Vibration-Induced Nystagmus –
A simple clinical test for peripheral vestibular asymmetry

British Academy of Audiology Conference

Manchester, Tuesday 19th November 2013

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Clinical tests for peripheral vestibular asymmetry

- Spontaneous / gaze-evoked nystagmus +/- fixation
- Head-shaking nystagmus (fixation removed)
- Head thrust

Laboratory tests for peripheral vestibular asymmetry

- Spontaneous / gaze-evoked nystagmus (VNG)
- Head thrust (vHIT)
- c-VEMPs / o-VEMPs
- Rotatory chair (VNG)
- Calorics (VNG) – the “Gold Standard”
Background


1995 - Halmagyi et al.: proposed the use of skull taps as a method of vestibular activation.

1999 - Hamann: indicated the importance of this test for the detection of acoustic neuromas.
Method

- Patient seated.
- Fixation abolished with infra-red video-Frenzel’s goggles. *(Or full VNG recording)*
- Observe for (baseline) spontaneous / gaze-evoked nystagmus.
Comparison of techniques for identification of peripheral vestibular nystagmus

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Abstract
Objective: To determine the best clinical method for identifying peripheral vestibular nystagmus, by comparing eye movement examination with optic fixation, and with fixation removed using Frenzel’s glasses, infra-red video-Frenzel’s goggles or an ophthalmoscope, with results of electronystagmography.

Method: One hundred patients referred for electronystagmography from the audiovestibular medicine clinic at Queen Alexandra Hospital, Portsmouth, were examined immediately before undergoing electronystagmography.

Results: Video-Frenzel’s goggles were highly effective at detecting peripheral vestibular nystagmus, with a sensitivity of 85 per cent (95 per cent confidence interval, 62.1–96.8 per cent) and a specificity of 65 per cent (53.5–75.3 per cent), compared with electronystagmography. Ophthalmoscopy had comparable sensitivity to Frenzel’s glasses (used in the dark), i.e. 26.3 per cent (9.1–51.2 per cent) compared with 31.6 per cent (12.6–56.6 per cent), respectively. Frenzel’s glasses as normally used in ENT clinics (i.e. in dim lighting) were ineffective, with a sensitivity of just 10 per cent (1.2–31.7 per cent).

Conclusion: Video-Frenzel’s goggles should be used in all clinics with substantial numbers of balance-impaired patients. Traditional Frenzel’s glasses have no place in clinical practice unless formal black-out facilities are available.

Key words: Vertigo; Diagnosis; Nystagmus; Electronystagmography; Frenzel’s Glasses
**Method**

- Patient seated, fixation abolished with infra-red video-Frenzel’s goggles.
- Observe for (baseline) spontaneous / gaze-evoked nystagmus.

**“Synapsys” Vestibular Vibrator**
- Frequency: (30), 60 and 100 Hz
- Amplitude: 1 mm
- Sound pressure level 95 – 101 dB
Synapsys Vestibular Vibrator
Method

• Patient seated, fixation abolished with infra-red video-Frenzel’s goggles.

• Observe for (baseline) spontaneous / gaze-evoked nystagmus.

“Synapsys” Vestibular Vibrator
  - Frequency: (30), 60 and 100 Hz
  - Amplitude: 1 mm
  - Sound pressure level 95 – 101 dB

• Demonstrate vibrator on (eg) back of hand.
  Vibrations of 60 & 100 Hz, applied (firm pressure) for 10 seconds to each mastoid in turn.
Best effect on mastoid tip or at level of EAM.
Test both sides.
(Original description suggested vibration over lower part of either sternocleidomastoid muscle. Avoiding carotid body!)

Observe for (sustained) nystagmus with fixation removed.
Positive Response

- Horizontal nystagmus that beats in the same direction, for vibration on both mastoids.
- Nystagmus that beats in different directions according to the side of vibration is considered either normal or of unknown significance.
- Nystagmus other than horizontal is considered either normal or of unknown significance.
- Nystagmus begins immediately and persists for the duration of the vibration. (No adaptation over 40 sec, Hamann, 1999.)
- Slow phase (generally) to the under-active side.
- To be considered abnormal, the slow phase of the nystagmus must be more than 2.9°/sec (Boniver, 2008).
Patient has a 60% left CP.

3 deg/sec right-beating spontaneous nystagmus increases to about 10 deg/sec when 60 Hz vibration is applied to either sternocleidomastoid.

(Taken from T C Hain website)
Case 1: 66 yr old lady – seen AVM July 2011

- 16 month history of BPPV
- S/B ENT 2004: 18 yrs mainly (R) sided tinnitus
  normal audiogram, normal MRI

On examination:
- No nystagmus +/- fixation
- “4/10” (R) BPPV

Treatment:
- (R) Epley manoeuvre

Outcome:
- Good result (phone message)
Re-referred October 2011

• No more vertigo
• Still “drunk” and off-balance, mainly on movement.

**Examination:**
Normal. Except…
• Mild **right** beating head-shaking nystagmus
• Intense **left** beating vibration-induced nystagmus 60>100 Hz

**Further History:**
Episode of acute vertigo 20+ yrs ago. (Forgotten all about it.)
? Vestibular neuritis. **If** so, **which ear?** - BPPV was on (**R**)

**Management:**
Vestibular (gaze-stabilisation) exercises
VNG / Calorics / VEMPs
VNG / Calorics / VEMPs

**VNG, eyes open in dark:**
- Spontaneous 2\textsuperscript{nd} degree nystagmus (2 deg/s) to (L)
- Head-shaking nystagmus: 24 deg/s to (R) for 25 sec
  (same as I found on exam)

**Calorics:**
- 51\% (R) canal paresis
  (brisk response on (L): 22 and 26 deg/sec)

**VEMPs:**
- Normal
Conclusion:

(L)-beating vibration-induced nystagmus correctly predicted 51% (R) CP.

(R)-beating head-shaking nystagmus was non-localising.
• Relationship between VIN and Calorics
Vibration-induced nystagmus in patients with vestibular disorders

M Ohki, T Murofushi, H Nakahara, K Sugasawa.

Subjects & Methods
• 100 patients with unilateral vestibulocochlear disorders (but no spontaneous nystagmus).
• Vibration (100 Hz) was applied to the mastoids and the forehead.
• Patients also underwent caloric testing.

Results
• VIN was more frequently evoked on the mastoids than the forehead.
• VIN slow-phase was usually toward the affected side.
• Some patients (especially with Meniere’s disease) showed nystagmus with slow-phase toward the healthy side.
• VIN was evoked in 39 of 43 (90%) of patients with Canal Paresis >50%.
Clinical Significance of Vibration-Induced Nystagmus


- 22 Vestibular Neuritis patients and 24 Meniere’s Disease patients tested using 100-Hz mastoid vibration and caloric testing.
- 21/22 VN patients had Canal Paresis >25% and VIN with slow-phase toward the lesioned side.
- 15/24 (63%) MD patients had VIN with slow-phase to the lesioned side.
- 9/24 (38%) MD patients had a pathologic CP. 8 of these 9 had VIN with slow-phase to the lesioned side.
- 8 other MD patients had VIN with slow-phase to the intact side. (3 of these had a CP on the intact side!)
- The VIN slow-phase velocity showed a significant correlation with CP in both VN and MD patients.

Conclusion:

- VIN may reveal peripheral vestibular asymmetry to low-frequency stimulation similar to that shown by the caloric test, but using a less unpleasant method of stimulation.
Review of literature - 2

• Relationship between VIN and calorics

• Comparison with head-shaking nystagmus
High-frequency skull vibration-induced nystagmus test in partial vestibular lesions.

Dumas G, Karkas A, Perrin P, Chahine K, Schmerber S.


- VIN at 30, 60 & 100 Hz compared with head-shaking and caloric tests.

- 99 patients with partial unilateral vestibular lesion.

- 131 patients with total unilateral vestibular lesion.

- 95 controls.
High-frequency skull vibration-induced nystagmus test in partial vestibular lesions.

Dumas G, Karkas A, Perrin P, Chahine K, Schmerber S.


• VIN at 30, 60 & 100 Hz compared with head-shaking and caloric tests.

• 99 patients with partial unilateral vestibular lesion.
  75% had VIN. Slow phase to affected side in 91%. Direction opposite to HSN in 30%.

• 131 patients with total unilateral vestibular lesion.
  98% had VIN. Slow phase to affected side in all. Direction same as HSN in all.

• 95 controls.
  6% with normal calorics had VIN (specificity 94%) ALL over 70.
Review of literature - 3

- Relationship between VIN and calorics
- Comparison with head-shaking nystagmus
- Test – retest reliability
Test-retest reliability of vibration-induced nystagmus in peripheral dizzy patients.


- 52 consecutive patients with VIN at four different stimulation sites (both mastoids and sternocleidomastoid muscles) underwent VIN test in 2 sessions, 30 minutes apart.
- Max. slow-phase velocities of VIN at different sites from first and second sessions were compared using Intraclass Correlation Coefficient and Pearson Correlation Coefficient.
- “Substantial-to-excellent correlation” was obtained for the max. slow-phase velocities at different sites.
- Incidence of directional changes of VIN was 0-4% at each site.
- 43 patients (83%) had abnormal results in the first session and 41 (79%) in the second session.
- Direction and max slow-phase velocities of VIN for different stimulation sites had excellent test-retest reliability.
Conclusions: *(Dumas et al.)*

- “The caloric test should no longer be considered as an absolute reference test in the diagnosis of PV lesions because a normal caloric cannot exclude vestibular pathology.”

- “…a vibration nystagmus test is required among other vestibular tests to improve the clinical assessment in vestibular diseases.”

*Calorics may be less “Gold-Standard” than “Flint-Standard” – they were cutting-edge in the stone-age.*
How does VIN work?

Simultaneous stimulation of both horizontal SCCs

*In total unilateral canal paresis, slow-phase velocity of VIN correlates with max. SPV of caloric response on intact side.*

Frequency too high to involve endolymph flow

*A “Vestibular Weber Test” analogous to bone conduction audiometry?*

Stimulates all 6 canals?

*Vertical VIN may be seen in unilateral Superior SCC dehiscence.*

Utricular test?

*100 Hz vibration may elicit an o-VEMP and affect subjective visual horizontal.*

VIN may test the whole vestibular labyrinth.
Case 2: 37 yr old male — seen AVM June 2013

- Variable symptoms most days since March.
- No vertigo.
- Palpitations / slight breathlessness when dizzy. (GP initially thought panic attacks.)

PW’s conclusion:
“Symptoms do not sound vestibular.”
On Examination:

- Borderline Unterberger to (L).
- Inconsistent 1st deg nystagmus to (R), fixation removed.
- Transient, asymptomatic head-shaking nystagmus to (R).
- Profound vibration-induced nystagmus to (R) at 60 & 100 Hz. Mildly dizzy.
- Milder hyperventilation-induced nystagmus (HIN) to (L). Mildly dizzy.

Conclusion:

- VIN to (R) suggests (L) peripheral vestibular hypofunction. in which case...
- HIN to (L) is irritative, consistent with a neural lesion.

Plan:

- Objective (audio)vestibular tests
Vestibular test results:

- 4 deg/sec 1\textsuperscript{st} deg nystagmus to (L), fixation removed. \(\text{(opposite to what I found)}\)
- 7 deg/sec head-shaking nystagmus to (R) \(\text{(as I found)}\)
- Vibration-Induced nystagmus to (R) \(\text{(as I found)}\)
Vibration-Induced Nystagmus

(my thanks to Andy Rutter)

60 Hz to RIGHT mastoid

100 Hz to RIGHT mastoid

60 Hz to LEFT mastoid

100 Hz to LEFT mastoid

7 deg/sec (R)-beating

9 deg/sec (R)-beating

6 deg/sec (R)-beating

12 deg/sec (R)-beating
Vestibular test results:

- 4 deg/sec 1\textsuperscript{st} deg nystagmus to (L), fixation removed.
- 7 deg/sec head-shaking nystagmus to (R).
- 6 – 12 deg/sec Vibration-Induced nystagmus to (R).
- 5 deg/sec Hyperventilation-Induced nystagmus to (L).
Vestibular test results (2):

Calorics:

<table>
<thead>
<tr>
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<th>(R)</th>
<th>(L)</th>
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<tbody>
<tr>
<td>Warm</td>
<td>(R)-beating 35 deg/sec</td>
<td>(L)-beating 5 deg/sec</td>
</tr>
<tr>
<td>Cool</td>
<td>(L)-beating 12 deg/sec</td>
<td>(R)-beating 2 deg/sec</td>
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76% (L) canal paresis; 37% (R) directional preponderance.

- The history was not suggestive of a vestibular disorder.
- (Paretic) VIN and (irritative) HIN were the ONLY abnormal tests on clinical examination.

“This is another case where the new VIN test has really proved its value”

Advised MRI for ? Vestibular Schwannoma
Hyperventilation-Induced Nystagmus

• Early literature suggested that dizziness induced by hyperventilation was psychogenic.  

  (Drachman and Hart, 1972)

• “Light-headedness” induced by hyperventilation may be a sign of psychogenic disturbance, but be wary if nystagmus is induced.

• Hyperventilation may unmask peripheral or central vestibular lesions, including cranio-cervical junction and cerebellar disorders. Nystagmus induced by hyperventilation is more likely to be indicative of a CPA lesion than any other single cause.

  (Choi et al, 2007)
Technique

• Patient seated.

• Fixation abolished with infra-red video-Frenzel’s goggles. (As with VIN, observe for (baseline) spontaneous / gaze-evoked nystagmus.)

• Asked to take 20 – 30 deep breaths (demonstrate).

• Examine for nystagmus with fixation removed.

• Ask if symptoms reproduced.
  
  (Symptoms with NO nystagmus may imply “Hyperventilation Syndrome”.)

• Patient may have to be told to stop!
Cautions...

- In known epileptics, hyperventilation *may* provoke a seizure.
Presumed mechanism of hyperventilation-induced nystagmus

- A drop in CO$_2$ levels causes a rise in CSF pH.
- Leads to a reduction in extra-cellular ionised Ca$^{2+}$
- Causes improved axonal conduction in a partially demyelinated nerve.
- Gives rise to excitatory nystagmus.

The Hyperventilation test is a useful addition to the standard clinical examination battery and should be performed when other tests are normal.

Irritative (usually) nystagmus provoked by hyperventilation may be indicative of a vestibular Schwannoma or of demyelination.
Conclusions: clinical value of Vibration-Induced Nystagmus test

- The VIN test is a rapid and fairly reliable method for detecting (and localising the side of) a unilateral vestibular lesion.
- With fixation removed, mastoid vibration induces a nystagmus that resembles that seen acutely, prior to compensation, with the slow phase generally towards the impaired side.
- Onset and offset are immediate with that of the stimulus.
- The test provokes little or no vertigo.
- Vibration-induced nystagmus persists over time, unlike spontaneous (or head-shaking) nystagmus.
- Therefore, VIN can be used to monitor recovery after (eg) vestibular neuritis, more easily than repeating calorics.
- VIN can be used in patients who cannot undergo calorics.