6° convegno
regionale
ORL e Audiologia

Le prove di stimolazione calorica

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20-21 Giugno 2014

Cerebellar infarction presenting isolated vertigo

Frequency and vascular topographical patterns

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Table 2 *Vestibular findings and imbalance in 24 patients with pseudo-vestibular neuritis associated with mPICA territory cerebellar infarction*

Findings	Patients
Head thrust test	Normal
SN	15
GEN	
Typical*	13
Unidirectional†	4
Gaze to only lesion side	7
Asymmetric pursuit‡	6
Asymmetric OKN	4
Canal paresis	None
Imbalance grade	
1	7
2	1
3	16
Direction	
Lesion side	17
Healthy side	7

Canal paresis was defined as side differences more than 22% at bithermal caloric stimulation.

British Society of Audiology

Recommended procedure

The caloric test

Date of publication: April 2010

Minor amendments: Sept 2010

Due for review: April 2015

3.2. Stimulus parameters

The stimulus parameters should be checked at least weekly or when a problem is suspected. Check that the air or water temperature at the outlet of the irrigator and the flow rate in free-flow are as indicted in Table 1 (Barber and Stockwell, 1980; Jacobson et al, 1993).

Table 1

Stimulus parameters[1]

	Temperature: 'cold'	Temperature: 'warm'	Flow rate
Water	30 °C ± 0.4 °C	44 °C ± 0.4 °C	250 ml ± 10 ml in 30 s
Air	24 °C ± 0.4 °C	50 °C ± 0.4 °C	8 l ± 0.4 l in 60 s

4.1. Referral and medication advice

Only patients with a written referral by medical staff should undergo this test. Advice about stopping medication (e.g. vestibular sedatives) should be given by the referring physician during the pre-test consultation. Ideally the physician should advise the patient to stop relevant medication at least 48 hours before the test. Patients should be advised not to consume alcohol for 48 hours before testing (Jacobson et al, 1993). Staff responsible for carrying out the caloric test should check that the patient has adhered to this advice.

Local protocol should be in place to establish whose has responsibility for ensuring appropriate information is provided to the patient.

4.2. Patient Travel

Check travel arrangements and ensure that patient is aware that they will be advised not to drive immediately following the test.

4.4. Contraindications

It is the responsibility of both the referring physician and audiologist to ensure that the patient is fit to undergo the test before it is conducted.

It is usually inappropriate to conduct the caloric test if any of the following are present:

- History of hypertension (uncontrolled, acute or de-compensated phase)
- History of cardiac problems (arrhythmias especially bradycardia or Stokes-Adams attack; acute or de-compensated phase) e.g. if someone has unstable angina, a recent myocardial infarction (within last 3-6 months) or is undergoing cardiac investigations[2]
- Psychotic/neurotic disorders (acute or de-compensated phase)
- Epilepsy (acute or de-compensated phase)
- Eye surgery (within the previous 3 months)
- Ear surgery (within the previous 6 months)

The next list shows the second level of contraindications / special precautions, which should be checked for compliance at the time of the appointment by the testier as well as by the referring physician and which also may contraindicate performance of the caloric test:

- Significant degree of space occupying wax
- Otitis externa
- Middle ear fluid/effusion
- Hypermobile[3] or atrophic tympanic membrane - care should be taken for severe hyper-mobility and a second medical opinion obtained

- Tympanic membrane perforation (may be suitable for air calorics)
- Patients with mastoid cavities may be considered for air calorics, but interpretation should be carried out with caution.

Staff performing the test should be aware of these contraindications and the specific relevant specialist should be contacted for advice on individual patients.

Appendix C. The caloric mono-thermal 'screening' test

2. The mono-thermal caloric asymmetry (MCA) is computed from the results of the two tests at the first temperature, thus: 100 x (R-L) / (R+L).

MCA criterion of < 15 %

Appendix D. Testing by direct observation

Recommended procedure for testing by direct observation:

1. Patient preparation as per Section 4 and check for spontaneous nystagmus as per Section 6.2

2. Use longer irrigation time with water stimulus (40 s).

3. Time the duration of the nystagmus. Timing starts at the beginning of the irrigation and ends at the last observable beat of the induced nystagmus (the 'end-point'). Note the direction of the nystagmus and any significant irregularity of the response.

4. At the end-point, switch the room lights off. Nystagmus is now likely to recur as a result of the removal of visual suppression. Observe this nystagmus using an infrared viewer or Frenzel glasses and time its duration (still continuing from start of irrigation). Observe for the end point.

5. The other irrigations should be carried out in a similar fashion.

6. Canal paresis and directional preponderance should be calculated using the expressions given in the previous Section 3.4, where WR, WL, CR and CL are now the durations of the response obtained from Section 9.4 (total time from start of irrigation to last observable beat) and compared to normal values. It is recommended that local normative data should be used as published literature on normal values by duration is scanty and that the limits of normal function should be ± 1.96 standard deviations from the mean.

7. Visual fixation index: A qualitative assessment of the effect of visual fixation can be made by waiting until the response ceases to be observable with visual fixation. Frenzel glasses or an infrared viewer can then be used in a darkened room to reduce or remove fixation. Watch for nystagmus to reappear in the absence of fixation.

$$\text{Visual fixation index (\%)} = \frac{\text{Duration with fixation}}{\text{Duration without fixation}} \times 100$$

Canal paresis (%) is given by (Jongkees et al, 1962):

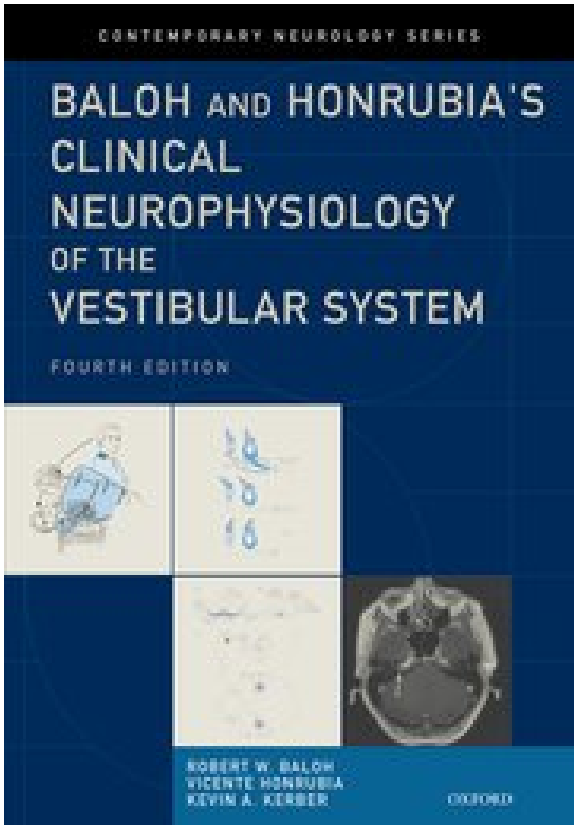
$$\frac{(WR + CR) - (WL + CL)}{WR + WL + CR + CL} \times 100$$

Directional preponderance (%) is given by:

$$\frac{(WR + CL) - (WL + CR)}{WR + WL + CR + CL} \times 100$$

An arithmetic correction for spontaneous nystagmus (see Section 6) should not be used when calculating these statistics, as the precise effect of spontaneous nystagmus on the caloric response is not understood. However, these statistics, and the overall caloric response pattern, should be interpreted in light of the direction and strength of any spontaneous nystagmus present.

…This is not to say that a central lesion cannot cause a vestibular paresis, bur to simply say that a central lesion is much less likely …



peripheral vestibular disease. For the central vestibular system to be the source, the lesion would need to involve the eighth nerve root entry zone. In animal studies focal lesions in different vestibular nuclei did not produce a vestibular paresis. This is not to say that a central lesion cannot cause a vestibular paresis, but to simply say that a central lesion is much less likely. A recent study reported that 43% (10 out of 23) of patients with stroke (defined as a relevant lesion with restricted diffusion on magnetic resonance imaging [MRI]) causing acute vertigo who underwent caloric testing had a vestibular paresis of >25%.[34] In 22% (5 out of 23) of these stroke patients the vestibular paresis was severe (>75%). Because peripheral causes of vestibular paresis are much more common than central causes, the likelihood of a central cause is low particularly when there are no other central ocular motor abnormalities (e.g., bi-directional gaze evoked nystagmus) or other brainstem signs.

A directional preponderance on caloric testing occurs with peripheral end-organ and eighth nerve lesions and with CNS lesions (from brain stem to cortex).[30] It indicates an imbalance in the vestibular system and is usually associated with spontaneous nystagmus: the velocity of the slow components of the spontaneous nystagmus adds to that of the caloric-induced nystagmus in the same direction and subtracts from that of the caloric-induced nystagmus in the opposite direction.[35] Occasionally, a directional preponderance will occur in patients without spontaneous nystagmus; most of these patients have peripheral lesions, although about 5% have central lesions.[36]

The need to distinguish between end-organ and eighth nerve lesions is a common clinical problem. Partial lesions of the eighth nerve should not, in theory, affect the duration of

Vertigo and Imbalance: Clinical Neurophysiology of the Vestibular System
Handbook of Clinical Neurophysiology, Vol. 9
S.D.Z. Eggers and D.S. Zee (Vol. Eds.)

CHAPTER 10

Caloric testing: background, technique, and interpretation

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testing.

Minimal ice water caloric testing is also suitable to distinguish between a peripheral and a central origin of spontaneous nystagmus and to monitor restoration of vestibular tone on the deafferented side in peripheral vestibular lesions. In the acute phase of a complete peripheral vestibular lesion the vestibular tone on the deafferented side is lost (Sirkin et al., 1984). The spontaneous nystagmus is due to unopposed tonic activity on the intact side. An ice water caloric stimulus induces a reduction of afferent vestibular activity on the irrigated side, mimicking a transient loss or severe reduction of function

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frontiers in BEHAVIORAL NEUROSCIENCE

Front Behav Neurosci. 2014; 8: 51.
Published online Feb 19, 2014. doi: 10.3389/fnbeh.2014.00051
PMCID: PMC3928537

Purchase decision-making is modulated by vestibular stimulation

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Abstract

Go to:

Purchases are driven by consumers' product preferences and price considerations. Using caloric vestibular stimulation (CVS), we investigated the role of vestibular-affective circuits in purchase decision-making. CVS is an effective noninvasive brain stimulation method, which activates vestibular and overlapping emotional circuits (e.g., the insular cortex and the anterior cingulate cortex (ACC)). Subjects were exposed to CVS and sham stimulation while they performed two purchase decision-making tasks. In Experiment 1