Benign paroxysmal positioning vertigo (BPPV) is the most common cause of vertigo in the elderly, with a reported prevalence of unrecognized BPPV of 9% in a recent population study by Oghalai et al. (2000), and is also very common in younger age groups. The pathophysiology of the disorder has been elucidated in recent years (Brandt, 1999). The etiology of this form of vestibular vertigo is the appearance of particles in the endolymph of the semicircular canals in the inner ear, probably caused by dislodged otoconia or spontaneously occurring calcium crystals, thus producing particles that are heavier than the endolymph. When the position of the subject’s head is changed, the particle will slowly descend through the semicircular canal, deflecting the cupula and thus producing vertigo and nystagmus of some seconds duration.

In the most common form of BPPV, the posterior canal type (pBPPV), the vertigo is most pronounced when lying down in bed, and especially so if the subject rotates his or her head to the affected side. Getting up from bed induces similar responses, while rotating the head from side to side in the supine position will produce inconsistent symptoms. In the less common, horizontal canal variety (hBPPV), the symptoms and signs are most readily induced by rotating the head in the supine position to either side.

Most patients with BPPV experience vertigo in the morning, either on awakening or when rising from bed, a pattern named “matutinal vertigo” by Fisher (1967). Although many BPPV patients complain of insufficient sleep, abnormal nocturnal awakenings from sleep accompanied by vertigo are not common complaints in these patients, in our experience. Possible explanations for this might be that patients suffering from BPPV move less during sleep compared to healthy persons, that awakenings caused by BPPV attacks might be too short to be remembered in the morning, or, alternatively, that these patients do not experience vertigo and nystagmus when subjected to vestibular stimulation during sleep. Some data has been published indicating that oculovestibular reflexes are diminished or absent during sleep (Reding and Fernandez, 1968; Kasper et al., 1992; Cabungcal et al., 2001). The present report of polysomnographic studies of three patients suffering from BPPV is intended to shed some light on this theory.

METHODS

Three patients with a clinical diagnosis of Benign Paroxysmal Positioning Vertigo, based on characteristic vertigo and nystagmus during positional testing (Hallpike Test), were studied by conventional polysomnography, including standard sleep EEG channels, electrooculogram (EOG), submental EMG, ECG, respiratory movement sensors, thermistor, pulse oxymetry and position sensors. The only non-standard feature of the recording was that the position sensor was taped to the forehead instead of the chest, as conventionally done.

Nystagmus, defined as repetitive eye movements with no pauses between movements and with an easily-discernible slow and fast phase, was identified in the EOG records using the standard gain and time constant (0.6 seconds).

In three elderly patients with a clinical diagnosis of Benign Paroxysmal Positioning Vertigo (BPPV), two with posterior canal BPPV and one with horizontal canal BPPV, polysomnography was performed with a position detector attached to the forehead. A total of 14 head movements were recorded during sleep. None of these were followed by nystagmus, while head movements during awake induced the expected nystagmus. However, a low sleep efficiency was seen in all cases, and 13 of the head movements were followed by at least one epoch of awake. BPPV might be a cause of sleep problems, but the polysomnographic findings are unspecific, as the oculovestibular reflexes are abolished during sleep.

CURRENT CLAIM: Head movements during sleep in patients suffering from benign paroxysmal positioning vertigo evoke no nystagmus but are associated with awakenings and might be a cause of sleep disturbance.
**Patient 1: Female, 79 yrs. old**
Right posterior canal BPPV of six-months duration.
Provoking factors: lying down, rising from bed, rolling from side to side.

**Patient 2: Female, 77 yrs. old**
Left horizontal canal BPPV of three-days duration.
Provoking factors: rolling from side to side in supine position.

**Patient 3: Female, 65 yrs. old**
Right posterior canal BPPV of one-year duration.
Provoking factors: lying down, rising from bed, neck extension, rarely rolling from side to side.

**RESULTS**

In Patients 1 and 3, both suffering from the more common posterior canal BPPV, rotational movements while awake only occasionally evoked any nystagmus; nystagmus was identified in 6 of 11 and 6 of 8 head movements to the right side (in two subjects) and 5 of 10 vs. 7 of 12 movements to the left side. This nystagmus was generally of short duration, despite the patients’ subjective feelings of vertigo in the seconds after most positional changes (Figure 1). During sleep, no positional changes evoked any nystagmus (Figure 2).

In Patient 1, three head movements during sleep, all from the supine to the right position were seen: one in Stage 1, one in Stage 2, and one in REM sleep. Awakenings from sleep were seen in association with two of the movements. In Patient 3, four changes in head position were seen: one from Stage 3 sleep and the other three from Stages 1 and 2, two from supine to the right, and two from the right side to supine position. All changes of head position were followed by at least one epoch of the awake state.

In Patient 2, who suffered from left horizontal canal BPPV, every rotational movement from supine to the left or right position evoked nystagmus during the awake state. A degree of habituation was seen as the nystagmus became shorter in duration on repeated tests. The most intense and long-lasting nystagmus was seen after positional changes to the left, especially when rotating the head from the right to the left position. During sleep, seven head movements were seen: one from Stage 4 and the other six from Stages 1 and 2. Three of the head movements were from the right side to a supine position, one from supine to the right, and three from the left side to supine. All head movements were followed by at least one epoch of the awake state. No nystagmus was seen immediately following any of the head movements. A few nystagmoid jerks were seen in one instance, ten seconds after the movement (Figure 3). Four head movements were recorded during the first 30 seconds after awakenings; no nystagmus was associated with any of these movements (Figures 3, 4 and 5).

Sleep fragmentation and low sleep efficiency were seen in all three subjects (Table 1). An apnea index of 5.5 was seen in Patient 1; no respiratory disturbances were seen during sleep in the other two patients. No other etiology for their sleep complaints was found. However, only two patients were screened for PLMS.

![Figure 1](image-url). Positional change from supine to the right side in the awake state evokes nystagmus—traces FP1A1 and FP2A1, last 10 seconds of page. Patient 1: head position in the “body” trace—RS for right side; SU for supine; lower trace “Lys”—light intensity; EEG channels—C3-A2 and C4-A1.
Figure 2. Change from supine to right side during awakening from Stage 2 sleep evokes no nystagmus. Patient 1: traces as in Figure 1.

Figure 3. Change of head position from supine to the right side during Stage 3 sleep in Patient 2 evokes no nystagmus; a few nystagmoid jerks are seen 10 seconds later.
Figure 4. Continuation of Figure 3: no nystagmus is seen during a new positional change (from right side to supine and back) less than 30 seconds after awakening; a new position change is seen in the last two seconds of the page, evoking nystagmus in Figure 5.

Figure 5. Continuation of Figure 4: intense nystagmus is evoked after a new positional change more than 50 seconds after awakening; the change from right side to supine occurred at the end of Figure 4.
BENIGN PAROXYSMAL POSITIONING VERTIGO AND SLEEP

Testing whether stronger, more unpleasant vestibular stimulation might thus be an arousal stimulus during movements might be located in the oculomotor nuclei. Sleep despite the absence of reflexive eye movements. The nystagmus during unilateral vestibular stimulation is generally thought to consist of two components: a three-neuron arc serving the primary compensatory movement of the eyes intended to keep the eyes fixated on target, and a secondary, multisynaptic pathway that generates fast repositioning of the eyes, i.e., the fast phase or saccadic part of the nystagmus.

It should be noted that the standard electrooculography, as performed during polysomnography, is not well suited to pick up an unidirectional compensatory eye movement during a head movement, owing to technical (movement) artifacts and the rather short time-constant used for EOG recordings. With this caution in mind, the findings described above indicate that the oculovestibular reflex, or at least the saccadic part of it, is abolished during sleep. This is in accordance with other data.

Head movements that would be expected to evoke vertigo and nystagmus during the awake state did not produce nystagmus during sleep in these BPPV patients; neither did head movements shortly after awakening (within 30 seconds) evoke any nystagmus in these patients. The nystagmus during unilateral vestibular stimulation in static roll and pitch.

Although it seems premature to include BPPV among the known medical causes of disturbed sleep in the elderly, it seems prudent to inquire for symptoms of the disorder when interviewing patients with sleep problems. Simply inquiring about vertigo when awakening in the morning is not sufficient. “Matutinal vertigo” is an unspecific phenomenon, occurring in other diseases of the peripheral vestibular system as well as the central nervous system, as found by Maton et al. (1980) and Berkowitz (1985); in the author’s experience, it is not an obligatory symptom in BPPV patients. The polysomnogram will not disclose any specific findings as the clinical sign of the disorder, nystagmus after positional change, disappears during sleep.

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REFERENCES


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Table 1. Total Sleep Time (TST), Sleep Efficiency (from lights out), Inter-sleep Wake Time and Sleep Stage Shifts in the Three Patients.

DISCUSSION

Weak, low frequency, vestibular (otolithic) stimulation does not disturb sleep (Woodward et al., 1990). There are no studies testing whether stronger, more unpleasant vestibular stimulation disturbs sleep. In BPPV, the spontaneous head movements during sleep will move the otoconial debris and stimulate the utriculus, producing a sensory stimulus that is unpleasant in the awake state. The present study gives some indications (but no proof) that BPPV might be a cause of disturbed sleep.

A low sleep efficiency was seen, and 13 of the 14 head movements during sleep were associated with at least one epoch of awake after the “movement time” epoch where the head movement was recorded. However, there is a lack of normative data regarding head movements during sleep. Whether the head movements recorded in the study caused the awakenings, or rather are a result of the awakenings, is not clear. A larger study with healthy controls will be necessary to finally decide on this issue.

Although it seems premature to include BPPV among the known medical causes of disturbed sleep in the elderly, it seems prudent to inquire for symptoms of the disorder when interviewing patients with sleep problems. Simply inquiring about vertigo when awakening in the morning is not sufficient. “Matutinal vertigo” is an unspecific phenomenon, occurring in other diseases of the peripheral vestibular system as well as the central nervous system, as found by Maton et al. (1980) and Berkowitz (1985); in the author’s experience, it is not an obligatory symptom in BPPV patients. The polysomnogram will not disclose any specific findings as the clinical sign of the disorder, nystagmus after positional change, disappears during sleep.


