HOW TO DO IT:

exercise

away vertigo

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INTRODUCTION

Vertigo generally has a good prognosis because:

- many forms of vertigo have a benign cause and are characterized by spontaneous recovery of vestibular function, or central compensation of a peripheral vestibular tone imbalance; and;
- most forms of vertigo can be effectively relieved by pharmacological treatment, surgery, psychotherapy, or physical therapy.

Physical therapies include exercises for vestibular rehabilitation, promotion of central compensation of an acute vestibular loss, habituation for preventing motion sickness, and improvement of balance skills in the elderly. The most important exercises are those that fall within the scope of this article: deliberate manoeuvres for positional vertigo (Brandt 1999).

BENIGN PAROXYSMAL POSITIONAL VERTIGO

Benign paroxysmal positional vertigo (BPPV) was initially defined by Bárány (1921). It is the most common cause of vertigo, particularly in the elderly. By the age of 70, about 30% of all elderly people have experienced BPPV at least once. This condition is characterized by brief attacks of rotatory vertigo and concomitant positional rotatory-linear nystagmus, provoked by rapid changes in head position relative to gravity.

Patients with typical BPPV report attacks of rotatory vertigo, postural imbalance, and sometimes nausea precipitated by the following manoeuvres:

- sitting up from a supine position (particularly after awaking in the morning);
- when first lying down in bed;
- turning over in bed from one side to the other;
- extending the neck (head) to look up or get something from above;
- flexing the neck (head) when bending over.

BPPV in the supine position is very disturbing. It makes patients afraid of falling backwards, an almost unique complaint. In the upright position, vertigo attacks produced by changes in head position are incapacitating. They can also be dangerous, for example when a sufferer looks up at the ceiling while standing on a ladder.

BPPV is a mechanical disorder of the inner ear in which the precipitating positioning of the head causes abnormal stimulation (Fig. 1), usually of the posterior semicircular canal (p-BPPV) of the undermost ear, less frequently of the horizontal (h-BPPV) and the anterior (a-BPPV) semicircular canal. The observation of positional nystagmus provides the definitive diagnostic criteria for typical p-BPPV. They include:

- latency: vertigo and nystagmus begin one or more seconds after the head is tilted toward the affected ear and increase in severity to a maximum;
- duration less than 40 s: nystagmus gradually lessens after 10–40 s and ultimately abates, even when the precipitating head position is maintained;
- linear-rotatory nystagmus: the nystagmus is best seen if the patient wears Frenzel's glass-

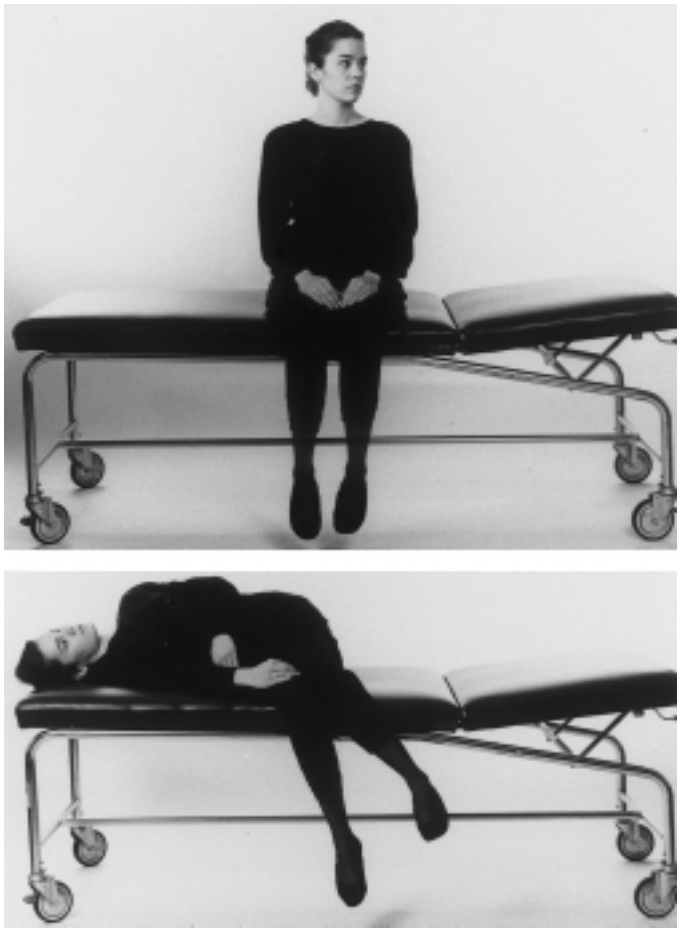

Fig. 1 Positional exercises for effective physical therapy for BPPV as proposed by Brandt & Daroff (1980). Patients are instructed to sit and then to move rapidly into the challenging position, to remain in that position for at least 30 s, and then to sit up for 30 s before assuming the opposite head-down position for 30 s. These exercises are repeated serially 5–10 times a day.

es (for example, lenses + 16 dpt), which prevent suppression by fixation. The nystagmus is linear-rotatory, with the fast phase beating toward the undermost ear or upward when gaze is directed to the uppermost ear;

- reversal: when the patient returns to the seated position, the vertigo and nystagmus may re-occur, but less violently and in the opposite direction;
- fatigability: constant repetition of the manoeuvre will result in ever-lessening symptoms.

These five criteria are crucial for further discussion of the confusing literature on the mechanism of BPPV. They provide the major arguments to prove or disprove any hypothetical explanation of cupulolithiasis or canalolithiasis as the causative factor.

FLOATING OTOCONIA IN THE LABYRINTH, CANALOLITHIASIS

It is now generally accepted that debris floats freely within the endolymph of the canal ('canalolithiasis'). The debris – possibly particles detached from the otoliths – congeals to form a free-floating clot or plug. Because the clot is heavier than the endolymph, it will always gravitate to the most dependent part of the canal during changes in head position that alter the angle of the cupular plane relative to gravity. Analogous to a plunger, the clot induces bidirectional (push or pull) forces on the cupula, thereby triggering the BPPV attack. Canalolithiasis explains all the features of BPPV (Brandt & Steddin 1993; Fig. 2):

- a latency of a few seconds (time needed for the clot-induced flow mechanism to develop by gravitational force);
- the ineffectiveness of a very slow positioning manoeuvre (then the clot would slowly gravitate along the undermost wall of the canal without affecting the cupula);
- the short duration of the positional vertigo/nystagmus (cupula deflection due to elastic restoring force ends when the heavy clot reaches its lowest position in the canal with respect to the earth's surface);
- the fatigability with repetitive provocation (explained by dispersion of single particles from the clot, which decreases the plunger effect);
- the reactivation of the vertigo after prolonged bedrest (the result of a new clot being formed by the particles);
- the direction of the nystagmus during the positioning manoeuvres (explained below).

PRECIPITATING POSITIONING MANOEUVRES CAUSE BOTH THE MALAISE AND ITS RELIEF

The positional exercises proposed in 1980 (Brandt & Daroff 1980) were the first effective physical therapy (Fig. 1). They were a sequence of rapid lateral head/trunk tilts, repeated serially to promote dispersion of the debris toward the utricular cavity. We instructed the patients to sit; to then move rapidly into the challenging position to induce the correct plane-specific stimulation of the posterior semicircular canal; to remain in that position until the evoked vertigo subsided, or for at least 30 s; and then to sit up for 30 s before assuming the opposite head-down position for an additional 30 s. Troost & Pat-

Fig. 2 Schematic drawing of the Semont liberatory manouevre in a patient with typical (posterior canal) BPPV of the left ear. Boxes from left to right: position of body and head, position of labyrinth in space, position and movement of the clot in the posterior canal and resulting cupula deflection, and direction of the rotatory nystagmus. The clot is depicted as an open circle within the canal; a black circle represents the final resting position of the clot. Panel 1: In the sitting position, the head is turned horizontally 45° to the unaffected ear. The clot, which is heavier than endolymph, settles at the base of the left posterior semicircular canal. Panel 2: The patient is tilted approximately 105° toward the left (affected) ear. The change in head position, relative to gravity, causes the clot to gravitate to the lowermost part of the canal and the cupula to deflect downward, inducing BPPV with rotatory nystagmus beating toward the undermost ear. The patient maintains this position for 3 min. Panel 3: The patient is turned approximately 195° with the nose down, causing the clot to move toward the exit of the canal. The endolymphatic flow again deflects the cupula such that the nystagmus beats toward the left ear, now uppermost. The patient remains in this position for 3 min. Panel 4: The patient is slowly moved to the sitting position; this causes the clot to enter the utricular cavity. Abbreviations: A, anterior semicircular canals; P, posterior semicircular canals; H, horizontal semicircular canals; Cup, cupula; UT, utricular cavity; RE, right eye; LE, left eye. (From Brandt *et al*. 1994.)

Complete recovery after a single manoevre is achieved in 50–70% of cases.

ton (1992) reviewed and diagrammed this exercise protocol. The Semont (Semont *et al*. 1988) and Epley (1992) liberatory manoeuvres require only a single sequence. Thus, they are preferable to the multiple repetitions over many days required by the Brandt-Daroff exercises. Now that canalolithiasis has been established as the mechanism of BPPV, we can explain the efficacy of the therapies according to anatomic and physical principles.

Figure 2 illustrates the Semont manoeuvre in a patient with typical (posterior canal) left-sided BPPV. The clot causes no deflection of the cupula in the upright position. When the patient is quickly tilted toward the affected left ear with 45° head rotation to the right (moving the left posterior canal to a plane corresponding to the plane of the head tilt), the clot gravitates toward the lower part of the canal, causing the cupula to deflect downward (ampullofugal), so triggering a typical BPPV attack.

If the patient is swung toward the opposite right side with the nose down, the clot will gravitate downward, causing stimulation of the posterior canal of the affected left ear (now uppermost). If no vertigo and nystagmus are elicited, we gently shake the patient's head in this position; this sometimes seems to facilitate settlement of the clot. The patient is then slowly moved to the upright posi-

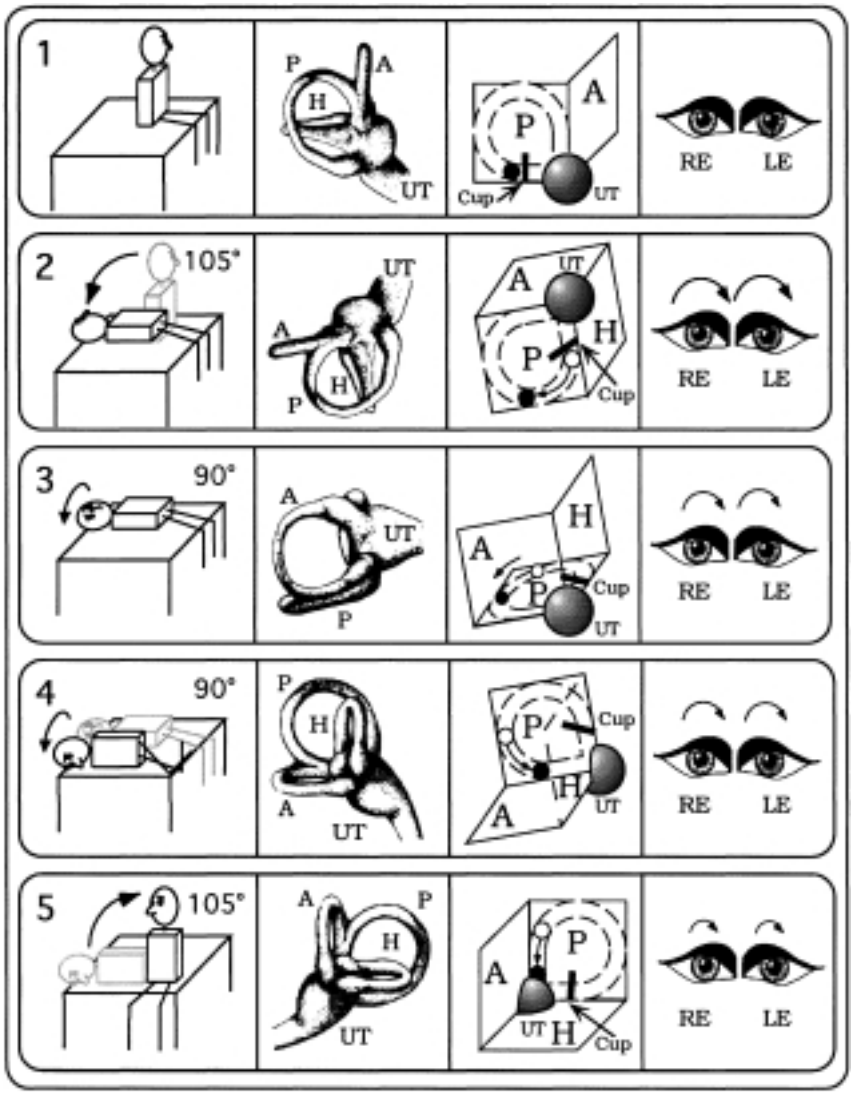

Fig. 3 Schematic drawing of the modified Epley liberatory manoeuvre. Patient characteristics and abbreviations are as in Fig. 2. Panel 1: In the sitting position, the head is turned horizontally 45° to the affected (left) ear. Panel 2: The patient is tilted approximately 105° backward into a slight head-hanging position, causing the clot to move in the canal, deflecting the cupula downward, and inducing the BPPV attack. The patient remains in this position for 3 min. Panel 3: The head is turned 90° to the unaffected ear, now undermost. Panel 4: the head and trunk continue turning another 90° to the right, causing the clot to move toward the exit of the canal. The patient remains in this position for 3 min The positional nystagmus beating toward the affected (uppermost) ear in positions 3 and 4 indicates effective therapy. Panel 5: The patient is moved into the sitting position. (From Brandt *et al*. 1994.)

tion; the clot will gravitate downward through the common crus of the posterior and anterior canals and enter the utricular cavity, where it becomes harmless. We share the experience of others (Serafini *et al*. 1996) that complete recovery after a single manoeuvre is achieved in about 50–70% of cases. Semont *et al*. (1988) recommended having the patient maintain the upright position for 48 h following the treatment, but we have not found this necessary.

Figure 3 illustrates the Epley manoeuvre (1992) as modified by Herdman *et al*. (1993) and others (Harvey *et al*. 1994) in a patient with typical (posterior canal) left-sided BPPV. The clot causes no deflection of the cupula in the upright position with the head turned horizontally 45° to the affected ear. When the patient is quickly tilted backward into a slight head-hanging position, the clot gravitates downward in the posterior canal, deflecting the cupula downward and inducing a BPPV attack. Rotation of the head and trunk toward the unaffected right ear causes further movement of the clot downward (ampullofugal) toward the exit of the canal, resulting in positional vertigo and nystagmus toward the affected (now uppermost) ear. The final uprighting of the patient causes the clot to enter the utricular cavity, where it becomes harmless.

Following effective physical liberation, approximately 50% of patients (Baloh *et al*. 1987) experience a recurrence of attacks; 10% to 20% occur in the first two weeks (Herdman *et al*. 1993). The recurrences may be due to re-entry of the debris into the posterior canal from the utricular cavity and should be treated with the same manoeuvre that induced resolution of the initial episode.

The process illustrated in Figs 2 and 3 explains the seemingly paradoxical observation (Semont *et al*. 1988) that the final liberatory positioning with the affected ear uppermost (Fig. 2, panel 3; Fig. 3, panel 4) induces nystagmus that beats toward that ear (Brandt & Steddin 1993). As described above, cupulolithiasis predicts an ampullopetal deflection of the cupula that would cause nystagmus to beat toward the undermost ear, whereas in canalolithiasis, the clot-induced endolymphatic flow causes ampullofugal deflection of the cupula and nystagmus beating to the uppermost ear.

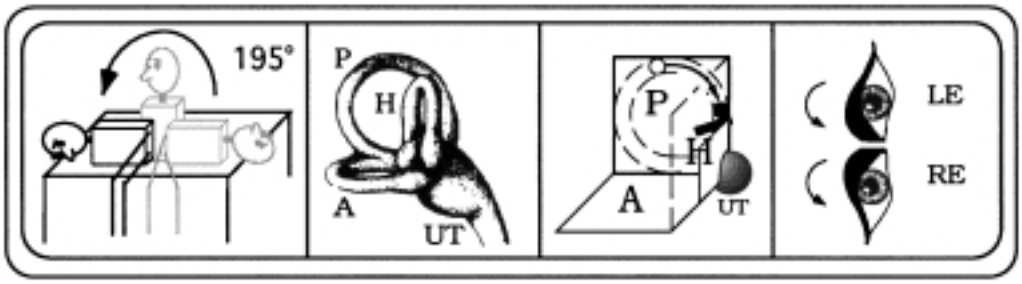

Fig. 4 Schematic drawing of an ineffective liberatory maneuver to be compared with Fig. 2, panel 3. After the patient is tilted to the right, the clot migrates back toward the cupula. Endolymph flow causes an ampullopetal cupula deflection with the nystagmus beating downward toward the unaffected ear. This indicates that the liberatory manouevre has failed. (From Brandt *et al.* 1994.)

Moreover, the upward direction of the nystagmus induced by the final positionings is a clinically relevant observation in that it provides reasonable certainty that the clot has exited the canal (or will so exit in the modified Epley manoeuvre) and the patient will be free of symptoms ('liberated'). If the nystagmus does not beat upward toward the affected ear, the clot is probably still inside the canal; if the nystagmus beats downward toward the unaffected ear (Fig. 4), the clot must have moved toward the cupula, causing an ampullopetal deflection. In either situation, the procedure should be repeated. If the nystagmus fails to beat upward following the second procedure and the BPPV persists, we schedule a return visit for the same manoeuvre. If the second session fails, we try a different liberatory manoeuvre (i.e. modified Epley, if we first used Semont, or vice versa). If both liberatory manoeuvres fail, we prescribe Brandt-Daroff exercises.

NATURAL COURSE

The natural history of BPPV is considered benign because it resolves spontaneously within weeks or months in most patients. However, in about 20–30% of the patients the condition persists when untreated, and it recurs in another 30% after variable periods for years.

SURGICAL PROCEDURES

In those rare patients who do not respond even to appropriate and prolonged physical therapy, surgical plugging of the posterior semicircular canal via a transmastoid approach or surgical transection of the posterior ampullary nerve via a middle ear approach can be considered.

In our experience with more than 1000 patients with typical BPPV, very few failed to respond to physical therapy, and ultimately required selective surgical transection or canal plugging. We believe that surgical intervention is still too frequently performed, before all possibilities of physical therapy are completely exhausted. This view is shared by Epley (1995), who has invented his own effective liberatory procedure. He believes that the disability ensuing from multiple, unpredictable recurrences is over the long term a more common indication for surgery.

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