The Epley maneuver for the treatment of benign paroxysmal positional vertigo

Abstract  In recent years, different forms of physical therapy have been proposed for the treatment of benign paroxysmal positional vertigo (BPPV). These mainly consist of maneuvers aiming to reposition and disperse free-floating endolymph particles in the posterior semicircular canal. We report our experience with one of these procedures, the Epley maneuver (EM), in treating 30 cases of BPPV. Twenty-six patients (87%) were cured, while four (13%) did not respond to treatment. These results are similar to those reported in the literature for the EM and the Sémont maneuver.

Key words  Benign paroxysmal positional vertigo • Cupulolithiasis • Canalolithiasis • Epley maneuver

Introduction

Benign paroxysmal positional vertigo (BPPV) due to pathology in the posterior semicircular canal is a syndrome characterized by brief attacks of rotational vertigo elicited by rapid head extension with lateral tilt. Diagnosis is based upon history and observation of a positional nystagmus provoked by the Hallpike maneuver. This typically features: (1) latency of a few seconds; (2) duration less than 1 min; (3) mostly rotatory nystagmus, with the fast phase beating toward the undermost ear; (4) reversal of direction with opposite positioning; (5) fatigability with repetition of the maneuver.

Barany [1] initially described BPPV in 1921 and suggested a macular pathology as its cause. In 1952, Dix and Hallpike [6] reported 100 cases and supported the macular origin of the disorder by finding degeneration of hair cells and the ototholic membrane of the utricular macula at necropsy in a patient with known BPPV. This early etiologic hypothesis was then abandoned when it was shown experimentally that no lesion or selective stimulation of the macula could induce nystagmus [11].

In 1969, Schuknecht [18] described post-mortem basophilic deposits on the cupula of the posterior semicircular canal in two patients with BPPV. The size of these deposits exceeded that found in normal temporal bones, giving support to otothith degeneration products as their origin.

The “cupulolithiasis” theory assumes that debris detached from degenerating utricular macula settles by gravity on the cupula of the posterior semicircular canal (which is located just below the utricle with the head in an upright position). Hence, the cupula acquires a higher specific gravity than the endolymph and becomes sensitive to the influence of gravity during changes in head position.

In recent years, several arguments have been raised against the theory of cupulolithiasis [3]. In particular, this condition should imply a long duration for positional nystagmus and vertigo, similar to that induced by alcohol ingestion. On the contrary, a brief positional nystagmus is elicited by rapid head movements.

During surgery for occlusion of the posterior semicircular canal in two cases of BPPV, Parnes and McClure [16] observed loose particles within the long crus of the canal. Following this report, the theory of “canalolithiasis” was proposed to explain BPPV: the loose particles described would be displaced during positioning maneuvers, inducing an ampullofugal endolymph flow in the posterior semicircular canal and causing typical BPPV attacks. Such a theory would explain all the features of paroxysmal nystagmus, including its pathogenesis from a rapid positioning maneuver and its short duration.

To date, physical therapy for BPPV has achieved the best clinical results. An attempt to adapt the central nervous system to abnormal stimulation coming from the vestibular end-organ was initially suggested by Cawthorne [4]. More recently, Norrè [13] has used vestibular habituation training (VHT) to achieve a good outcome in patients with BPPV.
Subjects and methods
From September 1993 to July 1994, we examined 30 patients with BPPV. There were 12 men and 18 women ranging in age from 25 to 73 years (mean 49 years).

Diagnosis of BPPV was based upon typical history and demonstration of positional nystagmus and vertigo with the Hallpike maneuver. Frenzel’s glasses were worn by all patients.

The time interval between the onset of BPPV and treatment with EM varied from 5 to 180 days, with a mean of 28 days.

Etiologies of BPPV were idiopathic (15 cases), head injury (two cases), neurotological (two cases), chronic suppurative otitis media (one case) and otosclerosis (one case). Nine cases were associated with one or more of the following cardiovascular risk factors and/or pathologies: arterial hypertension, myocardial infarction, diabetes, hyperlipidemia.

When diagnosis was established and the affected side identified by means of the positioning maneuvers, each patient was treated with EM. The patient was placed seated with legs stretched in a horizontal position and head turned 45° toward the affected side (step 1). The patient was then placed quickly in a provocative head-hanging position and remained in this position for 2 min (step 2). Next, the patient was rotated slowly, over the course of 1 min, onto the opposite (unaffected) side, with head and body turned 45° downward, and was kept in this new position for 2 min (step 3). Finally, the patient was returned smoothly to the seated position with the head turned 45° toward the unaffected side, and was kept in this position for a further 2 min (step 4).

Following this, the patient was re-examined with the Hallpike maneuver to detect any residual vertigo or nystagmus. In the two cases in which this was found, EM was repeated. All patients were requested to avoid sudden head movements and keep a straight head position for 2 days after treatment, as well as to avoid sleeping on the affected side in order to prevent oto- lithic debris from returning into the posterior semicircular canal. Two days after treatment, all patients were checked again with the Hallpike maneuver.

Unlike the original technique described by Epley, bone vibration was not used to facilitate detachment of debris from the wall of the posterior semicircular canal. Also, no antivertigo drug was given as premedication before therapy. Since all patients with BPPV were treated with EM, a valid control group of untreated subjects was not available for comparison.

Results
After re-examination with the Hallpike maneuver 2 days after treatment, patients were classified into two categories: (1) those free of positional vertigo and nystagmus, and (2) those with unchanged positional vertigo and nystagmus.

From the total group of 30 patients, 26 (87%) were considered to be cured, while four (13%) were unchanged. In no case was only partial clinical improvement found.

The four patients unresponsive to EM were then treated with a Sémont maneuver (SM), which involved a quick swing of the lying subject from the affected side to the opposite [19]. This was successful in two cases and had no effect in the other two.

All patients were examined 2 months after treatment. At this time three of the patients free of positional vertigo and nystagmus 2 days after treatment were found to have BPPV of the ipsilateral horizontal semicircular canal.

Table 1 summarizes findings during EM. Only one patient had both vertigo and nystagmus in all positions during EM. However, all patients developed rotatory vertigo with typical nystagmus during step 2. In step 3 (head turned 45° downward toward the unaffected side), 20 patients showed no vertigo or nystagmus, while five developed vertigo with rotatory nystagmus that beat toward the affected side. The remaining five patients developed only minor vertigo but without nystagmus.

Of the 5 patients with vertigo and nystagmus during step 3, four were considered to be cured at re-examination after 2 days, while one remained symptomatic.

At during step 4 of EM (i.e., on return to the seated position), 20 patients had no nystagmus or vertigo, while six developed rotatory nystagmus beating toward the affected side and four had only minor vertigo. Five of the six patients with vertigo and nystagmus during this last stage were considered to be cured at re-examination.

In our current study group there were no statistically significant correlations between success rate of EM and patients according to gender, age, duration of symptoms and etiology. In particular, duration of symptoms showed a clear absence of correlation with clinical outcome.

Discussion
The typical signs and symptoms of BPPV are now believed to be produced by an ampullofugal deflection of the cupula of the posterior semicircular canal as the result of cupulolithiasis or canalolithiasis. However, it is not possible to ascertain its pathogenesis from the usual signs and symptoms.

The therapeutic maneuver developed by Epley [7, 8] is based upon a hypothesis assuming the presence of free-floating oto- lithic debris within the long crus of the posterior semicircular canal.

When an affected patient is placed in a vertical plane with the cupula at the top (as in Hallpike positioning), after a brief latency period oto- lithic debris reaches the most dependent part of the canal under the influence of gravity.