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BLURRED VISION WITH HEAD MOVEMENTS — TESTING THE EFFICACY OF THE VESTIBULO-OCULAR REFLEX

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Abstract

The vestibulo-ocular reflex (VOR) maintains clear vision via a stable retinal image during head movements. Disorders of the labyrinth, cerebellum or brainstem may reduce the efficiency of this mechanism and patients may present with a main complaint of blurred vision, viz oscillopsia. The clinical test of visual acuity assessment during head movement is outlined and illustrated with a Case Presentation of a subject with a defective VOR. The neuroanatomical substrate is briefly discussed.

Key words: *Blurred vision, vestibular, visual acuity, VOR.*

INTRODUCTION

An interesting clinical dilemma arises on occasion when a patient presents complaining of blurred vision. The reasoning process that ensues is one of a process of elimination and then confirmation. Common aetiologies encountered include: static or dynamic errors of refraction, prismatic effects, sensory media defects such as corneal problems, cataract, retinal distortions or raised intraocular pressure, minimal diplopic separation of images in small angled strabismus, accommodation or convergence anomalies, acquired nystagmus, decompensating heterophoria, tearing, or visual field loss.

The mechanism is not always a purely optical distortion of the retinal image, and the perceptive abilities of patients often label quite distinct disorders such as lack of visual field or closely associated diplopia as blurriness. Visual symptoms resulting from a disturbance of the vestibular system, such as oscillopsia, will often be labelled similarly.[1] Whilst vertigo is an illusory sensation of motion of self or of the environment, oscillopsia is an illusory to and fro movement of the environment. This is often reported as vision becoming blurred with movement of the head or body.[2] The key to diagnosis of a vestibular aetiology relating to a complaint of blurred vision is a history of visual disturbance which only occurs during head movements.[1] Whilst it is important to determine the characteristics of the blur itself, its precipitation and relief features, it is imperative to evaluate its relationship to head and body movements.

THE VESTIBULO-OCULAR REFLEX

The vestibulo-ocular reflex is part of the central nervous system in which connections between the balance sensors within the ear, the brainstem, the cerebellum and the visual system function to stabilise gaze and ensures clear vision during head movements.[3] The cervico-ocular reflex is similarly involved with reflexes in neck and spinal cord movement and the optokinetic ocular reflex is effective in sustained body rotations.[4] At 70 milliseconds, visual retinal processing is too slow to maintain steady images in response to natural rapid head movements, whereas the latency of

the vestibulo-ocular reflex is only 16 milliseconds.[2,4] Total compensation of a prompt slow phase gaze movement to head rotation is not achieved, and in fact would be undesirable due to retinal stabilisation and fading of the image. A horizontal head turn to the right produces an equal eye movement in the orbit to the left, so that the summated movement does not change and the image of the world does not move significantly on the retina. During instances of sighting an object using a combination of eye and head movement in the same direction, this vestibulo-ocular reflex is suppressed, probably by the pursuit system.[2]

The stimulus to the reflex is head movement, which creates a change in the position of the fluid within the balance sensors of the ear. The impulses that are triggered follow a pathway through the brainstem to the extraocular muscles. The balance sensors involved are the labyrinthine apparatus or semicircular canals and otoliths. Impulses are transmitted by the VIII cranial nerve to the VIII cranial nerve nuclei, where communication occurs between both sides of the brainstem. (Figure). After synapsing in the VIII cranial nerve nuclei, impulses travel to the contralateral VI, IV and III cranial nerve nuclei to supply the extraocular muscles. Each semicircular canal within a labyrinth is directly linked with a pair of synergistic extraocular muscles. Simultaneous impulses travel to the cerebellum, which has projections back to the VIII cranial nerve nuclei as a monitoring system. Labyrinthine, brainstem or cerebellum dysfunction may lead to a malfunction of the vestibulo-ocular reflex, and some drugs will also decrease the vestibulo-ocular reflex gain. This gain of eye velocity to head velocity is ideally matched at 1.0.

Clinical or laboratory testing of the vestibulo-ocular reflex can be used to determine if this system is intact, and if it is efficient. Electrodiagnostic techniques, chair rotations or caloric testing (water stimulation of the semicircular canals within the labyrinthine system of the ear) give detailed investigations. Clinical methods include ophthalmoscopy with head movements,[2] watching for corrective saccades[2,4] and testing visual acuity with head movement.[3] Various

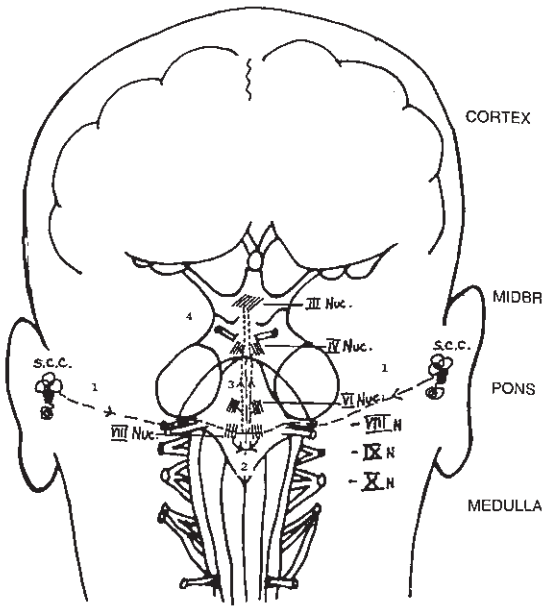

Figure 1: Pathway for the vestibulo-ocular reflex: (1) Stimulus from semicircular canals to VIII cranial nerve nucleus; (2) Communication between both VIII cranial nerve nuclei; (3) Contralateral innervation to VI, IV and III cranial nerve nuclei; (4) Innervation to extraocular muscles.

methods have been postulated for testing vision whilst a patient's head is rapidly turned. The most recent sensitive test shows a monitor with a triggered visual display of square wave gratings at intervals of 50 milliseconds.[5] Whilst this determines the degree of lack of retinal stabilisation as compensated for by the vestibulo-ocular reflex, the clinical test performed by an orthoptist of visual acuity assessment with head movements is sufficient in determining an influence on the visual system by vestibular dysfunction.

METHOD

The clinician firstly ascertains the optimum visual acuity of the patient viewing binocularly. This does not imply binocular single vision, but purely the visual functioning of the patient under normal viewing conditions with both eyes open. Snellen's Charts, Logmar Charts, E Charts[3] or the usual clinical methods may be employed. Having informed the patient of the nature of the

test, place open hands firmly but gently on either side of the head and perform small horizontal oscillations. The patient is to attempt to continue to read their optimum visual acuity levels. If this is not possible, the next best levels are indicated by the patient. The amplitude of the head movement does not need to be large, as it is the movement itself that induces the reflex. The speed of movement should approach that of natural rapid head movements, being two-three cycles per second.[2] If the movement is slower than this, optokinetic or cervico-ocular reflexes may be compensating for vestibular dysfunction and no defect will be seen. At speeds approaching natural head movements, only the vestibulo-ocular reflex is fast enough to generate compensatory eye movements, and if this system is impaired, a defect will be found. This defect will manifest as a reduction in visual acuity during head movements.

Three or more lines decrease in vision levels from head stationary to head movement readings indicate vestibular dysfunction.[3] However, suggestions have been made that any decrease in visual ability due to head movement may be indicative of pathology. Whilst there appears to be a large variability in the efficacy of normal vestibulo-ocular reflexes to compensate for head movements, in order to detect a degradation in vision levels due to retinal smear, the head rotations in normal subjects need to be at speeds three times faster as compared with those suffering vestibular disease.[5] It is therefore unlikely that a head rotation performed in a clinic would be fast enough to elicit poor efficacy in a normal patient, but be of sufficient speed to detect poor compensation as a result of vestibular disease.

The procedure is repeated with vertically induced head movements[1] and the results noted. No additional clinical equipment is required to perform this test and the time taken usually does not exceed 30 seconds.

CASE HISTORY

The following case illustration highlights the value of clinically testing the vestibulo-ocular reflex and shows its significance. MJ, aged 32, presented with a history of closed head injury five months previously, following an assault. Surgical evacuation of a left occipital extradural haematoma and resuscitation from respiratory and cardiac arrest formed part of his previous treatments. Formal eye assessment did not occur until this period of time post injury. He had initially spent one week comatosed with cerebral oedema and moderately dilated ventricles. The patient's main complaint was of blurred vision and diplopia. Visual acuity measured 6/9 in each eye, both improving to 6/5 with a pinhole. Cover testing revealed an intermittent alternating esotropia of the divergence weakness type with small left hypertropia seen with distance fixation. Ocular movements revealed the L/R to be greatest in left gaze positions, being the areas of most troublesome diplopia. Hess charting showed a small left lateral rectus paresis. Vertical saccades and pursuit were intact, but saccades were undershooting on left gaze, and cogwheeling occurred to both sides. Convergence and accommodation were intact and normal.

Pupils were reactive to stimuli of light and accommodation, but the resting state of the left pupil was greater than that of the right. Binocular functions were present and normal. Visual fields of the right eye were full, and a small relative superior arcuate defect was seen for the left eye. Intra ocular pressure was normal. A small congenital posterior lens opacity was seen in the left eye on dilation. Whilst signs appeared to indicate probable partial resolution of ocular paresis(es) and mild disruption to eye movement systems, the signs were not consistent with the symptom of the blurred vision. Further questioning revealed that the problem occurred with movement, as in travelling in a car, and disappeared when the patient was stationary. Results to clinical testing of the vestibulo-ocular reflex showed a decrease in visual acuity from BEO 6/9 with head stationary to BEO 6/60 with head movement.

DISCUSSION

Impairment of the vestibulo-ocular reflex can be quite debilitating. The patient in the case illustrated had a visual reduction from 6/9 to 6/60,

which simultaneously reduced the quality of his life with any head movement. The significance of performing this simple test can be many fold. The most obvious one is the diagnostic value to the clinician in localising a symptomatic visual problem to a vestibular dysfunction. Thereafter, appropriate referral can follow. Patients are also relieved and grateful if they can be told that they have a real and organic problem and do in fact require further investigation. This empathy and reassurance is most therapeutic to the patient. Irrelevant and fruitless investigations will be avoided as outlined elsewhere,[6] thereby reducing patient anxiety, discomfort and expense. Differentiation is aided, in the medico-legal sense, between a recent or longstanding problem. The onset of the symptoms will be quite abrupt, the timing well described by the patient and the reduction in vision seen easily with head movement testing. Orthoptic reporting will be made more comprehensive. The sequelae of vestibular problems may be that compensation occurs or that a level of stability is reached and maintained without further adaptation. In cases following vestibular neurectomy, clinical compensation was no further improved after one year as compared to one week post-operative.[4] Compensation may be similar in other disease processes. Visual therapies may not be directly applicable, but awareness of the problem by the patient may encourage some adaptive procedures. Improved knowledge in testing procédures and their underlying bases will benefit discussions between orthoptists and the relevant specialists involved, such as neurologists, ear, nose and throat specialists and ophthalmologists.

SUMMARY

When confronted with a history of blurred vision with head movement, the testing of the efficacy of the vestibulo-ocular reflex to prevent retinal slip during head movement can be performed during an assessment of the integrity of ocular muscle movement and the eye movement systems. It is a test that is very quick to perform, requires no additional clinical equipment and provides significant information on the relationship of the vestibular system to the ocular system, and can indicate when referral for further investigation may be required.

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References

1. Halmagyi GM, Henderson CJ. Visual symptoms of vestibular disease. Aust NZ J Ophthalmol 1988; 16: 177-179.
2. Leigh RJ, Zee DS. The neurology of eye movement. Philadelphia: FA Davis Company, 1983: 11-33.
3. Longridge NS, Mallinson AI. The dynamic illegible E (DIE) test: A simple technique for assessing the ability of the vestibulo-ocular reflex to overcome vestibular pathology. J Otolaryngology 1987; 16: 97-102.
4. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol 1988; 45: 737-739.
5. Henderson CJ et al. Visual acuity and gaze precision. In: Keller EL, Zee DS, eds. Adaptive processes in the visual and oculomotor systems. New York: Pergamon, 1986: 367-371.
6. Marra IM et al. Subjective oscillopsia ("jiggling" vision) presumably due to aminoglycoside ototoxicity — a report of two cases. J Clin Neuro-ophthal 1988; 8: 35-38.