Review Article

Special investigations used in the assessment of the dizzy patient

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Abstract
Audio-vestibular assessment is important when assessing the dizzy patient. Baseline vestibular function may be established and used to determine if a peripheral, central or mixed pattern of vestibular dysfunction is present. A detailed history is vital, but in some cases this may be vague and audio-vestibular testing assists in formulating a diagnosis. Test results are then used for guiding the management of the dizzy patient.

Keywords
Audio-vestibular tests, balance, dizziness.

Introduction
There are a number of diagnostic tests available for assessing the dizzy patient. Some, such as auditory brainstem response testing, have been largely superseded by the use of imaging. Others, such as posturography, are arguably more useful in guiding rehabilitation programmes. The tests that will be discussed in this article are currently favoured in many dizzy assessment clinics.

Audiological examination and testing
Otoscopy and tympanometry are primarily performed to ensure that there are no abnormalities that may affect stimulus presentation prior to vestibular testing, as specified in the BSA revised procedure for caloric testing (2009).1

Pure tone audiometry is important as some patients with balance disorders have related hearing loss. Low frequency sensorineural loss is associated with early Meniere’s disease, and an asymmetrical sensorineural hearing loss found in patients who have suffered from an episode of labyrinthitis or may be harbouring a vestibular schwannoma.

Oculomotor tests
The vestibular and visual systems work together to enable gaze stability. Dizziness may be due to altered visual function and oculomotor testing is used to assess the saccade and smooth pursuit pathways, to assess optokinetic nystagmus, as well as the vestibular-ocular reflex. These eye movements can be recorded using different detecting systems.2

Eye detection systems
Electronystagmography (ENG): works by quantifying the corneo-retinal potential, a small voltage between the front and back of the eye. ENG consists of an isolated instrumentation amplifier coupled to a chart recorder or computer. ENG recordings are, however, susceptible to electrical noise and are invasive with the application of the electrodes. Often, re-application is required due to electrode detachment causing drift in the chart recordings. For these reasons, many centres now use videonystagmography.

Videonystagmography (VNG): These devices usually combine a pupil-tracking card with a small video camera set in a swim-goggle type frame. The card detects the pupil as the darkest part of the eye, so a pre-test requirement is for patients to remove eye make-up, particularly mascara.

Infrared recording: This generally works by quantifying the difference detected by a phototransistor pair of sensors of the amount of infrared light reflected by the sclera. Although infrared allows subjects to be assessed in darkness, and suffers with less noise than ENG, baseline eye position is important (i.e. ± 10 deg from centre).

Scleral coil recording: The scleral magnetic eye coil is attached to a contact lens and placed on the subject’s eye. A thin wire attached to the lens allows accurate recording of eye movements. This is however uncomfortable and hence seldom used in the clinical testing.

Oculomotor testing is performed in a room with reduced illumination in order to optimise the definition of the test stimulus and essential when testing with ENG. Altered lighting conditions will affect the corneo-retinal
potential and calibration of the system. Patients must stop using vestibular system sedatives for 48 hours prior to testing as they could affect interpretation of results, by reducing gain. Eye muscle weakness and reduced visual acuity may also affect test results.

Saccades
Saccades are used to bring the eye rapidly from one point to another. Saccades to an unexpected stimulus normally require approximately 200 milliseconds (ms) to initiate and last for 200ms. Saccadic velocity is approximately proportional to saccadic amplitude for movement, between 5 and 20 degrees. The lower limit of velocity is 350 deg/sec; the upper limit 750 deg/sec. Saccadic velocity cannot be altered voluntarily and is not affected substantially by age or gender.

Saccades are either voluntary or involuntary and are measured for velocity, latency and accuracy.

Voluntary saccades are voluntary or “willed” eye movements in response to flashing or moving stimuli, or an unseen remembered target perceived on the peripheral retina. Visually guided voluntary saccades are initiated principally by the frontal eye fields with input from the “parietal and prefrontal cortex”. Involuntary saccades are non-visually guided eye movements that are initiated by direct retinal input to the superior colliculus. The fast eye movement component of this involuntary eye movement is called nystagmus (to be discussed with optokinetic nystagmus).

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Saccade production is described as shown in figure 1. The accuracy of eye movements that occurs once the saccade has been performed is regulated by a biofeedback loop involving the cerebellum (as discussed below with respect to the pursuit system).

When performing saccade testing, patients are asked to keep their head steady and move their eyes as quickly as they can to a target moving in a random saccadic way (moving between 20 degrees from primary position, horizontally or vertically). A typical trace reading is shown in figure 2.

Normal values for saccades

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Velocity</td>
<td>Between 350 and 750 degrees per second</td>
</tr>
<tr>
<td>Accuracy</td>
<td>100% +/- 2 degrees</td>
</tr>
<tr>
<td>Latency</td>
<td>200-400ms</td>
</tr>
</tbody>
</table>

Abnormal saccades
Production of saccades may be abnormal due to a combination of velocity, accuracy or latency. Any part of the pathway, as shown in the flow diagram, may be affected.

Slow saccades are often due to drug ingestion (very common) or drowsiness / fatigue (common). Oculomotor weakness due to cranial nerve palsies or myopathy, may also cause slow saccades. Fast saccades are either optic flutter or opsiclonus; these are back to back saccades, in opposite directions, without an intersaccadic interval. Flutter is seen in normal subjects but also can be seen with cerebellar or
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brainstem disorders. Opsoclonus differs from flutter in that it can be seen in vertical and torsional eye movements.

Inaccurate saccades may be due to undershooting or overshooting the visual target. An undershoot is a normal variant although may be due to peripheral visual difficulties or if occurring more than 50% of the time may indicate abnormality of the central nervous system affecting the superior colliculus. Overshooting is suggestive of a cerebellar dysfunction sign.

Square Wave Jerks are inappropriate saccades that take the eye off the target, followed by a nearly normal intersaccadic interval (approximately 200 ms). A corrective saccade then brings the eye back to the target. Square wave jerks are seen in normal subjects but may occur as a result of central pathology; they are non-specific. Macrosaccadic oscillation is a disorder where the eye makes hypermetric saccades back and forth about a target. This is usually a sign of cerebellar disorder.

Microsaccadic oscillation denotes a tiny (0.2 deg) back-back saccadic oscillation. It is generally benign although it can obscure vision. It is thought to help with maintaining a visual image on the fovea. Microsaccadic oscillations are difficult to record with ENG or VNG because it is too small and too fast. It is best seen with the ophthalmoscope.

Saccades that take time to produce (as above) are often due to visual difficulties such as having cataracts or more rarely due to basal ganglia abnormalities. Patients can sometimes anticipate saccades and so have reduced latencies. Asymmetric saccades indicate central system lesions in the superior colliculus, parietal cortex or occipital cortex.

Smooth pursuit

The smooth pursuit system is used to stabilise a moving target on the fovea of velocities up to 60 degrees per second or 1 Hz. Above this, the corrective saccade system is used together with the vestibular-ocular reflex. The neural circuitry underlying smooth pursuit is subject to debate, however, the first step towards the initiation of pursuit is to see a moving target.

Flow diagram
target. Various cortical areas are involved in pursuit initiation with cerebellar projections causing eye movement. Corrective eye positioning also involves the cerebellum.

The below shows the initial pursuit pathway (red arrows) and the corrective biofeedback pathway (green)

Patients are asked to keep their head steady whilst tracking a sinusoidal moving target (horizontally or vertically)

**Normal values for smooth pursuit**

*Gain:* For velocities up to 30 deg/s and 0.3Hz, gain should be close to 1 (eye velocity/target velocity); > 0.8 gain is deemed normal

*Morphology:* Should be smooth without saccadic interruptions

**Abnormalities of pursuit**

Any aspect of the pathway can be affected; it is hence a non specific test. Reduced gain (0.2-0.8) is often due to poor attention, reduced visual acuity, central system drugs (e.g. vestibular sedatives) or alcohol ingestion. Absent gain: (0-0.2) indicates a central nervous system disturbance, or reduced visual acuity (poor central vision). Poor attention, reduced visual acuity, drug and alcohol ingestion may cause poor morphology.

The top trace is normal, the middle trace shows poor attention and the bottom trace is of a central system abnormality

Asymmetric abnormality may highlight an eye muscle weakness eg congenital nystagmus, or latent nystagmus with amblyopia. It may be due to central system dysfunction in the parietal or frontal lobes.

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**Optokinetic nystagmus**

Nystagmus means 'head nod' in Greek. Nystagmus has 2 components, a slow phase and a fast phase. The direction of nystagmus is defined from the direction of the fast phase. The above diagram, representing chart recording of eye movement, shows the eyes drifting to the right (upwards on the chart - slow phase) followed by a sharp movement to the left (downwards on the chart – fast phase); this is left beating nystagmus.

With OKN, nystagmus is produced by slow tracking of movement within the visual field followed by rapid eye movement to relocate gaze onto new targets entering the field. When testing, OKN can be produced by the projection of moving stripes on a screen.

OKN is considered to be a primitive form of smooth pursuit involving the whole retina instead of the fovea alone. The eye tracking motion (slow phase of OKN) is interrupted by the fast corrective, involuntary saccade. Active OKN is smooth pursuit. Passive OKN involves movement of the visual field and reflex tracking of the eye with corrective saccade; it is extrafoveal. OKN is more robust than pursuit in that it is not affected as much by inattention and central vestibular sedatives due to combined fovea and extrafoveal responses. Below is the flow diagram generating the passive response, showing that the vestibular nuclei are involved.
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The patient is asked to look passively ahead, keeping their head steady, whilst the OKN stimuli (usually black and white stripes) move horizontally or vertically across the visual field.

Normal values for OKN
A symmetrical response with gain of >0.75 is considered normal.

Abnormal OKN
Symmetrical reduced gain usually is due to inattention, visual system disorders or congenital nystagmus.

Central abnormalities indicate pursuit system disorder (i.e. cerebellar floccular lesion or due to medication).

Fast phase disorder is of the saccade producing system such as progressive supranuclear palsy. With congenital nystagmus there is often a failure to reverse optokinetic nystagmus ith reversal of the target movement.

Asymmetrical responses indicate a complete unilateral peripheral vestibular disorder, and absence is linked with bilateral vestibular failure or a central vestibular abnormality.

Vestibular ocular reflex
The smooth pursuit system is used for tracking slow moving targets. Movement of the head is required to track faster targets and the vestibular ocular reflex is used to do this.

The semi-circular canals are functionally paired (anterior ipsilateral to superior contralateral, and the two lateral) and movement of the head causes at least one pair to be stimulated.

The vestibulo-ocular reflex. As a result of head rotation, endolymph flow within the semicircular canals causes movement of the cupulae within the ampullae of the lateral semicircular canals and relative shearing of the underlying stereocilia. Neural impulses increase on the right and decrease on the left. Neural connections to the IIIrd and VIth cranial nuclei result in contraction of the left lateral rectus and right medial rectus to stabilize gaze.
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The eyes move in the orbit until they reach physiological end point when a corrective saccade brings the eyes back to primary position, creating a nystagmus beat; the VOR provides the slow phase whereas the saccade provides the fast phase of the beat.

**Flow pathway**

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**Gaze testing/determining presence of spontaneous nystagmus**

The patient is asked to keep their head steady then maintain their eye position in the primary position and in the lateral eye positions (30 degrees from centre) with and without fixation on a visual target. Normally, the eyes are maintained in these positions without any deviation.

Nystagmus that is observed in the absence of head or body motion is called spontaneous nystagmus and that of 4 degrees per second or more is significant. That due to a peripheral vestibular dysfunction usually is suppressed with visual fixation.

Nystagmus that is present with fixation is due to: medication e.g. sedatives, alcohol or recreational drugs or ocular muscle fatigue. If not due to these, it usually indicates a central vestibular disorder.

Following eye movement examination, testing of the vestibular system by caloric, rotational chair and VEMP testing takes place.

**Caloric testing**

The caloric test assesses the VOR of each horizontal canal using water/air aural irrigation as stimuli.

The test is performed with the patient lying down on a couch inclined to enable the head to be at 30 degrees to horizontal. Irrigating the ears with water (44/30 deg C) or air (24/50 deg C) creates a thermal gradient in the middle ear cavity that, because of its proximity to the middle ear, causes endolymphatic flow in the horizontal canal by convection. Cold air/water causes endolymph flow in the opposite direction to warm air/water creating decreased/increased stimulation respectively of the vestibular nerve of the ear being irrigated. A total of 4 irrigations are performed.

The resultant VOR is monitored using the eye recording system (usually VNG). The maximum slow phase is measured then the patient is asked to view a fixation light in order to assess the ability to suppress the VOR.

Responses to right ear irrigation are compared to the left to provide a canal paresis comparison. Responses of left nystagmus (right cold/left warm) to right nystagmus (right warm/left cold) are compared to provide a directional preponderance calculation.

**Normal values**

- Individual responses >5 and <60 degrees/second
- Total reactivity (sum all 4 responses) > than 20 degrees/second
- Canal paresis: response comparison < 20%
- Directional preponderance: response comparison < 20%
- Suppression of the responses by fixation – is present
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**Flow pathway**

**Abnormal results**
- Canal paresis ≥ 20%: This indicates a unilateral peripheral vestibular dysfunction
- Directional preponderance ≥ 20%: This indicates either a peripheral or central vestibular system dysfunction
- Total reactivity (sum all 4 responses) ≤ 20 degrees/second: A bilateral peripheral vestibular hypofunction is indicated (failure is confirmed using ice cold water irrigation)
- Abnormal suppression: This is due to central vestibular dysfunction or a visual impairment

**Rotatory chairs**
Caloric tests are important to perform to in order to establish individual horizontal canal function. However, the caloric stimulus is non-physiologic at <0.01Hz. Rotary tests produce a stimulus of 0.1-1.2Hz which is more representative of normal body motion and can be used when patients have chronic ear infections or refuse to perform caloric tests. However, rotatory tests assess both inner ear function simultaneously and so it is more difficult to discern unilateral dysfunction.

Rotatory chair testing stimulates the vestibular system similarly to caloric testing. Phase, gain and symmetry of the responses are measured and abnormality usually indicates either unilateral or bilateral vestibular dysfunction.

**Flow pathway**
Vemps
The vestibular organs not only control eye movements; they are involved in postural control via the vestibular spinal reflex.

Caloric testing involves stimulating the horizontal canal and this is innervated by the superior branch of the vestibular nerve. The inferior branch innervates the saccule and posterior semicircular canal. Loud sounds stimulate the saccule leading to altered stimulation of the antigravity muscles of the neck and spine. Increased electrical activity can be recorded - the vestibular evoked myogenic potential (VEMP). A loud sound not only stimulates our hearing but also causes (via the otolith organs) contraction of muscles to look in the direction of the stimulus. The cervical (or C) VEMP is most commonly measured (using the sternoclenomastoid muscle – SCM), although the ocular (or O) VEMP is being assessed (which may give information of utricle function).

Flow pathway

The patient lies down with their neck either elevated, or sitting with their head turned to one side. This is to provide a resting muscle tension to monitor the VEMP against.

An electrode is placed on the SCM belly, clavicle and the forehead (earth).

Auditory stimuli (usually 500Hz tones) are presented into the ear and the VEMP recorded.

The P1 N1 complex is measured at different stimulus intensities. Measurements include: Amplitude of response, threshold at which it is present or the latency of the response.

It is important to establish normative data.

Normal values
This depends on the equipment set-up but a lowered threshold/increased amplitude indicates superior canal dehiscence or labyrinthine fistula. A prolonged latency indicates central vestibular system dysfunction or endolymphatic hydrops.

Summary
Audio-vestibular testing is important to perform for assessing and managing the dizzy patient.

Conflict of Interest
All authors have no conflict of interest to declare. No extraneous funding was obtained.

References
1. Recommended procedure: the caloric test – British Society of Audiology 2010
Approved by ENT-UK for Continuing Professional Development

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1. Pure tone audiometry is important to perform on patients having balance disorder. Which ONE of the following conditions is not usually linked to hearing loss?
   a. Vestibular schwannoma
   b. Menieres disease
   c. Benign paroxysmal positional vertigo
   d. Labyrinthitis

2. Videonystagmography (VNG) is largely used as the method for recording eye-movements when assessing the dizzy patient. Which of the following statements is not generally true for VNG?
   a. Wearing make-up doesn’t affect testing
   b. Electrical noise doesn’t affect testing
   c. A stable image is maintained throughout recording
   d. There is a need to recalibrate with changing light conditions

3. Abnormalities with VNG testing are usually associated with central or peripheral vestibular system dysfunction. However, which of the following may also cause abnormal results on testing?
   a. Alcohol consumption
   b. Taking vestibular sedatives
   c. Having ear wax
   d. Eye muscle weakness
   e. Reduced visual acuity
   f. Patient lethargy

4. Which of the following VNG tests when abnormal is most associated with detecting peripheral vestibular dysfunction?
   a. Saccades
   b. Smooth pursuit
   c. Optokinetic nystagmus
   d. Gaze testing/ assessing spontaneous nystagmus

5. Which of the following is not true of caloric testing?
   a. Test stimuli are physiologic
   b. Test stimuli can be air or water
   c. Information of the function of each horizontal semicircular canal is obtained
   d. Abnormal canal paresis calculation indicates unilateral peripheral dysfunction
   e. Abnormal directional preponderance calculation indicates central and/ or peripheral vestibular system dysfunction

6. Which of the following is not true of VEMP testing?
   a. Patients need to have normal hearing for testing
   b. It assesses otolith function
   c. It is used to diagnose superior canal dehiscence
   d. It can identify central vestibular system disorder

Multiple Choice Questions:

1. Bilateral vestibular failure may have abnormalities with which of the below tests?
   a. Pure tone audiometry
   b. Saccades
   c. Smooth pursuit
   d. Optokinetic nystagmus
   e. Gaze testing/ assessing spontaneous nystagmus
   f. Calorics
   g. Rotatory tests
   h. VEMPS

2. Patients with labyrinthitis may have abnormalities which of the below tests?
   a. Pure tone audiometry
   b. Saccades
   c. Smooth pursuit
   d. Optokinetic nystagmus
   e. Gaze testing/ assessing spontaneous nystagmus
   f. Calorics
   g. Rotatory tests
   h. VEMP