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EDITORIAL

PARTICLE REPOSITIONING MANEUVER

The main function of vestibular system must be appreciated before proceeding to rehabilitation, which is to sense the head movements in relation to body position and gravity especially involuntary movements and reflexesly counter them with body and eyes movements to prevent the body from falling. The signals from vestibular labyrinth by inertial sensors detect rotatory, linear and gravitational acceleration. Three pairs of semicircular canals of two sides are arranged orthogonally in complimentary and coplanar pairs[1] where one canal is stimulated other side is inhibited and otolith viz utricle and saccule senses gravitational and linear accelaration. Signals are transmitted by secondary vestibular neurons through vestibular nuclei in brain stem to (1) occulomotor nuclei to extra- occular muscles (vestibulo- ocular reflex) (2) cervico spinal motor nuclei to generate cervico occular reflex (3) lower motor spinal nuclei to generate the vestibulo spinal reflex. The somatosensory information from propiocep- tive sensors in the limbs contribute to the sense of vertical body position.[2] Postural information from major blood vessels and abdominal viscera by gravity receptors also provide postural information. It also imparts sensation to autonomic centre which controls the cerebral perfusion. Lastly, the vestibular input to cerebellum is necessary for coordination and adaptation of reflexes.

Image on retina also plays role in balancing the body. Movements of eyes in coordination with moving object maintain the focus on fovea. Retinal slip velocity (difference between velocity of the object and the velocity of eye movement), generates the occulo motor command signals. It is worth recalling that three semicircular canals are right angle to each other sensing rotational movement of head. The medial two third of utricle is stimulated on ipsilateral tilt and its lateral one third on contralateral tilt. Saccule is stimulated in downward and upward direction. According to the Ewalds Law, stimulation of the semicircular canals which are fluid filled, produce eye movements in the plane of canal independent of pupil position or head position. In lateral semicircular canal endolymph flows in the direction of utricle and ampulla when head is turned towards the same side in horizontal plane. Hence stimulation takes place. Taller end of stereociliary bundle points towards the utricle in horizontal canal. In vertical canals taller end of bundle are stimulated by movement of endolymph away from utricle hence, movement of endolymph away from ampulla result in stimulation by turning the face towards ipsilateral side in horizontal plane. Anterior semicircular canal is stimulated by flexing/ anterior bending/ head down and ipsi lateral tilt and post semicircular canal is stimulated by posterior bending/ extension / head up and tilt to ipsilateral side.

The endolymph becomes lighter by heating and heavier on cooling which is the basis of stimulation by caloric test. By virtue of density changes endolymph moves either towards or away from ampulla. In supine 30^0 head end up position warming stimulates the horizontal semicircular canal, endolymph rises towards the ampulla resulting in slow phase of nystagmus towards the opposite side and quick phase directed to same side.[3] The nystagmus is denominated by quick phase hence said that hot stimulation generates same sided nystagmus while cold stimulous generates opposite side quick phase nystagmus. Ewalds

Second Law states if there is asymmetry in excitation and inhibition, there is stronger reflex generation in the on direction.

Pathology of labyrinth mostly affects more than one canal and observing the axis of the nystagmus examiner can interpret what combination of semicircular canals are stimulated or diminished. When one labyrinth is irritated as after stapes surgery, slow phase of nystagmus is towards the contralateral side and torsional component moving the superior pole towards contralateral side and after left labyrinthectomy nystagmus will be leftward and counter clockwise and slow phase will be rightward and clockwise beating. Always remember magnitude of vestibular nystagmus is influenced by the gaze.[3]

Utricle senses linear acceleration which are tangential to its curved surface and in horizontal plane. Ipsilateral loss of utricular nerve activity results in ocular tilt reaction i.e. head tilts towards the side of lesion and disconjugate deviation as pupil of the side of lesion is depressed and intact side pupil is elevated.

Saccule lies in parasagital plane, excited by displacement away from striola, mostly senses up and down motion but some afferents can sense to and fro movements also. Afferents in the upper half are excited by upward acceleration as in sudden fall and compensatory reflex activates the extensor of trunk and body while flexor are relaxed to restore the tone and posture of the body, the otoconia are stimulated by gravity (9.8 m/sec^2) constantly puiling it towards earth.

The diagnosis of vertigo can usually be made by elaborate history and clinical examination but one has to devote sufficient time, preferably prepare an objective questionnaire. Vestibular disorder can be diagnosed more conveniently and Benign Paroxysmal Positional Vertigo (BPPV), the most common, is most easy to diagnose. The rehabilitation or repositioning therapy provides success/ relief in more than 85% cases. Failure is due to inaccurate or inadequate procedure or associated diseases.

The usual precipitating factor of BPPV is head injury or viral vestibular neuritis/ labyrinthitis though metabolic factors have also been attributed.[4]

The etiology of vertigo in BPPV is accumulation and movements of debris, otoconia in semicircular canal, hence repositioning the debris back into utricle is the cure but remember, this extra debris in utricle require some duration for habituation. Identification of affected canal is dependent upon history, head position inducing nystagmus and its direction and relief after repositioning.

Posterior Semicircular Canal

Being the most common, affected in about 85 % cases.[5,6] The Dix- Hallpike Maneuver is the gold standard. Patient should be seated comfortably on the examination coach, so that the head can be lowered or can hang below the level of table. The head is turned 45° to the affected side and patient is moved rapidly to lying down position maintaining the head in 45° side wise and simultaneously while lying down lower the head by 30° to 45°. Now the presence, latency, duration and direction of nystagmus should be observed and recorded.[7,8]

Positive Results

The nystagmus is usually delayed (latency 1-5 seconds) but immediate onset suggests central lesion. Visible nystagmus usually lasts 30 seconds, which is mixed torsional and vertical with the upper pole of eyes moving towards the affected ear. On repeating, the test vertigo/ nystagmus is diminished or abolished due to dispersion of otoconia in endolymph specifically in canalolithiasis. While in cupolothiasis nystagmus is lesser fatiguable and even results are poor on particle repositioning maneuver (PRM)

The patient should be explained the procedure, its implications and complications before doing the procedure specifically in cardiac or vascular cases (Vertebro Basilar System insufficiency) and risk of dislodged carotid artery atheromatous emboli.[9]

The PRM beginning is like Dix Hallpike's procedure, patient is put in lying down position with 30° head extended or hanging down with 45° lateralized.

When performing PRM, an assistant should be on side to support the patient body (shoulders) and neurotologist holds the head with one hand and neck supported by another hand.

The position is maintained for two more minutes after the complete cessation of nystagmus and vertigo.

Head is rotated 45° to the opposite side that is 90° from the previous position making the unaffected ear down keeping the head hanging/extended.

Patient is retained in this position for two more minutes after the cessation of nystagmus and vertigo.

This is followed by rolling the patient by 90° on his/her side (135°) from supine position towards the unaffected side, nose pointing towards the ground. Again patient is retained for two more minutes after the cessation of nystagmus and vertigo.

Patient is brought to sitting posture with the head held in same posture.

The patient head is turned to face forward with chin angled down by 20° and held for 2 more minutes after cessation of nystagmus and vertigo.

Repeat procedure after 30 minutes if there is vertigo/ nystagmus on Dix Hallpike maneuver.

Epley has used mastoid vibrator,[10] while Tirellin has used head shaking[11] or to achieve better success rate. Author performs it by gentle tapping over the head for better mobilization of debris.

One must differentiate between canalolithiasis/ cupulolithiasis and canalith jam.[12] They may exist in isolation or in combination. In cupulolithiasis where the head is placed in Dix Hallpike position latency is not there and nystagmus and vertigo last till the position is retained while in canalith jams, which present as sudden conversion of transient nystagmus to a rapid and sustained nystagmus irrespective to head position usually observed after PRM. It can be corrected by reversing the just completed maneuver.[12] Rarely the particle during PRM may migrate into other semicircular canals and repeating the procedure is not going to help. Case should be reviewed after 48 hours. Unnecessary repeating the procedure is not going to relieve the symptoms.

Lateral (Horizontal) Semicircular canal (BPPV)

The LSC BPPV, first described by Cipparrone et al and Mcelure in 1985, nystagmus is provoked by supine lateral head turn with nystagmus beating towards the under most ear.[13]

It may be geotropic or apogeotropic depending upon the canalolithiasis versus cupulolithiasis.

Geotropic as mentioned is characterized by short latency, prolonged horizontal nystagmus, beating towards the under most ear with poor fatigue ability.

Apogeotropic is characterized by short latency, prolonged horizontal nystagmus but the direction of beating is different that is it beats away from the undermost ear. Nystagmus is usually worse when the affected ear is upper most and spontaneous nystagmus may be seen occasionally in supine position, that usually beats towards the involved side, to further confirm the diagnosis. Cold caloric may be performed to further confirm the diagnosis, which is hypoexcitable more so in geotropic type with asymmetric vestibulo occular reflex and abnormalities in dynamic posturography.

PRM in geotropic is 360° rollover.

The procedure begins with the patient in supine position, head flexed 20° to 30° and laterally rotated by 45° towards the affected side, patient is rolled in steps of 90° towards supine and unaffected side by every 30 to 60 seconds (author prefers 2 - 3 min.) keeping the head flexed throughout. The procedure can be

repeated several times if the symptom persists.

In Apogeotropic type the 360° rollover maneuver is attempted initially if no response Gufani Maneuver which is performed as patient is sitting in beginning lyes down quickly to affected side and then rotating the head 45° downward maintaining the position for 3 minutes.[14] The Vannucchi-Asprella maneuvers were performed with the patient rapidly moving from the sitting to the supine position then turning the head rapidly to the unaffected side and returning to sitting where the head was then returned to midline. This maneuver is repeated 5 to 8 times in rapid succession.[15]

Anterior canal positional vertigo

It is rare which accounts for less than 2 % cases. It is characterized by predominant down beating nystagmus with small torsional component in response to D.H. test.[16]

The repositioning maneuver is identical to Epleys maneuver used for posterior semicircular canal of one side PRM is of otherside's anterior canal. The only difference is the anterior canal projects to the ipsilateral superior rectus muscle and to the contralateral inferior oblique muscle; the nystagmus is downbeating and torsional during the DH. If the downside ear is affected the direction of the torsional component will be the same as in contralateral PC BPPV.[17, 18, 19]

The incidence is low of anterior canal due to its anatomy as debris in canal is self clearing because posterior canal descend directly into common crus and utricle. In cases of recurrence or failure, case should be reviewed between central versus peripheral lesion followed by reconfirmation of diagnosis of BPPV. Simultaneously ischemic, metabolic, orthopaedic (cervical spondylitis) vascular (vertebral artery) abnormalities should be looked for.

Particle repositioning maneuver is simple, effective, safe procedure with high success rate and low morbidity. It can be taught to paramedical staff and physiotherapist. Patient should be motivated to lead active life along with rehabilitation exercises.

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TANEJA M.K.
EDITOR