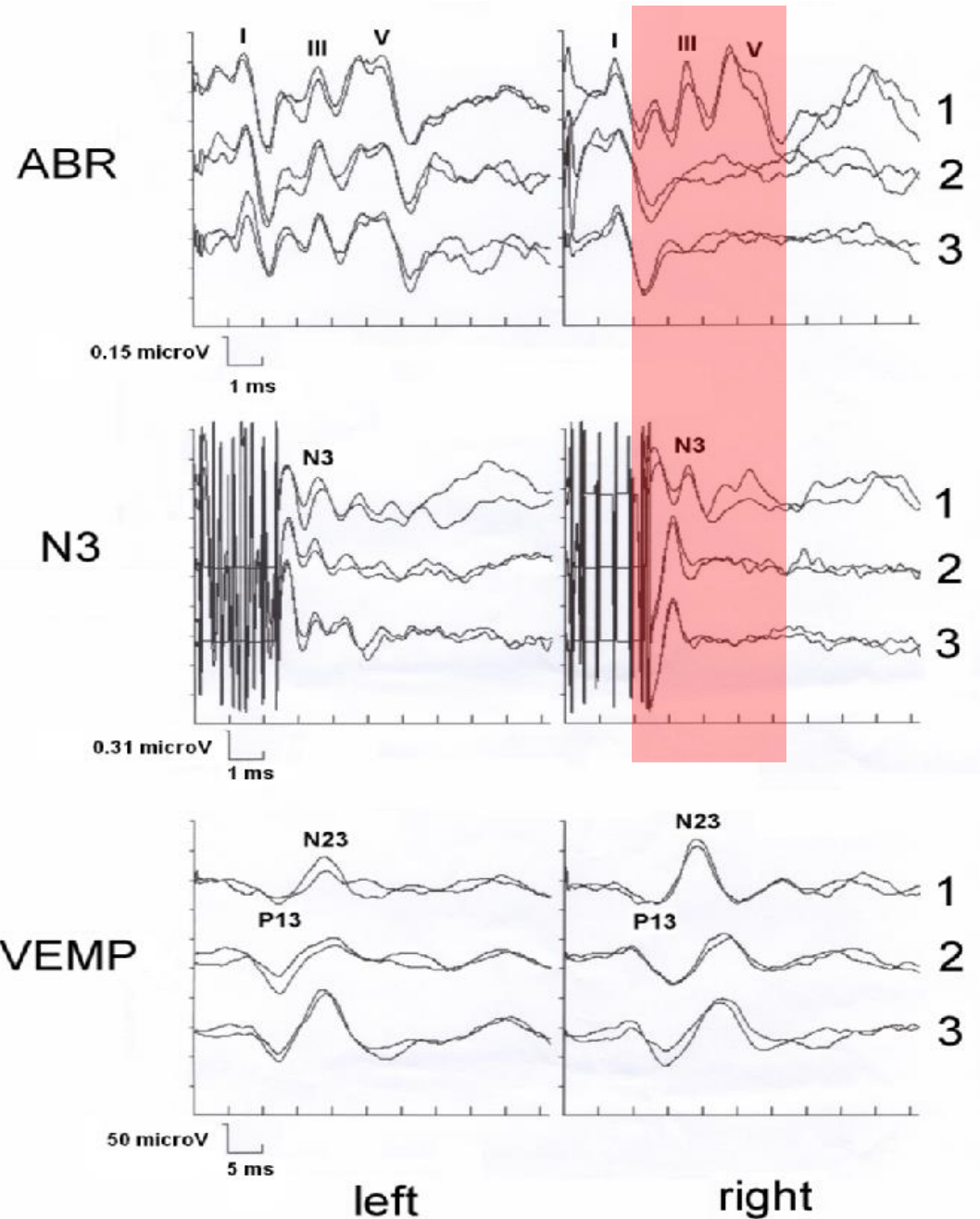
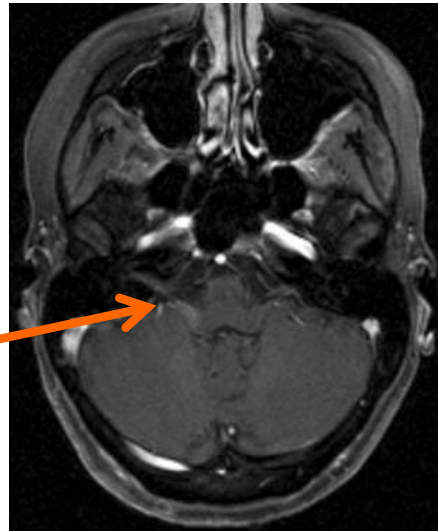
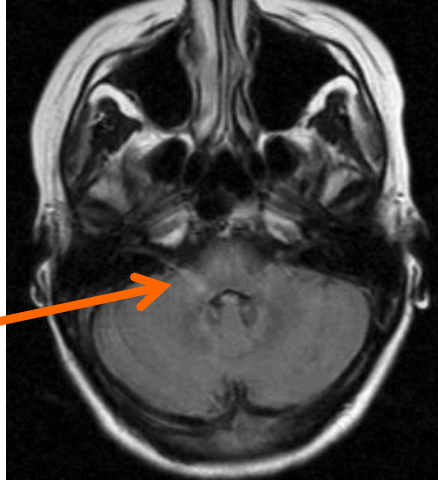
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“Neurite” vestibolare ed SM



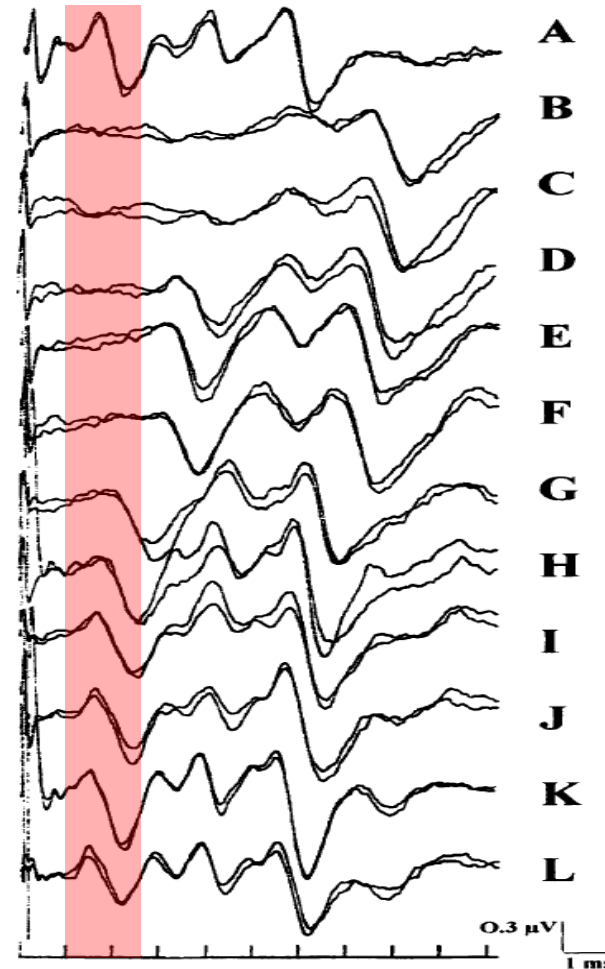
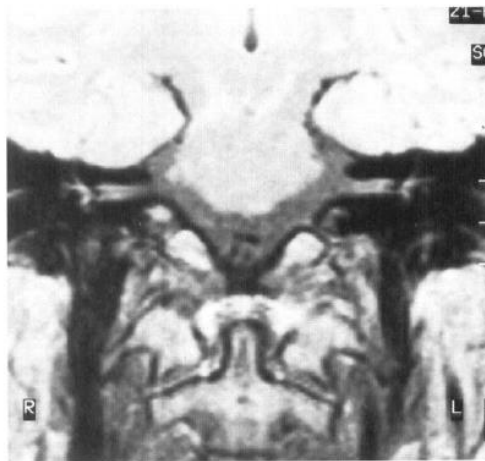
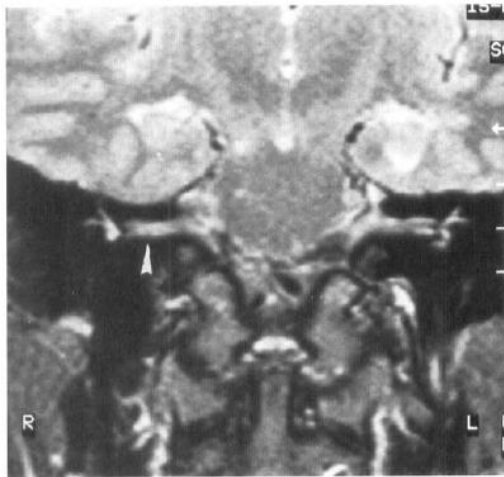
“Neurite” cocleare ed SM

**MRI and brainstem
auditory evoked
potential evidence of
eighth cranial nerve
involvement in
multiple sclerosis**

Article abstract—An MS patient experienced sudden hearing loss. Brainstem auditory evoked potentials, previously normal, showed substantial abnormalities that suggested the impairment of the distal part of the acoustic nerve. MRI detected a small hyperintense lesion along the acoustic nerve; the lesion decreased in size and then disappeared after steroid treatment. This demonstrates that a demyelinating lesion in the distal tract of the eighth cranial nerve may cause an acute hearing loss in MS.

NEUROLOGY 1997;48:270-272

R. Bergamaschi, MD; A. Romani, MD; F. Zappoli, MD; M. Versino, MD; and V. Cosi, MD



Pseudo-neurite vestibolare ed SM



Bedside differentiation of vestibular neuritis from central "vestibular pseudoneuritis"

C D Cnyrim, D Newman-Toker, C Karch, T Brandt and Michael Strupp

J. Neurol. Neurosurg. Psychiatry 2008;79:458-460
doi:10.1136/jnnp.2007.123596

Table 1 Clinical characteristics, frequencies of categorical clinical signs, group differences and correlation between signs/parameters and final diagnosis.

	Vestibular pseudoneuritis	Vestibular neuritis	Group difference (test)	R ² (correlation assessed by)
n	43	40		
Age (y) (mean (SD))	53 (17)	54 (14)	p = 0.82 (t test)	
Sex (F:M)	24:19	20:20	p = 0.60 (χ^2 test)	
Side of lesion (left:right)	23:20	24:16	p = 0.55 (χ^2 test)	
Frequency of smooth pursuit deficit (%)	88	20	p < 0.01 (χ^2 test)	0.37 (χ^2 test)
Frequency of gaze evoked nystagmus (%)	56	17	p < 0.01 (χ^2 test)	0.12 (χ^2 test)
Frequency of skew deviation (%)	40	0	p < 0.01 (χ^2 test)	1.00 (logistic regression)
Frequency of pathological head thrust sign (%)	39	92	p < 0.01 (χ^2 test)	0.26 (χ^2 test)
Frequency of subjective visual vertical (%)	100	100	p = 1 (χ^2 test)	0.01 (logistic regression)

Oftalmoplegia internucleare

