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# Curing the BPPV with a Liberatory Maneuver

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## *Introduction*

From 1897, when Adler first described paroxysmal vertigo, to 1952 with the well-known 'Hallpike maneuver' [1], then, later, with studies of nystagmus by Katsarkas and Outerbridge [2], Baloh et al. [7], and Stahle and Terrins [6], benign paroxysmal positional vertigo (BPPV) has been well known.

Even if there are still discussions about the cause, our preoccupation was with the treatment. No medical treatment proved to be effective. We know that attacks spontaneously disappear mostly in 1 month. But recurrence comes 1 year later after the primary attack and afterwards it may recur every 3 months and sometimes never disappears. This means that medical treatment is a psychological security. Surgical treatment was proposed in 1974 by Gacek [3] with good results but with the possibility of hearing loss after surgery.

In 1979, Norre and Dveerd [4] suggested applying to BPPV patients what we had already carried out in other patients with vestibular diseases, since 1968 with the collaboration of Sterkers [8]: vestibular habituation training. Two hypotheses of what generates BPPV topped all others: (1) cupulolithiasis, as described by Schuknecht [5]; (2) heavy material floating in the endolymph, as suggested by Hall, MacClure et al.

In our opinion, these two considerations are complementary, leading to the same mechanical disturbance of the cupula of the posterior semicircular canal. In these conditions the density of the cupula is modified and then acts as an otolithic system under gravity.

One of us (A. Semont) suggested a maneuver that would free the cupula using the addition of the pressure of the endolymph and the inertia of the

'heavy materials'. The results were so extraordinary and unbelievable that nothing was said except allusions to the facility of curing BPPV [8].

Then Brandt and Daroff suggested a therapy with the idea of habituation training performed by the patient himself. In their published results, one could read that about one third of the patients were cured after one maneuver. That confirmed our astonishing results after only one maneuver.

It is this maneuver, which has been performed on over a thousand patients, that we present here.

## *Cases and Method*

Among the patients treated, only 711 of them have been correctly recorded. Most of these cases were idiopathic and some were post-traumatic. Age range was between 55 and 60 years; 64% of the patients were female. Post-traumatic patients were mainly between 20 and 45 years and the idiopathic patients were from 45 to 85 years old.

No hearing problems were related to BPPV. Caloric tests showed such abnormalities as : slightly reduced or increased values (sick ear). When EOG was done one month after therapy, everything was back to normal (sick ear).

In our opinion the main characteristics of BPPV are: (1) the nystagmus is, when observed on a well-centered eye in orbit, of a rotatory type, rolling toward the lower ear; (2) the nystagmus is inverted when the patient is brought back to the primary position (orthostatism); (3) the provoked nystagmus stops after 30 s maximum, this being correlated with the decrease in the vertigo as felt by the patient.

## *The Maneuver*

The patient is laid on the ipsilateral side to the sick ear with his head slightly declined. The nystagmus can appear: in this condition one must wait until it stops. If nothing happens the head is turned 45° facing up in order to have the cupula in a perpendicular plane to gravity. In this position, after a variable latency, the paroxysmal rotatory nystagmus rolling toward the examination table appears. One waits until it has completely stopped and then the patient is left in this position for 2 or 3 min.

Then, holding patient's head and neck with two hands, he is *swung* quickly to the opposite side. The speed of the head must be zero at the very moment the head touches the examination table. Then a rotatory nystagmus appears *still* rolling toward the sick ear which is now the *higher* one. *It must not be an inverted nystagmus.* The nystagmus is slightly different: wide amplitude, slower frequency, not so paroxysmal as the original one.

If nothing happens the head is slowly turned nearly to 90° facing up and then quickly turned to 45° facing down. Then the nystagmus occurs. The patient must stay in this last position for at least 5 min and is brought back to orthostatism very, very slowly.

The patient is then asked to keep his head absolutely vertical in space during at least 48 h day and night. He is asked to avoid fast head movements upward or downward and

not to sleep on the vertigo-generating side for a week. If the maneuver is not successful, it is performed again a week later.

## *Results*

After 8 years of practice we have 83.96% positive results with one maneuver, 92.68% positive results with 2 maneuvers, recurrence 4.22%.

Some European teams to whom the maneuver has been correctly taught have similar results: R. Boniver (Belgium); J.P. Demanez (Belgium) 90% with 2 maneuvers; G. Guidetti (Italy) 90% within 3 maneuvers; A. Hadj Djilani (Switzerland) 86%; R. Hausler (Switzerland) 84%; J. and C. Robert (France) 95% with 2 maneuvers.

## *Discussion*

The maneuver obviously works. The results are reproducible in other hands than the authors' and are of scientific value. The results have nothing to do with the average time of spontaneous disappearance of the attacks. Some patients had suffered from BPPV for 20–30 years and were cured with one maneuver.

The results have nothing to do with habituation either, because when the maneuver is carried out on normals nothing can be seen or recorded. The results confirm the hypothesis of the cupula being modified in its density, but cannot discriminate between cupulolithiasis and floating substances.

The fact that patients not suffering from vertigo (10% of idiopathic cases of BPPV) sometimes complain of a 'floating sensation' when lying on the side — manifested by a small downward nystagmus that persists and does not revert with orthostatism — might suggest another hypothesis for the etiology of BVVP: i.e. biomechanical dysfunction of the cupula due to variations of pressure between perilymph and endolymph.

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