BENIGN PAROXYSMAL POSITIONAL VERTIGO OF THE HORIZONTAL CANAL: A FORM OF CANALOLITHIASIS WITH VARIABLE CLINICAL FEATURES

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Abstract—Benign paroxysmal positional vertigo of the horizontal semicircular canal (HC-BPPV) is a well-defined syndrome characterized by direction-changing horizontal positional nystagmus. We report the clinical features of 5 patients who illustrate the possible variables of the syndrome. In most cases, nystagmus is geotropic and more intense when the pathological ear is lowermost; less often the syndrome presents with apogeotropic nystagmus that is more intense when the affected ear is uppermost. The nystagmus pattern may vary in time in the same patient, changing from apogeotropic to geotropic even in observations at short intervals. In some patients, the features indicate involvement of more than one canal, either simultaneously or in succession. It is sustained that the clinical findings can be explained by movement of endolymph caused by displacement of otoconia in the semicircular canals and that variants are due to different positions of the otoconia within the canals.

Keywords—horizontal semicircular canal; benign positional vertigo; paroxysmal nystagmus; canalolithiasis.

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most frequent vestibular syndrome. In 1969, it was commonly known as “cupulolithiasis.” Schuknecht (1) postulated that the disorder was caused by a deposit on the cupula of the posterior semicircular canal (PC) that made the cupula sensitive to gravitational forces. In 1979, Hall, Ruby, and McClure (2) suggested that the pathogenetic mechanism of BPPV was due to “something” moving inside the canal rather than adhering to the cupula of the PC. This theory, known as “canalolithiasis,” was sustained by the observations of Parnes and McClure (3), who found many free-floating particles in the PC of patients undergoing surgical occlusion of the canal because of incapacitating BPPV. Theoretical considerations of the features of nystagmus during therapeutic maneuvers in most cases support the canalolithiasis theory (4–7).

In 1985, it was reported that BPPV might originate from the horizontal semicircular canal (HC) (8). The features of HC-BPPV are such that the pathogenesis can be explained on the basis of particles or a viscous plug moving in the posterior part of the HC (9). In 1989, we reported a series of 15 patients with HC-BPPV and described the clinical features of paroxysmal geotropic nystagmus, observing the possibility of a variant characterized by apogeotropic nystagmus (10). Three patients with persistent direction-changing nystagmus consistent with debris leaving the PC and becoming attached to the cupula of the HC were
recently described (11). Today, HC-BPPV is recognized as a well-defined entity (12,13), although controversy about its pathophysiology is occasioned by certain features of the paroxysmal nystagmus and throws doubt on the type of material responsible for the disorder (14,15). Unlike for the PC form, no liberatory maneuver has been found, but recent therapeutic solutions have had encouraging results (16,17).

Since 1989, many other cases of HC-BPPV have indicated that the syndrome is more frequent than we supposed (18). Here we describe certain clinical characteristics that have recently emerged, discussing them in the light of recent pathogenetic hypotheses.

Case Reports

The authors have now diagnosed a total of 123 cases of HC-BPPV (75 women and 48 men, ranging in age from 25 to 88 years, mean age 55 years). No spontaneous (central gaze, seated position) or gaze nystagmus was detectable in any subject. Saccades, pursuit, and neurological examination were normal. Frenzel glasses were used to test for positional nystagmus in semidarkness. In many cases, especially when vertigo and nystagmus persisted or showed atypical features, magnetic resonance imaging (MRI) was performed to exclude CNS lesions. We chose 5 patients who can be regarded as paradigmatic in terms of nystagmus pattern and course of symptoms. These cases raise interesting considerations on the possible clinical variables of this disorder.

Case 1. Right Horizontal Canal BPPV with Geotropic Paroxysmal Nystagmus

A 44-year-old man had experienced bouts of positioning vertigo for about a week 2 years ago. Vestibular examination, performed when he was symptom-free, was normal. He was later referred to us after 3 days of severe vertigo triggered by rolling over in bed. The symptoms forced him to lie supine. There were no symptoms while standing or walking, except for unsteadiness especially during head movements. He had slight but constant nausea.

Nystagmus. A few seconds after moving from sitting to supine position, a small, horizontal, left-beating nystagmus, inhibited by fixation, was observed. Two seconds after rolling the head onto the right ear, a strong, horizontal, right-beating (geotropic) nystagmus was observed. This was paroxysmal, that is, with an initial, extremely rapid increase in frequency and a subsequent progressive decline in frequency. After about 30 seconds it ceased, and after about 50 seconds it reversed its direction to become apogeotropic (to the left). This time it was not paroxysmal; it was inhibited by visual fixation and persisted for about 2 min, declining very slowly. Horizontal left-beating (again geotropic) nystagmus was observed a few seconds after the patient’s head was rotated to the left side. It was paroxysmal but not as strong as the geotropic nystagmus on the left side. After about 40 seconds, another reversal was observed; it consisted of a few beats of long period to the right for about 30 seconds (Figure 1). Severe autonomic symptoms prevented repetition of the maneuver. The patient was told to lie on his left side for several hours and to come back 2 days later. At that stage he was symptom-free, and no positioning nystagmus was observed.

A caloric test showed 30% right canal paresis. Pure tone audiometry and auditory brainstem response were normal.

Comments. This case is a typical example of HC-BPPV with geotropic paroxysmal nystagmus. We found this kind of positioning nystagmus in about 80% of patients with HC syndrome. The features of the nystagmus suggest that heavy material (presumably otocoonia) located in the posterior part of the right HC moves towards the right ampulla when rotation of the head places the right ear lowermost (excitatory nystagmus) and away from the ampulla when right ear is uppermost (inhibitory nystagmus) (Figure 2). In this case, we have identified the right ear as the affected ear, because rolling the head so that the right side is downward produces a stronger geo-
tropic nystagmus than when the left side is downward. Caloric testing showed slight right canal paresis and was therefore useful in confirming the affected side.

The change from sitting to supine position (with the head undeflected) caused a slight nystagmus, beating to the left. We consider that this nystagmus is due to movement of debris that gravitates to the lowest part of the right HC, causing inhibitory displacement of the cupula away from the utricle.

Secondary apogeotropic nystagmus appearing after the stronger (primary) geotropic nystagmus without any change in position was
Figure 2. Diagram of the mechanism of canalolithiasis of the right horizontal canal with geotropic nystagmus. (A) Patient in supine position with particles in the posterior part of the right HC. (B) When the patient rolls onto his right side, particles move towards the ampulla, producing a flow towards the utricle and triggering intense horizontal, right-beating, geotropic nystagmus. (C) When the patient rolls onto his left side, otoconia fall in the opposite direction, causing a flow away from the utricle and triggering left-beating, geotropic nystagmus, less intense than in B.
3.1 Horizontal Canal BPPV observed in more than half our cases and is probably due to mechanisms of sensory adaptation (19-21). Reversal of nystagmus occurring on either side, as in this case, is less usual.

Case 2. Right Horizontal Canal BPPV with Apogeotropic Nystagmus Changing to Geotropic

A 60-year-old woman had a 3-year history of mild hypertension, treated with antihypertensive drugs. She came to our attention after 5 days of slight nausea, unsteadiness of gait "like walking on pillows," and bouts of spinning vertigo when lying on her left side.

Nystagmus. No nystagmus was observed when the patient was moved from sitting to supine position. Rotating the head to the left side did not produce nystagmus. About 1 to 2 seconds after the patient's head was rotated to the right, an intense, horizontal, long-lasting, left-beating (apogeotropic) nystagmus was observed. When the head was rotated to the left again, a very intense, paroxysmal, right-beating (apogeotropic) nystagmus was observed, with severe autonomic symptoms that forced the patient to sit up. After an hour, she was well and the maneuvers were tried again. Rotation of the head to the right side from supine position provoked a strong, paroxysmal, right-beating (geotropic) nystagmus. Rotation to the left again caused geotropic, horizontal nystagmus, less intense than before. The patient was told to sleep on her left side and return for further tests in two days. When she returned she was free of symptoms. She refused the caloric test.

Audiometric examination showed mild symmetrical sensorineural hearing loss above 2000 Hz. Auditory brain stem response was normal.

Comments. In about 20% of our patients with HC-BPPV, we found apogeotropic paroxysmal nystagmus, as in case 2. The features of the case are compatible with displacement of the cupula towards the utricle when the head is rotated to bring the right ear uppermost and with displacement away from the utricle when the same ear is brought downward. In this case the otoconia are presumably located in the anterior part of the right HC and move in the canal in the opposite direction to that of the geotropic form (case 1). The result is that the more intense, excitatory nystagmus is apogeotropic, that is, it beats towards the right, affected, ear, which is uppermost, and that the less intense, inhibitory nystagmus (again apogeotropic) beats towards the left, healthy side (uppermost) (Figure 3). The absence of nystagmus on the first rotation to the left could be due to the fact that the debris was very close to the cupula. When the head was brought back to the left after it had been rotated to the right, the stronger apogeotropic nystagmus appeared, probably because the debris was now further from the ampulla and, on responding to gravity, exerted a greater hydrodynamic impulse on the cupula.

When tested 1 h later, the direction of the nystagmus changed: rotating the head to the right produced a strong geotropic nystagmus; rotating the head to the left produced a less intense geotropic nystagmus. This change of direction can be explained by displacement of the debris from the anterior to the posterior part of the canal during the maneuvers.

The apogeotropic nystagmus could be explained by otoconia attached to the cupula, but the subsequent transformation into the more common geotropic form suggests that the true cause was canalolithiasis. It is important to underline that when the nystagmus was apogeotropic it was more intense with the left ear down, whereas in the subsequent geotropic form, it was more intense with the right ear down.

Case 3. Left Horizontal Canal BPPV with Apogeotropic Nystagmus without Any Change in Direction

A 38-year-old woman (the wife of D.N.), who suffered from recurrent premenstrual migraine, experienced severe spinning vertigo on rotating the head while in bed the day after an hour-long operation at the dentist. She did not experience vertigo on rising, but had nausea all day, exacerbated by head movements. There was no instability.
Figure 3. Diagram of the mechanism of canalolithiasis of the right horizontal canal with apogeotropic nystagmus. (A) Patient in supine position with particles in the anterior part of the right HC. (B) When the patient rolls onto his left side, the particles fall towards or onto the cupula, triggering a strong right-beating, apogeotropic nystagmus. (C) When the patient rolls onto his right side, the particles move away from the ampulla and the cupula is deflected by suction in the same sense, producing left-beating, still apogeotropic nystagmus.
Nystagmus. Two days after the start of symptoms, no nystagmus was observed when the patient moved from sitting to supine position. A few seconds after rotating the head to the right, a horizontal, apogeotropic (to the left), paroxysmal nystagmus of medium intensity, lasting 15 to 20 seconds, was observed. When the head was rotated to the left, a stronger, paroxysmal but very slowly declining (2 to 3 min) right-beating (apogeotropic), horizontal nystagmus appeared. This was evident even without Frenzel glasses. Rolling the head back to the right side provoked paroxysmal nystagmus much more intense than the first time and again apogeotropic. Examination had to be interrupted because of vomiting. Three and 7 days later, the clinical picture was the same, with less intense vertigo. Contrast MRI (Gadolinium) was normal. Various liberating movements were unsuccessful, as were treatment with an antimigraine drug and baclofen. A month later, apogeotropic bipositional nystagmus still occurred, but was better tolerated. On the right side it was more intense and no longer really paroxysmal, with a progressive onset, a stable period, and a very slow decline (total duration more than 3 min). Visual fixation partially decreased the nystagmus. On the left side, the nystagmus had completely lost its paroxysmal character and was constant in frequency, but it was transient, dying away in about 100 seconds. Visual fixation completely inhibited this nystagmus (Figure 4). Starting from prone position, nystagmus was always apogeotropic. After 3 months, the symptoms and nystagmus subsided. After 13 months, there was another period of apogeotropic horizontal positioning nystagmus, this time only on the right side, less intense than the first time, and disappearing after a week. She now complains only of brief sensations of vertigo after certain head movements, but no nystagmus can be detected.

Caloric testing, performed when she was almost free of symptoms, revealed no asymmetries. Audiometric examination was within normal limits.

Comments. Case 3 is another example of apogeotropic nystagmus that does not transform into the geotropic form. We have observed only 4 cases of this type. The affected side was identified as the left in case 3 because the stronger, excitatory nystagmus beat towards the left. Audiometric examination and caloric testing did not help us to determine the affected ear. Resolution of symptoms was very slow, and there was a progressive decline in the paroxysmal character of the nystagmus. In such cases, otoconia may be attached to the cupula on the vestibular side, however, it is also possible that they may be trapped on the canal side in the ampulla region. It should be noted that after a month, the character of the nystagmus was quite different, principally due to the fact that it was no longer paroxysmal.

Case 4. Left Horizontal Canal BPPV with Geotropic Nystagmus Changing to Left Posterior Canal BPPV

A 51-year-old woman complained that for 3 days she had experienced very intense vertigo and vomiting when rotating the head in either direction in bed. She remained immobile in bed and was brought to the hospital by ambulance.

Nystagmus. When the patient was moved from sitting to supine position, a small, brief, right-beating nystagmus seemed to occur, detectable only with Frenzel glasses. A few seconds after rotating the head to the right, a strong paroxysmal, right-beating (geotropic) horizontal nystagmus was observed, lasting about 60 seconds. Rotation to the left caused an immediate, more intense, paroxysmal, left-beating (geotropic) horizontal nystagmus that did not reverse its direction and subsided in about 40 seconds. The patient was extremely frightened and anxious, and because of her fear of vertigo, the maneuver could not be repeated. She was instructed to lie on her right side for at least 12 hours. After 3 days she told us that the symptoms had changed: the vertigo occurred mainly during movements in the vertical plane (lying down and rising) and no longer during those in the horizontal plane. Further examination revealed a typical tor-
sional paroxysmal nystagmus provoked by the left Hallpike maneuver. A liberatory maneuver was performed and the patient was cured (no nystagmus at follow-up after 2 days).

The patient refused caloric testing. Tonal audiometry and auditory brainstem response were normal.

**Comments.** Case 4 is an example of transformation of HC-BPPV into PC-BPPV. We have observed this kind of transformation in 10 cases. It always occurred in a brief span of time without any symptom-free interval. In this case we identified the affected side as the left because the stronger geotropic nystagmus beat to the left. The side of the subsequent BPPV of the posterior canal was again the left, suggesting that the debris had moved from one canal to another because of movements of the patient or the diagnostic maneuvers.

**Case 5. Right Posterior Canal BPPV Changing to Right Horizontal Canal BPPV with Geotropic Nystagmus**

A 56-year-old woman with a 10-year history of mild hypertension treated with anti-hypertensive drugs complained of bouts of
spinning vertigo in the last year. The vertigo was sometimes provoked by lying down and getting up and sometimes by turning in bed. Six months earlier, right PC-BPPV had been diagnosed and cured with the Semont maneuver (22). In the 15 days before the present observation, vertigo was caused only by rolling over in bed. She had constant mild nausea and slight unsteadiness of gait.

**Nystagmus.** Positioning from sitting to supine position caused a small, low frequency, horizontal, left-beating nystagmus lasting more than 2 min and inhibited by visual fixation. Rotating the head to the right immediately produced a strong, paroxysmal, right-beating (geotropic), horizontal nystagmus that reversed in direction after about 30 seconds. This apogeotropic, secondary nystagmus was nonparoxysmal and long-lasting (about 2 min). A few seconds after the head was rotated to the left, a left-beating (geotropic), horizontal nystagmus was observed. This was less intense than the one on the right side and had a slow decline. Nystagmus was evident even without Frenzel glasses. We tried to treat the patient by having her lie on her left side for several hours, but after 15 days, the clinical picture was almost the same: only the secondary nystagmus disappeared.

Contrast-enhanced MRI showed small post-ischemic scars in the white matter and normal brainstem and cerebellum. Pure tone audiometry and auditory brainstem response were completely normal. Caloric testing, performed at the check-up after 15 d showed 70% right canal paresis. Six months later, the patient was still dizzy, though positioning nystagmus was less intense. After a year, she had only long-lasting, right-beating, geotropic nystagmus with a 5-second latency, inhibited by visual fixation, without associated vertigo. The subject showed signs of an anxiety-depressive syndrome.

**Comments.** Case 5 is an example of PC-BPPV that evolves into HC-BPPV with time. We have observed this transformation in about 20 patients, in some cases at a later date, in others during the same diagnostic session. In this case we identified the affected side as the right because the stronger nystagmus beat towards the right side. The patient had also had BPPV of the right PC, and caloric testing indicated right canal paresis. A year later, the syndrome had not resolved. Nystagmus was still geotropic but occurred only on the affected side; it was neither paroxysmal nor associated with vertigo.

**Discussion**

The 5 patients selected from a population of 123 cases can be regarded as paradigmatic for the purposes of this discussion. The high number of HC-BPPV diagnoses is probably due to the fact that almost all patients with vertigo in our area come to us and we examine them immediately in the acute phase. This permits us to diagnose this very transient syndrome. In all patients the possibility of a neurological cause was considered, but the benign clinical course and negative neurological examination were convincing evidence of a peripheral disorder. In the 2 patients (cases 3 and 5) in whom symptoms persisted in time, MRI was performed but failed to reveal signs compatible with lesions of the central vestibular system.

These 5 cases demonstrate that subjective symptoms are very helpful in distinguishing PC-BPPV from HC-BPPV: HC syndrome is characterized by very intense vertigo, mainly when the patient is supine and rolls to one side in bed. In the beginning, however, vertigo and autonomic symptoms may be so intense and triggered by the slightest movement, that patients report only spontaneous and not undiagnosed because vestibular examination is normal, could in fact be due to HC-BPPV, tested for in a nonactive period.

HC-BPPV is diagnosed on the basis of horizontal paroxysmal nystagmus provoked by rotation of the head or body to one side with the patient supine. The nystagmus is clearly observable, even without Frenzel glasses. It is very difficult to determine the effect of visual fixation: nystagmus is often so strong that it
is unlikely to be inhibited by fixation, and autonomic symptoms often prevent proper observation of its character. ENG recording, which is, in our opinion, of limited diagnostic utility in this kind of disorder, is also difficult to perform in these circumstances.

In most cases HC-BPPV is characterized by geotropic paroxysmal nystagmus: rotation onto the affected side causes a very intense nystagmus after a brief delay. This nystagmus beats towards the lowermost ear and generally lasts less than 60 seconds. In more than 50% of patients, this primary phase is followed by a less intense but longer lasting apogeotropic nystagmus (secondary nystagmus) without any further change in head position. Rotation to the other side causes a less intense, usually paroxysmal and longer lasting, geotropic nystagmus. If the latter is very intense, it too may be followed by a secondary phase (Figure 1).

This nystagmus profile is well explained by the presence of particles free to move inside the posterior part of the HC: rotation of the head to either side causes the debris to fall under gravity, towards or away from the ampulla, creating ampullopetal and ampullofugal displacement of the cupula, respectively, by a pump or suction mechanism, or by hydrodynamic drag (Figure 2). Our impression is that gravitation is an important factor in determining nystagmus, since we have sometimes observed very intense responses even with slow movements. This kind of geotropic nystagmus cannot be due to cupulolithiasis because a heavy cupula would move in the opposite direction, away from the utricle, causing nystagmus to beat towards the uppermost ear.

The excitatory nystagmus (primary phase) is stronger and lasts about twice as long as that observed in PC-BPPV. This is probably due to the greater velocity storage of the HC with respect to the vertical canals (15). Secondary nystagmus is also observed in about 50% of cases with PC-BPPV (23) and is probably a sensory adaptation (19–21). In the semicircular canal of the frog, mechanical stimulation of ampulla receptors determines a response in the vestibular nerve that resembles the temporal pattern of positioning nystagmus: at the onset of the excitatory stimulus (a microsyringe acting as plunger), there is an intense increase in ampullar nerve firing rate that soon attenuates and gives rise to a less intense but more prolonged undershoot before the stimulus is removed (24). The enhancement in nerve firing rate could correspond to the paroxysmal geotropic nystagmus and the undershoot to the secondary apogeotropic nystagmus.

The inhibitory nystagmus beating towards the healthy ear is less intense and may correspond to the nystagmus that appears in PC-BPPV when patients return to sitting position after the Hallpike maneuver. The difference in intensity is presumably due to the difference in excitation and inhibition of afferent nerve firing (Ewald's second law). Baloh and colleagues (15) sustain that the magnitude of slow phase velocity of positioning nystagmus is not consistent with this theory. They propose that amplitude asymmetry is due to the fact that the mass has a different distance to move in the canal.

In some patients, such as our cases 1, 4, and 5, the mere change from sitting to supine position (with head undeflected) caused a slight nystagmus beating towards the healthy side, mimicking in duration a spontaneous persistent nystagmus. We consider that this nystagmus is due to movement of debris that gravitates to the lowest part of the HC, causing inhibitory displacement of the cupula away from the utricle. The low intensity of the nystagmus may be due to the fact that the detritus moves only a short distance and because gravitation acts in a plane different from that of the canal. The long duration of this nystagmus is not so easy to explain.

In a smaller percentage of cases, HC-BPPV presents with apogeotropic nystagmus. This is again horizontal, paroxysmal, and beats towards the affected ear when this is uppermost. Apogeotropic paroxysmal nystagmus could be due to cupulolithiasis or to a different initial position of the particles inside the HC. In our opinion, in cases like case 2, in which apogeotropic nystagmus later becomes geotropic, the phenomenon is due to canalolithiasis. In these cases the otoconia are presumably initially located in the anterior part of the HC and move in the canal in the opposite direction with re-
spect to the geotropic form. Hence, rotation of the head to the affected side causes displacement of the debris away from the ampulla, and the resulting suction causes apogeotropic nystagmus, beating towards the healthy, uppermost ear. Rolling onto the opposite side causes the debris to return towards the ampulla, triggering more intense nystagmus, again apogeotropic, beating toward the affected, uppermost ear (Figure 3). The transformation of apogeotropic nystagmus into geotropic, observed in most cases, may be due to displacement of otoconia from the anterior part of the canal to the posterior part. This displacement may be occasional or determined by the diagnostic maneuvers.

In very few cases, like in case 3, this transformation was not observed. Case 3 resembles Baloh’s patients: the only difference is that initially case 3 had a very intense paroxysmal nystagmus. A month later it became nonparoxysmal. In these cases, cupulolithiasis is more probable, as suggested by Baloh and colleagues: otoconia could be attached to the cupula, probably on the vestibular side. However, the possibility that it may be the result of canalolithiasis cannot be excluded, since the otoconia may be situated close to the ampulla and somehow prevented from moving.

Whether nystagmus is apogeotropic or geotropic, it is always more intense on one side. In our opinion, this reflects ampullopetal stimulation in the affected HC, so that the affected side is indicated by the direction in which the stronger nystagmus beats. With geotropic nystagmus, the affected side is lowermost, and with apogeotropic nystagmus, it is uppermost. In some cases (cases 1 and 5), the caloric test can help to determine the affected side. This test cannot always be performed because of the intensity of the symptoms, but when it is possible, ipsilateral canal paresis may be found. Tonal audiometry and other audiological examinations generally do not help to diagnose the side of HC-BPPV.

Free particles can migrate from one canal to another, causing the transformation of HC-BPPV into PC-BPPV and vice versa, like in cases 4 and 5. This transformation may occur as a result of the diagnostic maneuvers or due to movements of the patients. It has already been reported that both forms may occur simultaneously (10,15). Epley (6) reported that HC-BPPV may be a complication of the “canalith repositioning procedure” devised by him to treat PC-BPPV. We agree with Epley that this is unequivocal proof that the two syndromes are caused by the same material, which may pass from one canal to another.

HC-BPPV is generally characterized by stronger nystagmus, vertigo, and autonomic symptoms, but the active phase is usually shorter than that of PC-BPPV. In many patients, the syndrome may escape diagnosis because examination is performed after the symptoms have resolved. However in some cases, like in patients 3 and 5, the course is atypical: there may be slow resolution with a progressive decline in paroxysmal character, or the nystagmus may completely lose its peculiar characteristics. In the chronic phase, we may observe positional nystagmus, which is very different from the paroxysmal nystagmus of the initial period and which may be interpreted completely differently, for example, as a manifestation of CNS pathology.

In most cases, PC-BPPV can be cured by liberatory maneuvers that displace the debris from critical areas. To our knowledge, the only therapeutic maneuver for HC-BPPV is that recently proposed by Vannucchi and colleagues. The method is very simple: the patient lies supine, facing straight ahead, then rotates the head or whole body to the healthy side and stays in this position for at least 12 hours. In this position, the floating particles can leave the canal. This treatment is suitable only for HC-BPPV with geotropic nystagmus, but it is very effective: 92% of our patients treated in this manner became symptom-free within 2 days. A maneuver that can be regarded as liberatory was recently proposed and has been successful in the few patients treated so far. We are currently testing this maneuver.

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