

Side Lying Test for Anterior Semicircular Canal Benign Paroxysmal Positional Vertigo

INTRODUCTION

The diagnosis, localization, and lateralization of the involved semicircular canal (SCC) in benign paroxysmal positional vertigo (BPPV) are solitarily dependent on the oculomotor patterns of nystagmus elicited on the diagnostic positional tests: Dix-Hallpike test (DHT) for the vertical canals (posterior and anterior semicircular canals) and supine roll test for horizontal semicircular canal. After following observations in 100 patients, Dix and Hallpike^[1] (1952) propounded a symptomatic definition and a provocative positional test, which they referred to as “positional nystagmus of benign positional type.” In the maneuver innovated by them, the patient was first seated upon the examination couch with his head turned to one side and the gaze fixed firmly on the examiner’s forehead. Thereupon, the examiner would grasp the patient’s forehead firmly between his hands and briskly push the patient back into the critical position 30° below the level of the couch and subsequently turn the head approximately 30°–45° to one side. They noted torsional nystagmus with the upper pole of the eyes beating toward the ground and on retesting, this response fatigued. Response latency of approximately 5s with a crescendo-decrescendo type of nystagmus and its reversal as the patient sits up was also observed. The DHT now in use, as per the clinical practice guidelines of the American Academy of Otolaryngology, Head, and Neck Surgery Foundation, recommends taking the head 20° below the level of the examination table instead.^[2] The DHT orients the vertical canals (posterior and anterior semicircular canals) in a manner that the ampullary end of the canal displaces to a relatively superior location facilitating the excitatory ampullofugal movement of the otoconial debris. The ampullofugal cupular deflection during the DHT generates the excitatory impulses in the posterior [in posterior semicircular canal-BPPV (PSC-BPPV)] and anterior (in ASC-BPPV) ampullary nerves resulting in the diagnostic oculomotor patterns (upbeating in PSC-BPPV, downbeating in ASC-BPPV; ipsitorcional in either case). The DHT is difficult to perform in patients with a limited cervical range of motion in the pitch plane, and those with obesity, fear, and musculoskeletal limitations. In addition, the biomechanics of the DHT has the potential to strain the back region of the clinician.

Side-lying test (SLT) as an alternative test for assessing the function of vertical semicircular canals was first described by Herdman and Tusa^[3] in 1998 but has been never reported for diagnosing the ASC-BPPV. We report here a middle-aged male patient seen by us in the 2nd week of January 2021, who was diagnosed left ASC-BPPV, based on observing the elicited downbeating left torsional (from the patient’s perspective) positional nystagmus on the SLT to his left. The mechanism of

downbeat ipsitorcional positional nystagmus during left-sided SLT is discussed.

CASE DESCRIPTION

History

A 41-year-old male patient presented in the 2nd week of January 2021 with 3 weeks history of vertigo on lying supine, getting up from supine, and in either of the lateral recumbent positions. Neither there were associated symptoms to suggest central vertigo nor any history of preexisting medical, surgical, or orthopedic illnesses contraindicating diagnostic and/or therapeutic maneuvers for BPPV.

Examination

The general physical examination and vitals of the patient were normal. The screening assessment of the cervical spine did not reveal any limitation of movement. The examination of the back region did not reveal kyphoscoliosis. The examination of the lumbosacral spine, including straight-leg raising (SLR) test and reverse SLR test, was normal. The neurologic examination revealed normal cranial nerve examination; strength was grade 5/5 in all 4 limbs with normal deep tendon reflexes, and bilateral flexor plantar reflexes. The examination of the cerebellar system revealed no spontaneous or gaze-evoked nystagmus, and there was no appendicular or axial incoordination. The Oto-neurological examination revealed normal vertical and horizontal saccadic and smooth pursuit eye movements. The head impulse test was bilaterally normal. The routine blood investigations and magnetic resonance imaging of the brain with screening of craniovertebral junction were normal.

Before carrying out positional tests, the presence of spontaneous nystagmus was ruled out by using takeaway Frenzel goggles.^[4] The patient did not consent for the DHT, and for this reason, SLT was done to assess the vertical canals as per British Society of Audiology guidelines.^[5] The SLT was carried out with the patient in short sitting on the examination table. The head is rotated 45° in the yaw axis opposite to the side to be tested and quickly brought down to the side opposite to the direction to which the head is yawed. Nystagmus direction is observed and associated vertigo is enquired. A similar sequence of positioning was also carried out to the opposite side. Additional examination with downward tilting of the examination table 20° during the side-lying position theoretically increases the diagnostic yield of the SLT by orienting the ampullary arm of the vertical semicircular canals in a near-vertical position with the ampulla at its superior-most location provided the head excursion angle in the yaw-axis to the opposite side is about 45°.^[6,7] On side-lying to left side, after a latency of 8 s, a downbeating left torsional (from the patient’s perspective) positional nystagmus is elicited that

lasts for 55 s [Video 1, Figure 1]. The supine roll test was carried out to evaluate the horizontal SCC and rule out the rare non-fatigable central horizontal positional nystagmus, which was negative in the test.^[8]

Intervention

The clinical history, as well as the otoneurologic examination, was consistent with the diagnosis of left ASC-BPPV. The patient was treated with Yakovino maneuver (YM) [Video 2, Figure 2].^[9] A total of 5 YM were done.

Diagnosis and intervention

The aforesaid clinical history, otoneurologic examination, and response of vertigo to the therapeutic YM are consistent with the diagnosis of left ASC-BPPV. Verifying SLT undertaken at 1 h [Video 3] and 24 h after the YM neither elicited the downbeating positional nystagmus nor the associated vertigo.

Prognosis and outcome

The patient was telephonically questioned weekly regarding the recurrence of rotational vertigo for the next 4 weeks, and it was confirmed that the patient remained symptom free till then.

DISCUSSION

Subjecting a patient with a history of rotational vertigo triggered by changes in the position of head relative to the gravity, to any provocative positional test, opens a Pandora’s box of a diagnostic dilemma if a positional

downbeat nystagmus is elicited. If such a patient does not have any other central nervous system symptoms or signs, the responsible lesion could be either serious localizing to the intracranial posterior fossa, craniovertebral junction; or benign due to conditions like ASC-BPPV. SLT to left with the head rotated 45° to right [Figure 1a and 1b] orients the ampullary end of the left ASC to a relatively superior location resulting in the ampullofugal displacement of the otoconial debris [Figure 1d and 1e]. This generates excitatory impulses in the left anterior ampullary nerve [Figure 1f], leading to generation of slow-phase vestibulo-ocular reflex (VOR), which moves both eyes upward and with torsion to the right (from the patient’s perspective) due to co-contractions of the left superior rectus and right inferior oblique muscles [Figure 1c]. Therefore, the fast phase VOR, which is clinically appreciated as positional nystagmus, is downbeating and left torsional from the patient’s perspective [Video 1]. A recently described variant of PSC-BPPV of the opposite side due to nonampullary canalolithiasis, called apogeotropic PSC-BPPV (*apo*-PSC-BPPV), is an important diagnostic consideration in patients in whom positional tests (right and left Dix-Hallpike’s and straight head-hanging positioning) elicit a torsional downbeating nystagmus.^[10-14] SLT to left with the head yawed 45° to the right is unlikely to result in the ampullopetal movement of otoconial debris situated in the non-ampullary arm of the right posterior SCC [Figure 3]. Justifiably, the patient was diagnosed as left ASC-BPPV,

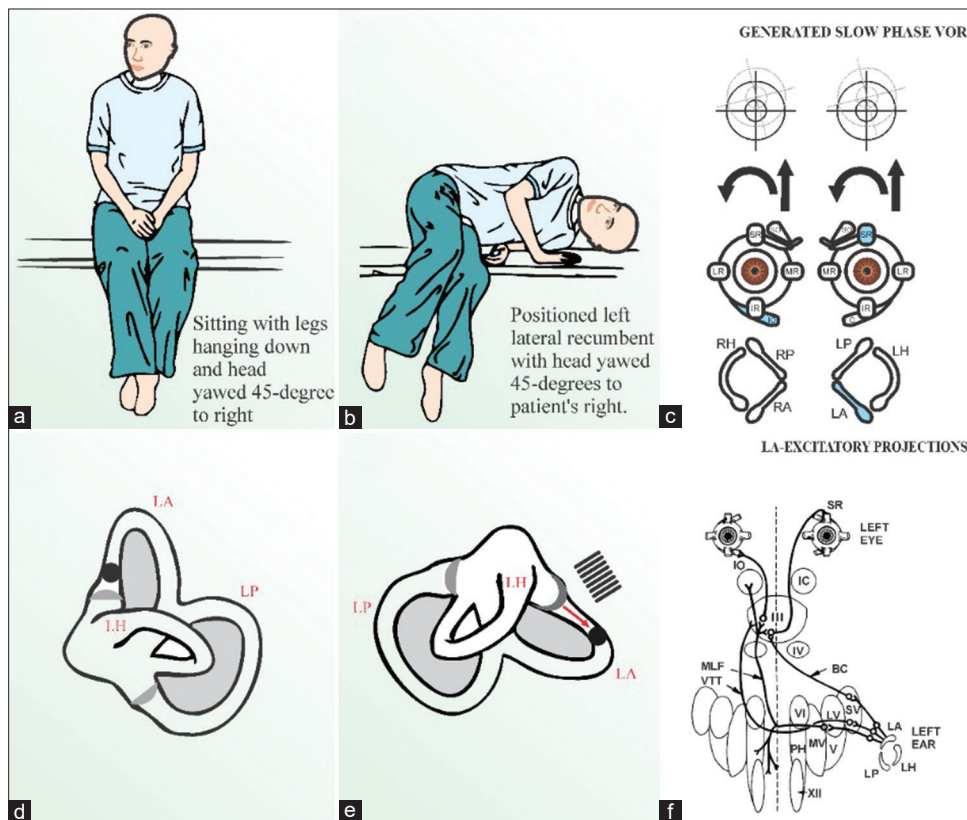


Figure 1: Side-lying test to left (a, b) illustrating ampullofugal otoconial movement and cupular deflection (d, e), generating slow phase VOR (c) due to excitatory projections of LA to left SR and right IO (f)

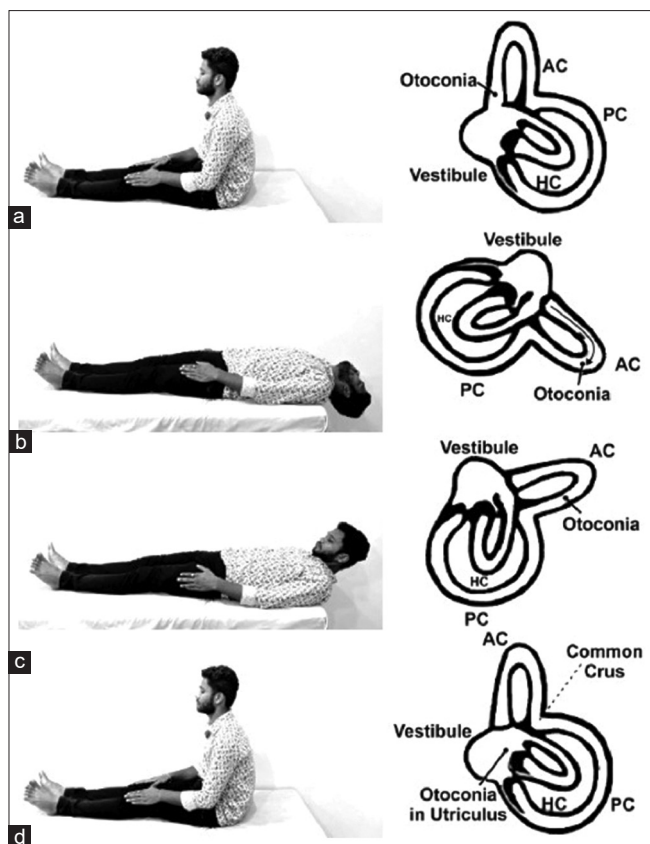


Figure 2: With patient in long sitting on examination table (a), supine positioning is done with head extended 30° on the short edge (b) for a minute. Thereupon, head is flexed about 30° above horizontal (chin to chest position) for a minute (c). Finally, patient is upright to long sitting (d). Abbreviations: AC, PC, and HC; anterior, posterior, and horizontal semicircular canals respectively

treated with the sessions of YM, and followed up at 1 h after 24 h post physical therapy. An avid response to YM with disappearance of positional left torsional downbeating nystagmus as well as associated vertigo at 1 h post YM [Video 3] and after 24 h established the diagnosis of left ASC-BPPV. Califano *et al.*^[11] (2014) proposed criteria for the diagnosis of ASC-BPPV with three categories “Certain,” “Probable,” and “Possible,” based on elicited nystagmus and its characteristics on the straight head hanging test. It is interesting to note in the proposed classification that patients eliciting positional downbeating nystagmus during the diagnostic straight head hanging test but switched canal after therapeutic maneuver were classified as “Certain” while those who directly resolved as “Possible.” The straight head hanging maneuver orients the membranous labyrinth in a manner that otoconial debris in the non-ampullary arm near the crus commune of the posterior SCC displaces towards the cupula resulting in ampullopetal cupular deflection. In the case reported by us, otoconial debris in the non-ampullary arm near the crus commune of the right posterior SCC could have resulted in an identical left torsional (from patient’s perspective) positional downbeating nystagmus due to the generation of inhibitory discharges in the right posterior ampullary nerve due to ampullopetal cupular

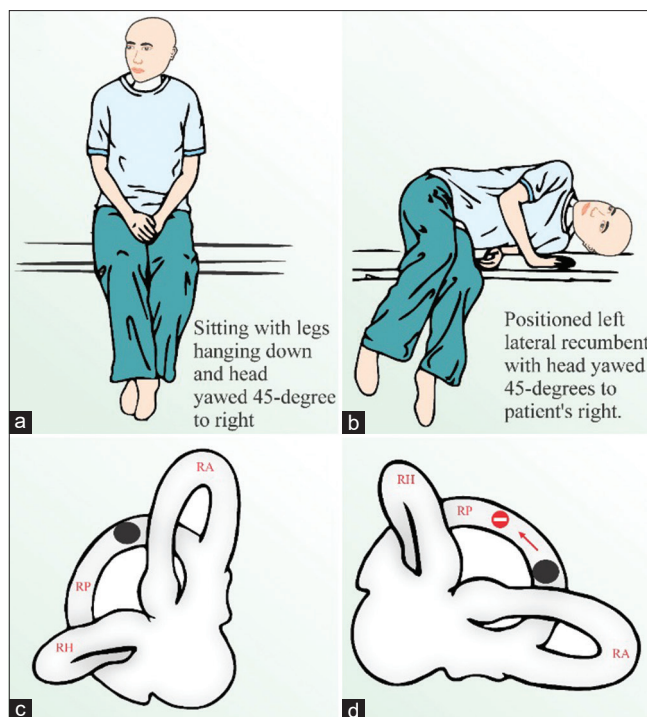


Figure 3: Side-lying test to left (a, b) orients the right posterior semicircular canal such that otoconial debris in its non-ampullary arm is relocated to gravity-dependent position, and cannot displace ampullopetal to effect cupular deflection (c, d). Abbreviations: RA, RP, and RH; right anterior, posterior, and horizontal semicircular canals respectively

deflection [Figure 4] but SLT to left with the head yawed 45° to the right is unlikely to do so [Figure 3]. We suggest SLT as an additional assessment tool for the cases suspected of vestibular lithiasis, where straight head hanging test elicits a torsional downbeating positional nystagmus.

CONCLUSION

The localization of peripheral positional downbeating torsional nystagmus is challenging because an ipsilateral ASC-BPPV is almost impossible to differentiate from a contralateral *apo*-PSC-BPPV (non-ampullary posterior semicircular canalolithiasis) based on the oculomotor patterns generated during the straight head hanging test. A phenomenon of canal switch and elimination of positional downbeating nystagmus, after the therapeutic maneuver, is deemed essential for a “Certain” or “Possible” diagnosis, respectively. SLT appears to be a promising clinical tool for eliciting positional downbeat nystagmus imputable to ASC-BPPV.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

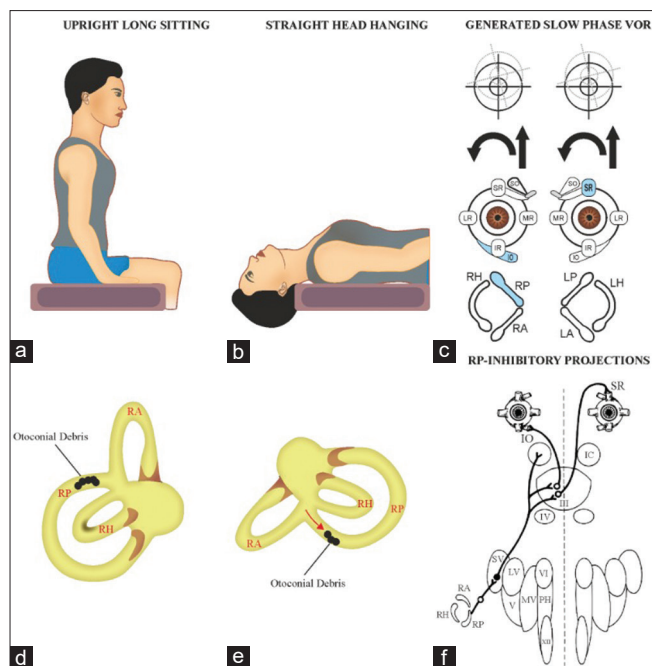


Figure 4: Straight head hanging test in a patient suffering from apo-PSC- BPPV (a, b) displaces the otoconia in a manner resulting in ampullopetal cupular deflection (d, e). The inhibitory impulses in right posterior ampullary nerve project to right IO and left SR (f) resulting in slow phase VOR (c)

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Conflicts of interest

There are no conflicts of interest.

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