Eye Movements in Vesibular Function and Dysfunction: A Brief Review

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ABSTRACT

It is well known that there is a very close relationship between the vestibular system of the inner ear and eye movements, however symptomatic outcomes of this relationship are not common in general eye clinics. Stimulation of the semicircular canals by rotation or caloric testing results in vestibular nystagmus and this can be used clinically to assist in the diagnosis of peripheral and organ vestibular disorders. Testing of otolith dysfunction, however, has been less straightforward. It has recently been shown that eye movements can be elicited by otolithic stimuli, delivered either as air or bone conducted sound. These eye movements are small, but reliable, and can assist in the diagnosis of vestibular disease or dysfunction.

Keywords: eye movements, otolith dysfunction, bone conducted sound

INTRODUCTION

The function of vestibular eye movements is to maintain a steady image on the retina despite both angular (rotational) and linear translations of the head[1,2]. These functions are controlled by the short neural pathways from the vestibular system of the inner ear via the brainstem to the extraocular muscles, with close relationships with the cerebellum and other neural areas that control ocular motility.

The peripheral sensory organs for vestibular eye movements are the two membranous labyrinths that lie within the temporal bone of each inner ear. A labyrinth with its neural innervation is shown schematically in Figure 1[3]. Each labyrinth contains three semicircular canals that are more or less orthogonal with respect to each other and sense head rotation; and the maculae of the utricle and saccule (the otoliths) that sense linear motion and static changes in gravitational forces. The labyrinth also contains the cochlea, the primary auditory sensory organ. This short, fast[3] neuronal arc underlies the fast vestibulo-ocular response.[2]

The sensory receptors for rotational acceleration, the cristae, are located at the base of each semicircular canal in an enlarged area, the ampulla. Each ampulla consists of a gelatinous sail like structure (the cupula) in which are embedded in the crista's hair cells. The cupula bends in response to movement of the endolymphatic fluid within the semicircular canals, which in turn exerts force on the cilia of hair cells. These hair cells contain many small processes (stereocilia) and one larger kinocilium. Bending of the cilia towards the kinocilium causes the cell to depolarise, increasing the firing rate of the afferent fibre, whereas bending of the cilia away from the kinocilium causes hyperpolarisation resulting in a decreased firing rate.

The maculae of the otoliths, the sensory receptors for linear acceleration and static changes in gravity with respect to the head, are located in two vestibular sacs, the utricle and saccule. Each macula consists of a gelatinous mass (the otolithic membrane), on the upper surface of which are embedded crystals of calcium carbonate (otoconia) and the cilia of hair cell receptors (stereocilia and a kinocilium) that project into the under surface of the otolithic membrane. When the head is in the upright position, this tissue is located on the floor of the utricle and on the wall of the saccule. The utricle is therefore oriented to respond best to lateral or fore-aft tilts and side to side translations of the head, whilst the saccule responds best to up-down translations of the head[1]. Motion or changes in gravity cause shearing movements of the otoconial layer that bend the hair cells, causing polarization and hyperpolarisation in a manner similar to that in the semicircular canals.

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Afferents from the vestibular apparatus pass to Scarpa's ganglion, located within the internal auditory meatus and then as the vestibular nerve to synapse in the vestibular nuclei. Neurons in the vestibular nuclei project to the cerebellum which project back to the vestibular nuclei, controlling neural transmissions from the receptor in the periphery to the oculomotor nuclei. The organisation of this sensory-motor system is such that neurons from each canal excite and inhibit complementary muscles in each eye (See Table 1).

This tight linkage between semicircular canals and the eye muscles was clearly shown by the pioneering work of Cohen[1] and Suzuki[4] who delivered isolated stimulation of the nerve to each semicircular canal in cats and monkeys and recorded the direction of eye movements that were produced. This linkage can assist in the diagnosis of lesions of the vestibulocochlear complex (Figure 1).

Table 1. Relationship between each semicircular canal and the extraocular muscles[4].

Canal	Excites	Inhibits
Horizontal	ipsilateral medial rectus	ipsilateral lateral rectus
	contralateral lateral rectus	contralateral medial rectus
Posterior	ipsilateral superior oblique	ipsilateral inferior oblique
	contralateral inferior rectus	contralateral superior oblique
Anterior	ipsilateral superior rectus	ipsilateral eye inferior rectus
	contralateral inferior oblique	contralateral superior oblique

CHARACTERISTICS OF VESTIBULAR EYE MOVEMENTS

Vestibulo-Ocular Reflex (VOR)

The function of this reflex is to stabilise the image on the retina during rotations of the head. This reflex depends on the semicircular canals that detect head movements and initiate a rapid compensatory movement in the opposite direction with a latency of less than 16 msec. The VOR is more efficient in yaw (head rotates horizontally) and pitch (head rotates vertically) than in roll movements (ear to shoulder), as in these roll movements the image is not moved off the fovea[1]. Sustained angular rotations of the head produce vestibular

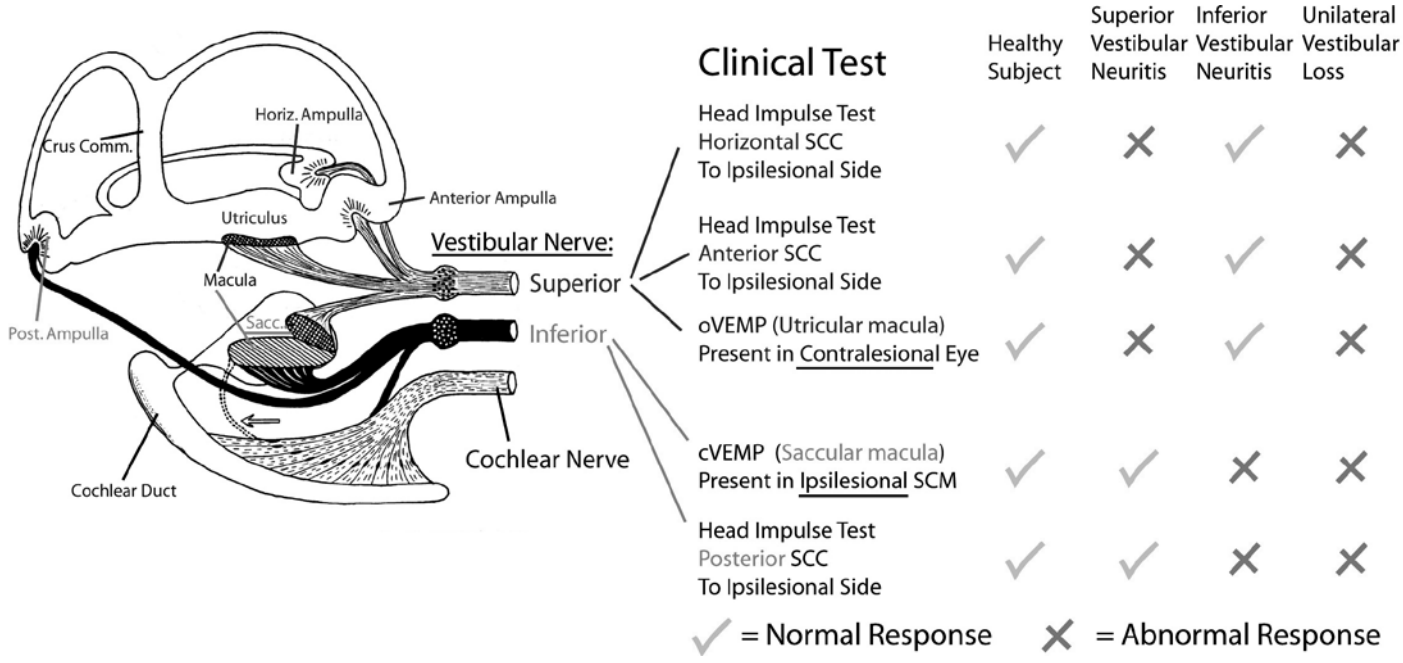

Figure 1. Clinical testing of vestibular and auditory dysfunction can provide identification of specific lesions of the vestibulocochlear complex. Eye movements stimulated by bone conducted sound (identified in this figure as "oVEMP") are absent or abnormal with lesions of the contralateral superior vestibular nerve. (Modified from de Burlet,26).

oVEMP (Vestibular Induced Muscle Potential from the (extra) ocular muscles)

cVEMP (Vestibular Induced Muscle Potential from the cervical muscles)

SCC (Semi Circular Canals)

nystagmus, with the fast phase beating to the same side as the direction of the head.

Translational movements of the head also initiate a compensatory horizontal or vertical eye movement if the object of regard is near to the subject. These movements are initiated by the otolith system (translational, or linear VOR).

Ocular Counter-Roll (OCR)

When the head makes a static tilt to the side, the eyes respond by making a small static conjugate torsional movement with the 12 o'clock meridian of the eye rotating in the opposite direction to the tilt. This reflex is otolith induced. Although this reflex may have originated to maintain a horizontal horizon in vertebrates with laterally placed eyes, in humans it only compensates for approximately 10% of the tilt[5].

Cervico-Ocular Reflex

A rotation of the torso about the vertical (Z) axis, relative to a stationary head produces very small amplitude horizontal nystagmus and a conjugate horizontal eye movement opposite to the movement of the torso. A forward inclination of the torso with respect to the head produces vertical eye movements. Ott[6] considers that a sideways tilt of the torso in the absence of utricular input does not induce eye torsion, although others[7-11] have suggested that there may be some effect of proprioception of the sterno-cleido-mastoid neck muscles on ocular counter-roll when otolith function is reduced or absent.

Skew Deviation

When animals with laterally directed eyes roll tilt the head to one side there is a compensatory movement whereby the lower eye moves up, and the higher eye moves down (skew deviation). In humans a similar response may occur following lesions of the midbrain where the eye on the same side as the head tilt moves up and the contralateral eye moves downward. There is evidence that very small skew deviations can occur on head tilt in normal subjects[12-16]

CLINICAL IMPLICATIONS

This close relationship between the vestibular system and eye movements may be used in assessing the functional status of lateral eye movements in babies (by observing the eyes as the infant is rocked or rotated) or in suspected supranuclear conditions such as Progressive Supranuclear Palsy (where eye movements may only be elicited in response to head movements) but despite the complex and close anatomical relationships between the vestibular and oculomotor systems, clinical manifestations of vestibular disturbance are not commonly recognised in orthoptic practice. However, subtle disturbances of vision that are not explicable by routine testing should be suspected as being possibly of vestibular origin, especially if there is a history of head injury or other neurological signs. A reduction of visual acuity by three lines or more with the head moving at about two to three cycles per second, as opposed to acuity when the head is steady, indicates vestibular disturbance that should be further evaluated[17], possibly by caloric testing or rotational tests that induce vestibular nystagmus.

While most clinical tests for vestibular dysfunction are based on stimulation of the semicircular canals, testing otolith function has been more difficult, especially for ill or bedridden patients. It is now becoming apparent that evaluation of small eye movements in response to otolithic stimuli can also be used to assist in the diagnosis of neurological disease. Suzuki, Tokumasu and Goto[4] showed that electrical stimulation of one utricular nerve in the cat produced eye movements that were mostly rotational (away from the side of stimulation) vertically divergent (with the ipsilateral eye moving upwards) and with small conjugate horizontal movements (towards the contralateral side).

As noted above, the otoliths respond to linear acceleration, and one form of linear acceleration is produced by bone conducted vibration. This stimulus generates many rapid changes in linear acceleration at the mastoid which is an optimal stimulus for one class of primary otolithic neurons. Physiological evidence from guinea pigs shows that irregular primary otolithic stimuli are selectively activated by such vibration, while few semicircular canal neurons are activated by this vibration at physiological levels.

It is now recognised that sound, delivered either through headphones (air conducted, or AC) or through vibration of the skull (bone conducted, or BC) in humans can evoke short latency vestibular induced extraocular muscle potentials (oVEMPs), that peak at approximately 10 ms[7,18-21]. These negative surface potentials are the pre-cursers to the actual rotation of the eye and are probably due to the barrage of neural activity to the inferior rectus and inferior oblique muscles recorded from surface electrodes usually placed beneath each eye. However, there have been fewer reports on the direct measurement of the eye movements themselves. Jombik & Bahyl[20], have recorded sound evoked responses based on electro-oculograms (EOG), although Rosengren[19] has suggested that these may represent synchronous activity of all extraocular muscles. Halmagyi[22] has commented that surface EOG recordings are likely to be "dominated by oVEMP responses and should not be interpreted as eye movements".

Studies using high precision scleral search coils have confirmed the presence of these very small, but predictable eye movements following air and bone conducted sound. Aw et al[23] used search coils to measure eye movements evoked by AC sound in normal healthy subjects. These were primarily upwards, to the side of the stimulated ear and with horizontal rotation away from the side of stimulation. Todd et al[11] also used scleral search coils to demonstrate different eye

movement responses to both AC and BC stimulation. With air conduction (using headphones) these movements were usually upwards and towards the contralateral side, while bone conducted vibrations, delivered to the mastoid process using a clinical bone conductor on the mastoid (Radioear B71), typically produced downward movement towards the ipsilateral side. Torsional rotation was away from the side of stimulation in both conditions. These responses suggest that the ipsilateral superior oblique, and its antagonist, the contralateral inferior rectus, are primarily responsible for these movements. The amplitudes of the vertical eye movements averaged +2.6 mdeg (for AC sound) with a mean latency of 16.6 ms. For bone conducted sound amplitudes ranged from +3.0 to -19.3 mdeg with a mean of -7.4 mdeg.

Our recent studies have used bone conducted vibration at 500Hz by a B71 bone stimulator on the mastoids of a ten of healthy subjects and have recorded eye movements using new very fast fire wire cameras with a high enough resolution to detect very small eye rotations. The results have confirmed that bone conducted stimulation to the mastoid generates predominantly downward eye movements.

Iwasaki et al[21,24-25] delivered bone conduced vibration to the forehead at the hairline (Fz) and recorded short latency surface potentials from beneath both eyes (oVEMPs) in nine normal, healthy subjects and in ten subjects with vestibular or hearing abnormalities. Eight of these patients had had total unilateral vestibular deafferentation (uVD) and hearing loss two or more years after excision of a vestibular schwannoma. One had bilateral vestibular loss but preserved hearing due to gentamicin vestibulotoxicity, and one had profound hearing loss from congenital Rubella but normal vestibular function. Healthy subjects showed symmetric, short latency negative (excitatory) potentials from beneath both eyes, however these potentials were absent from the eye contralateral to the unilateral vestibular deafferentation in all eight uVD subjects. They were bilaterally absent in the patient with gentamicin vestibulotoxicity but were present in the profoundly deaf patient with preserved vestibular function. These findings clearly demonstrate the vestibular origin of these induced eye movements. This has now been replicated in a further 11 UVD subjects[25]. In light of the fact that the stimuli generate linear accelerations (otolithic stimuli) these responses appear to be a new way of using eye movements to measure otolithic vestibular function.

CONCLUSION

It is now evident that eye movements in response to sound, either air or bone conducted, can be used in the diagnosis of vestibular (otolith) dysfunction, and is a growing area of research that has applications in clinical practice in neuro-ophthalmology and vision sciences.

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