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High-Resolution Three-Dimensional Magnetic Resonance Imaging of the Vestibular Labyrinth in Patients with Atypical and Intractable Benign Positional Vertigo

Bruno Schratzenstaller[a] Carola Wagner-Manslau[b] Christoph Alexiou[a]
Wolfgang Arnold[a]

[a]Department of Otolaryngology, Klinikum rechts der Isar, Technical University of Munich, and
[b]Institute of Radiology and Nuclear Medicine, Klinikum München-Dachau, Dachau, Germany

Key Words
Benign paroxysmal vertigo · Atypical positional vertigo · High-resolution magnetic resonance imaging · Three-dimensional reconstruction

Abstract
Benign paroxysmal positional vertigo (BPPV) is a most common cause of dizziness and usually a self-limited disease, although a small percentage of patients suffer from a permanent form and do not respond to any treatment. This persistent form of BPPV is thought to have a different underlying pathophysiology than the generally accepted canalolithiasis theory. We investigated 5 patients who did not respond to physical treatment, presented with an atypical concomitant nystagmus or both with high-resolution three-dimensional magnetic resonance imaging of the inner ear. This method provides an excellent imaging of the inner ear fluid spaces. In all 5 patients, we found structural changes such as fractures or filling defects in the semicircular canals which we did not find in control groups. One patient clinically presented with the symptoms of a 'heavy cupula'. Whereas cross-sections through the ampullary region and the adjoining utricle showed no abnormalities, there were significant structural changes in the semicircular canals, which are able to provide an explanation for the symptoms of a heavy cupula.

Introduction

Many patients consult their physician because of dizziness or poor balance. Benign paroxysmal positional vertigo (BPPV) is probably the most common cause of vertigo [1] and the most common peripheral vestibular disorder [2, 3]. It is a positional vertigo of sudden onset, triggered by rapid changes of head and body position and with concomitant nystagmus of short duration. The most common provocative movements are lying down, rolling over in bed, bending over, straightening up and extending the neck upward [4].

In 1897, Adler [5] already presented a case report of a patient suffering from positional vertigo, and in 1921 Bárány [6] first described the symptom complex known as

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Dr. Christoph Alexiou
Department of Otolaryngology, Klinikum rechts der Isar
Ismaningerstrasse 22
D–81675 Munich (Germany)
Tel. +49 89 41402371, Fax +49 89 41405153

paroxysmal positional vertigo. Bárány discussed a pathology of the semicircular canals or the otolithic organs as the cause for the paroxysmal vertigo attacks and rotatory nystagmus he observed in his patient [6]. In 1952, Dix and Hallpike [4] coined the descriptive term 'benign paroxysmal positional vertigo' and their studies supported Bárány's theory that the pathophysiological mechanism of this disorder is located within the vestibular labyrinth.

In BPPV, free-floating debris of degenerative otoconia in the endolymph of a semicircular canal (canalolithiasis [7]) or debris that becomes adherent to the cupula of the semicircular canal (cupulolithiasis [8]) have been postulated. Recent investigations strongly support the theory that canalolithiasis may be the underlying pathophysiology in BPPV.

The modern understanding of BPPV started with Schuknecht's observation that this dysfunction results from the gravity-dependent movement of fixed dense material to the cupula of the posterior semicircular canal (cupulolithiasis) [9, 10]. However, Schuknecht's hypothesis is not consistent with all clinical features of the disease and the highly effective canalith reposition liberatory maneuvers for BPPV. Dix and Hallpike [4] described a method of maximally provoking the attack by moving the patient rapidly from a sitting position to a position with the head hanging with one ear downward and first defined all cardinal symptoms of vertigo and nystagmus of BPPV: (1) it is elicited in a critical provocative position with the affected ear down, (2) nystagmus is predominantly torsional with the fast phase beating towards the undermost ear, (3) it has latency of onset, (4) it is transient (usually less than 30 s), (5) it reverses when the head is returned upright and (6) its response declines on repetition of the provocative maneuver.

In 1995, Epley [7] defined the general criteria for the diagnosis of canalolithiasis in *any* canal by obligatory and facultative symptoms. Obligatory symptoms are (1) transient nystagmus, (2) latency of nystagmus, (3) nystagmus accompanied by severe vertigo and (4) provocation by head positioning. Facultative symptoms are (1) response decline with repetition of the provocative maneuver, (2) reversal of the nystagmus direction in the same plane upon repositioning of the head and (3) curability of vertigo and nystagmus by canalith repositioning maneuvers.

Based on histological evidence Schuknecht's cupulolithiasis theory attributed the clinical manifestations to abnormal, basophilic dense particles acting directly by attachment to or impingement on the cupula of the posterior semicircular canal [10]. Schuknecht believed that nystagmus occurs when the posterior semicircular canal is exposed to gravity by changes in head position, thus creating an ampullofugal displacement of the cupula, because the cupular deposits are denser than the endolymph. The posterior semicircular canal localization of BPPV could be histologically verified [11], but the concept of a 'heavy cupula' has been brought into question for several reasons: gravity-dependent deflection of a cupula would continue as long as the provocative orientation is maintained, thus evoking sustained nystagmus. In contrast, the typical nystagmus persists only for a maximum of about 30 s and then ceases. It also fails to account for the latency period before the onset of nystagmus and its fatigability upon repetition of the positioning maneuver.

Although BPPV has long been recognized, its underlying pathology is still a matter of discussion [12]. The modern understanding of the pathophysiology of BPPV started with the proposal of Epley [7], Hall et al. [13] and Parnes and McClure [14] that the dysfunction resulted from the accumulation of free-floating dense material in the posterior semicircular canal, creating a density differential between cupula and endolymph ('free-floating particle theory'). According to this theory these particles will move within the endolymph of the posterior semicircular canal whenever the head position is changed with respect to gravity. When performing the Dix-Hallpike maneuver, these free particles do not act directly on the cupula. Rather, they induce an endolymph flow resulting from their hydrodynamic drag as they gravitate to a more dependent position away from the cupula. The result is vertigo due to ampullofugal deflection of the cupula. In both the erect and supine positions, the posterior semicircular canal is the most dependent part of the vestibular labyrinth and thus detached sediment collects in its lumen. This would explain the high frequency of posterior canal disease [2, 12].

This condition has come to be called 'canalithiasis' or 'canalolithiasis'. These 'clots' or 'canaliths' [7] probably consist of utricular otoconia which have become dislodged from the macula secondary to infection, ischemia, surgery and for trauma or aging [15].

Histological proof was provided by Parnes and McClure [14], who directly observed an accumulation of free dense particles resembling displaced otoconia during a surgical intervention in the posterior semicircular canal of a patient suffering from BPPV.

Canalolithiasis is at present the generally accepted theory for the pathophysiology of BPPV as it is able to explain all phenomena occurring during the examination of

this sickness: The paroxysmal nature and latency of onset of the provoked nystagmus and vertigo are dependent on the time needed for gravity to set the canalith in motion. Usually a few seconds are required to overcome the inertia and resistance of the endolymph and the cupula before clot-induced endolymph flow leads to a cupular displacement [16]. The severity of the nystagmus is thought to be due to the magnitude of the displacement of the cupula [17]. The term 'paroxysmal' reflects an important and essential characteristic of the disorder: the vertigo is episodic rather than persistent.

The vertigo fatigues and the response of the concomitant nystagmus decline: with each repetition of the provocative maneuver, the clotted particles become more and more dispersed or trapped within another part of the vestibular organ, thus inducing a minor hydrodynamic drag on the cupula [7, 13]. Reactivation of vertigo and nystagmus may result from reformation of the dispersed material again forming a clot [2].

The limited duration results from the time needed until the canaliths stop gravitating when they reach the most dependent position in the posterior semicircular canal [16]. When the patient is returned to the upright position, the particles gravitate backwards in the posterior semicircular canal, causing an ampullopetal drag on the endolymph. The following cupular deflection causes torsional nystagmus in the reverse direction [7].

Four Factors Apparently Predispose to Paroxysmal Positional Vertigo

Advanced age in combination with inactivity, head and cervical spine trauma and concomitant ear diseases are predisposing factors [7]. In older patients, this effect is probably related to the shedding of otoconia from the utricular macula that begins with middle age. Especially in persons with sedentary lifestyles there seem not to be sufficient head and body movements to disperse the particles which collect in the posterior semicircular canal. The most frequent cause of BPPV however is idiopathic [3, 18].

BPPV is usually a self-limited disorder [19] but can also be severely incapacitating and last for months or even years. For this reason specific bedside maneuvers have been developed. Their purpose is to disperse the debris within the labyrinth (Brandt-Daroff exercises [1, 16], liberatory maneuver of Semont [20, 21]) or to relocate the particles floating within the canal back to the utricle through a sequence of head movements (canalith repositioning maneuvers [22–26]). In most cases there is a high success rate and instant and long-lasting relief.

Schuknecht and Ruby [9] have already described three forms of BPPV: self-limited, recurrent and permanent. The permanent forms do not respond to noninvasive treatment and are considered intractable [27]. Hall et al. [13] postulated that the self-limited form and the form with remissions and recurrences have a different underlying pathophysiology than the permanent form of this disorder.

There is also a small fraction of patients who complain of positional vertigo and demonstrate nystagmus that is coincident with the vertigo symptoms, but nystagmus is not typical (i.e. beating in another direction, horizontal, no fatigability etc.). Epley [7] classified these forms of positional nystagmus that do not fit all Dix-Hallpike criteria as 'atypical'. The most common cause seems to be free-floating debris in an atypical location in the semicircular canals [24, 28–30].

Although the canalolithiasis theory provides a useful theoretical working hypothesis, it sometimes fails to explain all clinical features in patients with intractable or atypical BPPV. The key in determining the diagnosis of BPPV is still the examination of the patient performing positioning maneuvers with or without the use of Frenzel glasses. The direction of the nystagmus is considered to be the most useful feature in determining the involved canal [28]. The differential diagnosis can be difficult when the patient also suffers from concomitant neurological diseases or there is a preceding ear pathology, as is the case in about 10–20% of BPPV patients [7, 18, 19]. Also more canals or both ears can be affected at the same time, creating a confusing diagnostic picture and making it impossible to provide an effective form of therapy.

The aim of this study has been to provide a new diagnostic approach to patients with therapy-resistant and atypical BPPV by three-dimensional reconstruction of the vestibular labyrinth using a high-resolution magnetic resonance imaging (MR) technique (HR-3D-MR). Our special interest has been whether – as already postulated by Hall et al. [13] – there are structural changes in the semicircular canals or the cupula, which could be identified in patients with incurable or atypical vertigo, compared to those with self-limited disease. Three-dimensional MR of the inner ear has been done by various groups with excellent visualization of the structures of the membranous labyrinth [31–33]. As HR-3D-MR provides a 'gapless imaging' [31], an obliteration of labyrinthine structures is depicted as a filling defect of the inner ear fluid spaces [34]. This method yields most reliable results in detecting intralabyrinthine tissue formations as labyrinthine fibrosis, traumatic lesions with partial obliterations, labyrin-

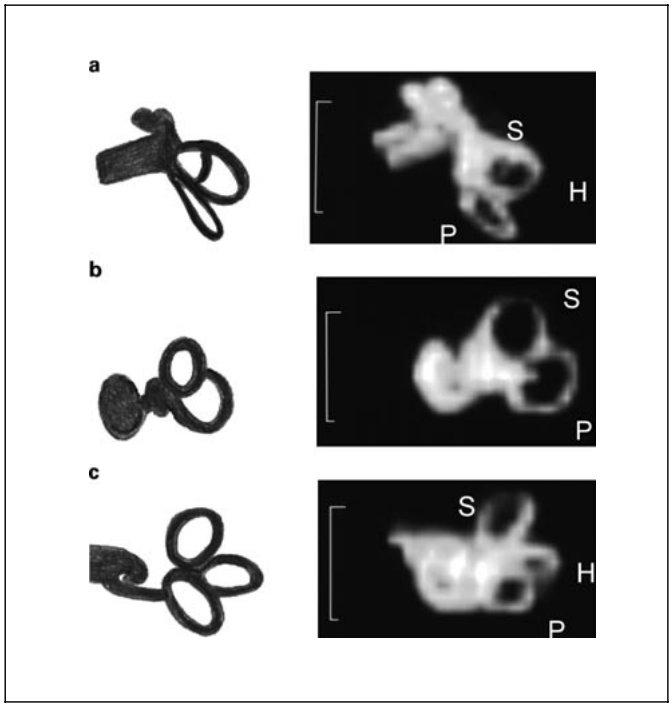

Fig. 1. Anatomical definitions and views of the three-dimensional reconstructions of the inner ear in three different planes. S = Canalis semicircularis superior (= ventral, = anterior); H = canalis semicircularis horizontalis (= lateral); P = canalis semicircularis posterior (= dorsal). All studies were reformatted in the same way. The inferior view (transversal) shows S/H/P (**a**), the lateral view (sagittal) S/P (**b**) and the anterior view (coronal) S/H/P (**c**).

thitis and malformations. HR-3D-MR for BPPV has been done by Jäger et al. [35], who found no pathological changes in the inner ear structures. Up to the present time, this is the first study dealing with HR-3D-MR of patients with atypical or incurable paroxysmal vertigo.

Subjects and Methods

In this study, 5 patients with atypical paroxysmal vertigo were clinically examined as described below and then underwent HR-3D-MR.

As control, we performed HR-3D-MR of both vestibular organs in 20 patients, 9 men and 11 women, aged between 55 and 68 years who underwent MR of the brain for other reasons than positional vertigo. Additionally, 10 patients, 6 men and 4 women, aged between 38 and 78 years with unilateral 'typical' BPPV had also been investigated with MR. Each patient of this group had the typical symptoms and signs of BPPV as described by Dix and Hallpike [4]. MR was performed within 3 days after diagnosis. In 1 patient, the symptoms disappeared spontaneously, the other 9 patients were treated with the Epley maneuver using vibration. Six patients were symptom-free after 1 session, 2 after 2 sessions and 1 patient needed 3 sessions. Within 2 weeks after diagnosis, none of those patients showed signs of vertigo or positional nystagmus.

For each subject the same data set comprising systematic series of axial MR images and three-dimensional data were obtained using a 1.5-tesla Signa HS whole-body MR system (GE, Milwaukee, Wisc., USA). The manufacturer's head coil was used for all acquisitions. The major concern of the diagnostic study was the identification of anatomical and pathological sites of the semicircular canals, and these data are optimally depicted on heavily T_2-weighted images on a three-dimensional data set which was reformatted afterwards (FSE T_2 3D Sequenz, TR 4,320 ms, TE 238 ms, slice thickness 1 mm, matrix 512 × 512, FOV 22 × 22, 1.0 NEX).

Besides three-dimensional reconstruction of the inner ear, we also performed cross-sections through the utricle in every patient.

Figure 1 shows the anatomical definitions of a three-dimensional reconstruction of a normal right inner ear in three different planes.

Results

In HR-3D-MR reconstructions of the membranous labyrinth the semicircular canals, vestibule, cochlea and the beginning of the 8th cranial nerve have always been completely visible. We found absolutely no morphological changes in the semicircular canals of the control groups with and without BPPV. Also cross-sections through the utricle showed no pathological changes. However in all 5 patients suffering from atypical paroxysmal vertigo, HR-3D-MR revealed significant structural abnormalities in the vestibular organ.

Patient 1

This 55-year-old female patient complained for 3 days of brief attacks of vertigo when turning her head in a lying position to the right side. A tympanoplasty type I had been performed on the *left* side 12 years ago. The Dix-Hallpike maneuver showed a severe counterclockwise torsional nystagmus when turning the head to the *right* side which lasted about 20 s. No therapy was necessary as the patient recovered spontaneously 6 days after the onset of symptoms. HR-3D-MR performed 2 days after diagnosis showed a 2.2-mm filling defect in the superior semicircular canal of the *left* side, with no pathological changes on the *right* side (fig. 2).

Patient 2

A 58-year-old woman had been given the diagnosis of Ménière's disease of the left ear 9 years previously. At that time she suffered from severe hearing loss and tinnitus of the left ear and recurrent attacks of vertigo lasting up to 4 h. For that reason she had been treated with a gentamy-

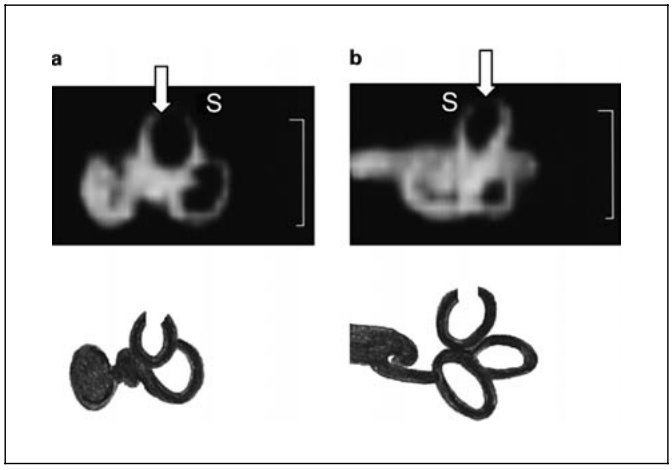

Fig. 2. Three-dimensional reconstructions of the inner ear of patient No. 1: 2.2-mm defect of the superior semicircular (S) canal (arrows). **a** Left lateral view. **b** Left anterior view.

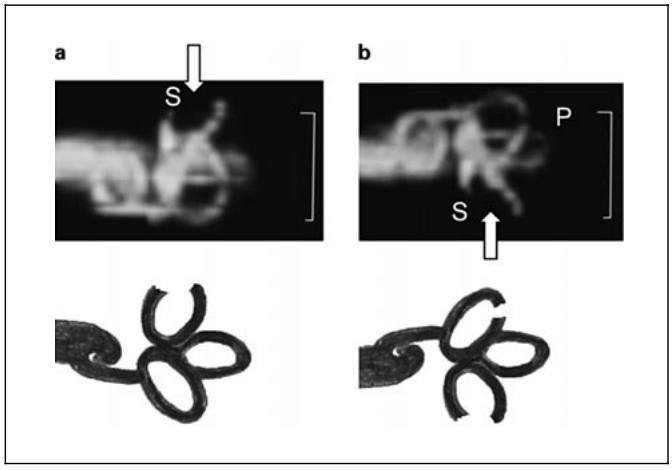

Fig. 3. Three-dimensional reconstructions of the inner ear of patient No. 2: 3.6-mm defect of the superior (S) semicircular canal (arrow), 1.7-mm defect of the posterior (P) semicircular canal (arrow). **a** Left inferior view. **b** Left inferior oblique view.

cin therapy, applied via the left tympanic membrane through a ventilation tube 6 years previously. After completion of the gentamycin therapy she suffered no more from Ménière attacks but developed positional vertigo when turning her head in a lying position to either side, which lasted ever since.

The Dix-Hallpike maneuver provoked a clockwise torsional nystagmus on the left side lasting for 25 s and a counterclockwise torsional nystagmus on the right side lasting for 20 s. Nystagmus and vertigo sensation were severer on the left side. In the audiogram she showed normal hearing function of the right ear and a pancochlear hearing loss of about 70 dB of the left ear. Caloric irrigation with 44°C water showed a malfunction of the left vestibular organ.

Although Epley's maneuver had been applied many times on both sides and the patient performed Brandt-Daroff exercises for weeks, vertigo and nystagmus have never completely ceased up to the present day, with more recurrences on the *unaffected right* side. In HR-3D-MR we distinguished a 3.6-mm defect in the left superior canal and a 1.7-mm defect in the left posterior canal, whereas on the right side we could not distinguish any morphological changes (fig. 3).

Patient 3

This 39-year-old patient had been suffering from severe attacks of vertigo when turning his head over the right shoulder since a skull trauma 4 years earlier. Cranial

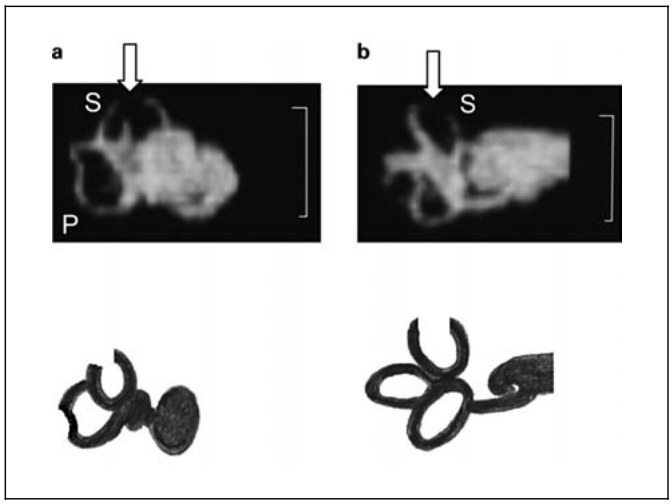

Fig. 4. Three-dimensional reconstructions of the inner ear of patient No. 3: 2-mm defect of the superior (S) semicircular canal (arrow), traumatic bending of the posterior (P) semicircular canal (arrow). **a** Right lateral view. **b** Right anterior view.

CT, MR and conventional X-ray photographs revealed no bone fractures or damage of brain structures; also cerebral angiography and clinical neurological investigation showed no abnormalities. Audiometry showed a symmetric slight high-frequency sensorineural hearing loss.

When turning the patient's head over the right shoulder in a sitting position, an intense counterclockwise rota-

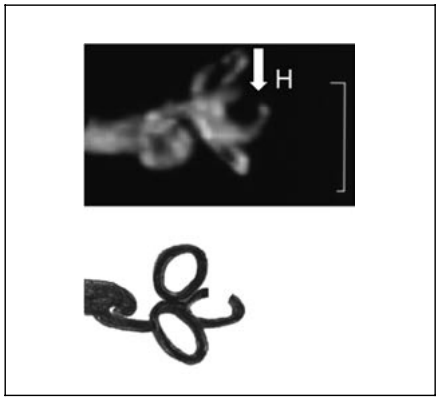

Fig. 5. Three-dimensional reconstruction of the inner ear of patient No. 4: left anterior view, 1.8-mm defect of the horizontal (H) semicircular canal (arrow).

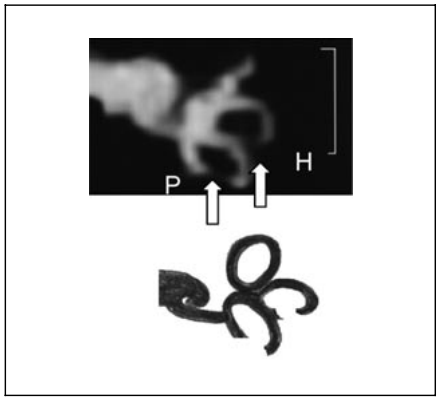

Fig. 6. Three-dimensional reconstruction of the inner ear of patient No. 5: left anterior view, 2-mm defect of the posterior (P) semicircular canal (arrow), 2.1-mm defect of the left horizontal (H) semicircular canal (arrow).

tory nystagmus with a latency between 5 and 10 s could be provoked on every occasion the patient had been investigated, lasting about 10–15 s and declining in response. The spinning sensation was so intense that no testing of fatigability could be performed. Epley maneuvers had been applied several times and the patient had also performed Brant-Daroff exercises for several weeks without any success.

In this patient HR-3D-MR showed an infraction of the right posterior semicircular canal and a filling defect of 2 mm on top of the right superior canal (fig. 4).

Patient 4

An 84-year-old female patient complained of positional vertigo attacks for more than 1 year when turning her head in 'the wrong direction'. The Dix-Hallpike maneuver provoked a horizontal, direction-changing geotropic nystagmus, which lasted about 30 s on the left side and about 10 s on the right side. Vertigo and nystagmus were much severer when the head was turned to the left side. The same pattern of nystagmus and vertigo appeared when the head was turned in the supine position.

Audiometry showed a pancochlear hearing loss on both sides; caloric irrigation could not be performed as the patient refused this investigation. Vertigo and nystagmus disappeared 3 days after performing Brand-Daroff exercises under medical supervision. HR-3D-MR showed a 1.8-mm defect in the left horizontal semicircular canal (fig. 5).

Patient 5

A 70-year-old woman presented with a history of positional vertigo for about 1 week when turning her head around in bed. The vertigo ceased when she brought her head back to the supine position and the attacks were described as severer on the right side. We found a high-frequency positional horizontal nystagmus with the fast phase beating away from the ground (ageotropic) on the left side, both when conducting the Dix-Hallpike maneuver and when turning the head with the patient lying in the supine position. The nystagmus persisted as long as the position was held (over 3 min) with the patient complaining of persistent vertigo. There was again an ageotropic horizontal nystagmus when turning the head to the right side, which was less severe but also persisted. The nystagmus showed no fatigability upon repeated head positioning to both sides and had no latency of onset. Caloric irrigation with 44°C water was symmetric; audiometry showed normal hearing.

We advised the patient to perform Brandt-Daroff exercises after performing HR-3D-MR. Under this therapy the patient was free of symptoms with no signs of nystagmus after 4 days. In HR-3D-MR we found a 2-mm defect in the left posterior canal and a 2.1-mm defect near the ampulla in the left lateral semicircular canal (fig. 6). Additionally, in a cross-sectional view through the horizontal ampulla, a minor filling defect of 1 mm in diameter could be distinguished (slice not depicted).

Discussion and Case Interpretation

Although BPPV is usually a self-limited disease with spontaneous recovery after weeks or months, or shows a high response to physical maneuvers, in some patients the vertigo persists [1]. Dornhoffer and Colvin [26] reported about 1% of patients with no improvement after repeated canalith repositioning maneuvers, which is in accordance with our own experience. Clinicians should be aware that incurable paroxysmal positional vertigo, although 'benign', can be a disabling and severely incapacitating disease, lasting for years or decades. As canalith reposition and liberatory maneuvers generally yield excellent results, our point was that in patients with prolonged atypical and incurable BPPV, there must be, as already postulated by Hall, a different underlying pathology [13]. Considering the modern theories of 'free-floating particles' and 'canalolithiasis', in our opinion these phenomena can only be explained by structural damage in the vestibular labyrinth which might be identified in HR-3D-MR.

So far this method has mainly been established for the assessment of neoplastic and infectious inner ear diseases and yields excellent results in assessing fibrotic or osseous obliteration in the intralabyrinthine fluid spaces [34, 36]. HR-3D-MR has also been applied in patients with vestibular neuritis and 'typical' BPPV with no proof of morphological changes except in posttraumatic cases [35]. Up to now there has been no publication dealing with HR-3D-MR investigations of patients with atypical BPPV.

In the 20 patients without vertigo who served as a control collective, we could not find any significant filling defects within the endolymphatic fluid spaces.

The 10 patients with 'typical' BPPV we included in this study responded excellently to physical treatment or recovered spontaneously. HR-3D-MR did not show any pathological configurations in the fluid spaces of the vestibular organ in these cases. We believe that these observations do not contradict the theory that 'typical' BPPV is caused by free-floating particles within the endolymph of one or more semicircular canals. Because of the difference in diameter between the ampulla and semicircular canal, displacements of the cupula can be effected by a small mass of loose otoconia ('leaky piston' [7]) which are beyond the resolution capacity of HR-3D-MR.

In our own patients examined, of about 350 persons with BPPV, we found only 2 who had been intractable up to the present date (patients 2 and 3) and 1 subject (patient 5) who presented with the symptoms of a heavy cupula without having undergone a canalith repositioning procedure before [37]. Concerning the 5 cases which clearly showed morphological alterations in HR-3D-MR investigations we try to perform an individual interpretation for each case.

Patient 1

This patient presented with symptoms of BPPV of the *right* side; a tympanoplasty type I had been performed 12 years prior on the *left* side. HR-3D-MR showed a 2.2-mm-long filling defect in the *left* superior semicircular canal (fig. 2a, b).

Parnes and McClure [14] were the first to notify that 'cupulolithiasis of the superior semicircular canal would result in exactly the same response as cupulolithiasis of the contralateral posterior semicircular canal'. Naturally this should also apply to canalolithiasis, as theoretically demonstrated by Honrubia et al. [38]. The symptoms occur when the affected ear is brought to the uppermost position which occurs during the performance of the Dix-Hallpike maneuver for the posterior semicircular canal of the contralateral side. In this position the clot begins to move and causes an ampullopetal deflection of the superior canal cupula with the nystagmus beating toward the unaffected ear [38].

BPPV of the superior semicircular canal is a rare occasion. Out of 292 patients with BPPV Honrubia et al. [38] suspected involvement of the superior canal in only 4 cases (without being able to offer direct proof). In sections, the superior semicircular canal and its cupula showed a lower incidence of basophilic deposits compared to both posterior and lateral canals and cupula [39, 40].

Due to the action of gravitational forces, loose granular sediment cannot collect or even conglomerate in the superior canal because it is the highest point of the vestibular labyrinth with the head in an erect position. These particles should leave the superior canal alone by getting from the supine position to the upright position [2]. For this reason we suspect that in cases with superior canal involvement, underlying pathological changes must be present, which should be recognizable in HR-3D-MR. In this patient this was a 'clot' with a diameter of 2.2 mm (fig. 2a, b) which was obviously inactivated by position changes alone, without any further treatment. A relation between preceding ear surgery and BPPV has frequently been described in the literature [7, 12, 41] and is explained by mechanical damage done to the macula of the utricle, leading to an increased release of otoconia into the endolymph.

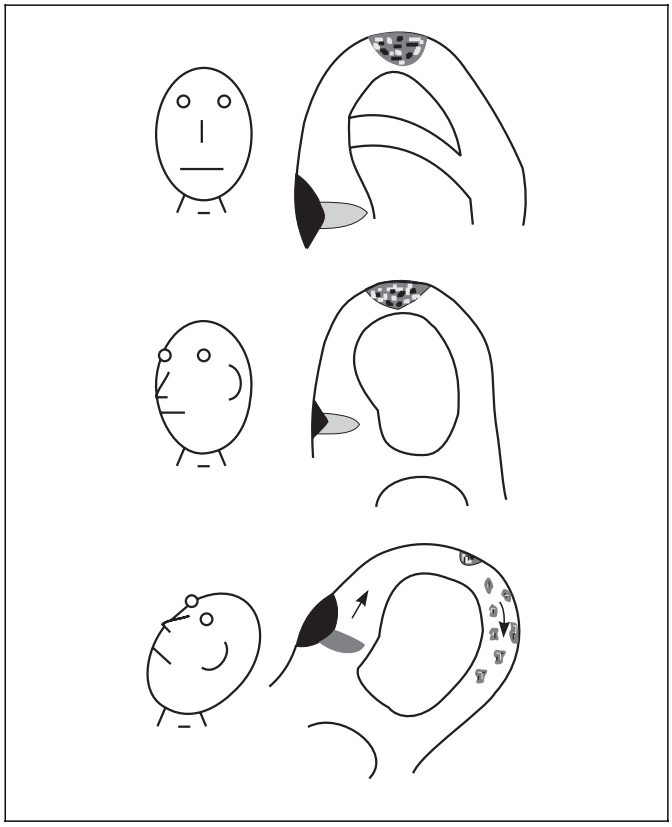

Fig. 7. Mechanism of cupular deflection in the right superior semicircular canal of patient No. 3.

Patient 2

Patient 2 presented with the symptoms of BPPV of both sides after a history of Ménière's disease and gentamycin therapy of the left ear.

The literature reports that in a high percentage of BPPV cases there are preceding concomitant ear diseases [7, 13, 18, 19, 42, 43]. Hughes and Proctor [44] found that Ménière's disease was the most frequent codiagnosis in BPPV. Gross et al. [27] held that Ménière's disease even predisposes to the intractable form of BPPV. The association with inner ear diseases is explained by hydropically induced damage to the maculae of the utricle and saccule of the membranous labyrinth. Permanent hydropic damage to the maculae may result in the release of permanently free-floating otoconia within the canals which collect in the posterior semicircular canal as the most dependent part of the labyrinth [44]. Hydropic damage to the semicircular canals may also result in adhesions, stenosis or occlusions by loss of the membranous labyrinth's resiliency [27, 45].

HR-3D-MR in this patient showed a 'clotting' of almost half of the lumen of the left superior semicircular canal (fig. 3a). This accounts for the recurrent nystagmus and vertigo when performing the Dix-Hallpike maneuver on the *right* side, because, as explained before, canalolithiasis of the superior canal of the *left* side elicits the same response as canalolithiasis of the *right* posterior canal [14]. Nevertheless, BPPV of the superior semicircular canal is considered very rare, because it is the highest point of the vestibular labyrinth. In this special case, we propose that both repeated hydropic distension *and* the following gentamycin therapy had done severe damage not only to the macula organ but also to the membranous labyrinth, thus creating a 'sticky' endothelium within the semicircular canals which causes adherence of the free-floating otoconia to the canal membrane and is responsible for the incurability of this case of canalolithiasis. Unfortunately the literature about the association of gentamycin therapy and consecutive BPPV is very rare. Vyslonzil [46] found significantly often deposits of otoconia in the posterior semicircular canal in sections of patients who had been treated with streptomycin (as gentamycin an aminoglycoside). The 'clot' we found in the left posterior semicircular canal accounts for the recurrent BPPV of the left side (fig. 3b).

Our findings are in accordance with Hughes and Proctor [44], who reported that in 41 patients with Ménière's disease 16 had BPPV in the same ear, but 18 had bilateral BPPV. We propose the hypothesis that at least in a part of these patients an endothelium damage of the superior canal of the Ménière-affected side might have been responsible for the phenomenon of a 'bilateral' positional vertigo also on the 'contralateral' side. Another reason may be that usually patients try to avoid provocative head movements and therefore aggravate the disease, with a possible development also on the contralateral side, as free-floating debris in the vestibular labyrinth seems to be, to a certain extent, a physiological process [39, 40].

Patient 3

This patient suffered from severe attacks of vertigo when turning his head over the right shoulder, accompanied by a high-frequency torsional counterclockwise nystagmus. The only possible mechanisms which can explain BPPV in that particular head position would be debris on top of the right superior canal, moving down the nonampullated arm and creating an ampullofugal cupula deflection (fig. 7). HR-3D-MR shows indeed an obstruction of the upper part of the right superior canal (fig. 4a). In the posterior canal we could determine an infraction, but no

complete obstruction (fig. 4b). Caloric irrigation showed a slightly decreased response on the *left* side without presence of head-shaking or vibrational nystagmus [47]. We therefore conclude that the morphological damage done to the right posterior canal had not been followed by a functional deficit.

BPPV occurring in the described position has hithero not been reported in the literature. As explanations we propose two mechanisms: (1) trauma-induced damage to the maculae of the saccule and utricle with increased release of otoconia; (2) the traumatized area of the superior canal may serve as a 'cristallization point' for the adhesion of free-floating endolymph particles, thus enabling them to collect at that particular location, as seen in HR-3D-MR (fig. 4a).

Otoliths adherent to the membranous labyrinth have been postulated as a possible mechanism of BPPV. Dissipation of the congealed mass within a semicircular canal by appropriate movements would result in an endolymph drag and fatigable nystagmus response [14].

This offers the only possible explanation for the underlying pathology in that case, because for gravitational reasons the top of the superior semicircular canal is considered to be the one location most unlikely to cause BPPV [2].

Patient 4

This patient showed a positional geotropic horizontal nystagmus on both sides with concomitant vertigo being severer on the left side when performing the Dix-Hallpike maneuver *and* when the head was turned in the supine position. We considered this to be a lateral canal variant of BPPV resulting from otolithic deposits in the left horizontal canal.

In 1985, Cipparrone et al. [48] first proposed the existence of a BPPV of the horizontal canal. In the same year, McClure [30] described the natural history and clinical symptoms of 7 patients suffering from that disorder. Pagnini et al. [49] presented 15 patients in 1989 and Vanucci et al. [50] 35 patients in 1997 confirming the hypothesis of McClure [30]. The horizontal canal variant of BPPV is characterized by a paroxysmal vertigo that is provoked when the patient lies in the supine position, turning his head to the right or left side. Its features are a geotropic paroxysmal direction-changing nystagmus on both sides, with nystagmus being more intense in one of the lateral positions, which is usually the affected side [37, 50]. HR-3D-MR accordingly shows a 1.8-mm filling defect in the left horizontal canal (fig. 5). When the patient turns his head to the affected side, the particles move to the ampullary side of the horizontal canal, causing an ampullopetal endolymph flow with a deflection of the cupula in the same direction and a nystagmus beating to the same ear (geotropic). When the patient turns his head to the other side, the particles move towards the nonampullary end of the horizontal canal producing an ampullofugal flow with a horizontal nystagmus beating to the nonaffected ear (also geotropic). Geotropic horizontal direction-changing paroxysmal positional nystagmus seems to be the most common type in horizontal canal vertigo. Nuti et al. [51] found it in 90%, Waespe [52] in 7 of 10 patients and Takegoshi et al. [53] in 8 of 11 patients. Fife [28] found 84% of patients had geotropic direction-changing nystagmus, compared to 16% with the ageotropic form.

Honrubia et al. [38] drew the same conclusions but distinguished between horizontal BPPV of the semicircular canal and horizontal BPPV of the cupula with otoliths being attached directly to the cupula. The latter case would be a sort of heavy cupula with the resulting nystagmus being ageotropic on both sides. As will be discussed below, such mechanisms may be possible, but according to our own experience we consider them to be very rare. Also Nuti et al. [29] describe the rare ageotropic variant of horizontal canal BPPV, with the starting position of otoconial debris being close to the ampulla, and the more common geotropic variant with the particles starting to get into motion farther away from the ampulla. In accordance with Pagnini et al. [49], we found the remission of vertigo in this patient to be more abrupt than in typical BPPV; after only 3 sessions of Brandt-Daroff exercises both nystagmus and vertigo disappeared completely.

Patient 5

This patient was diagnosed as suffering from atypical BPPV because she showed a *persistent* direction-changing ageotropic horizontal positioning nystagmus when performing the Dix-Hallpike maneuver and when turning her head in the supine position. In the literature there are only few reports about this rare form of BPPV [30, 37, 54] and it was the only such case we observed in our own patients. The only pathophysiological mechanism which can explain this phenomenon is the existence of a heavy cupula in one of the horizontal canals. Baloh et al. [37] observed 3 patients with the same phenomenon; 2 of them had a preceding history of typical posterior semicircular canal BPPV and developed bidirectional ageotropic nystagmus after a canalith repositioning maneuver, the third patient had no prior history of vertigo. Casani et al. [54] observed 9 patients with ageotropic horizontal bidirectional nystagmus provoked by lateral head movements

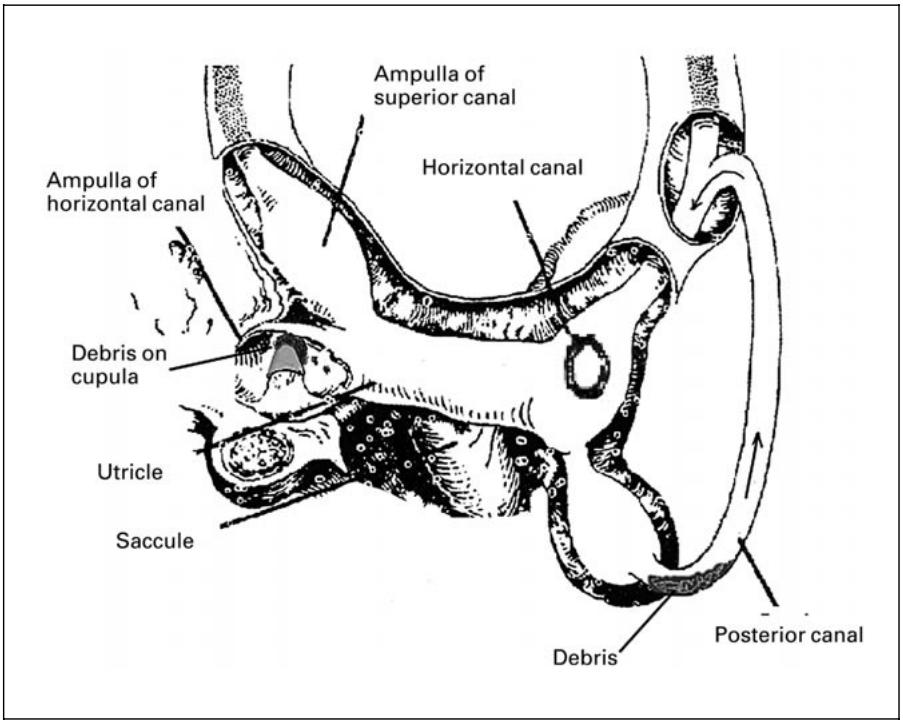

Fig. 8. Mechanism of the formation of a heavy cupula in the horizontal canal by debris originally collecting in the posterior canal and traversing through the common crus (adapted from Baloh et al. [37] with slight modifications).

while the head was in the supine position. In all cases, the nystagmus showed poor or no fatigability. They considered it as an atypical feature of horizontal canal BPPV, the most probable cause being a heavy cupula. Baloh et al. [37] concluded that this nystagmus resulted when the debris moved from the posterior semicircular canal into the utricle and became attached to the cupula of the horizontal canal (fig. 8). Buckingham [55] proposes a similar mechanism. Although this theory offers the best possible explanation, sections of temporal bones show that deposits on the cupula seem to be a physiological process without necessarily causing positional vertigo [39, 46, 53].

In our opinion it is a question of the mass of the debris being attached to the cupula. Because of the cross-sectional differential in diameter of approximately 1:5 between the ampulla and the semicircular canal, particles of equal weight gravitating through the canal would be 25 times more effective in overcoming cupular resistance than they would be if they adhered to the cupula ('leaky piston theory' [7]). So the quantity of the attached debris must be the critical factor which decides whether the cupula becomes sensitive to gravity or not. If the cupula of the horizontal canal is burdened with a critical mass (heavy cupula) this mass would cause an utriculofugal deviation resulting in nystagmus beating away from the undermost ear when the patient is turned to the abnormal side in the supine position. As the cause for the deflection of the cupula is not a hydrodynamic drag but a continuous gravitational force, the nystagmus should persist as long as the position is held, being a 'positional' rather than 'positioning' nystagmus. When the head is turned back to the vertical position, the attached mass no longer leads to a deviation of the cupula and the nystagmus ceases. The opposite effect occurs when the head is turned to the side of the normal ear, resulting in a direction-changing persisting nystagmus. In our patient we observed all these features described above. HR-3D-MR showed large filling defects in the left posterior and the left horizontal semicircular canal (fig. 6). We hold that this finding is in accordance with the theory of Baloh et al. [37]: A part of the 'clotted' material is supposed to have left the posterior semicircular canal via the common crus and to have impinged itself on the cupula of the horizontal canal (fig. 8). Still another part must have left the utricle through the nonampullated end of the horizontal canal, forming an additional clot in this canal. As it is generally accepted that the cupula 'seals' the ampulla and does not allow debris to pass through [28, 55], the adhering particles must have their origin in one of the semicircular canals, in this case most probably in the posterior canal (fig. 8).

During examination with positioning maneuvers, this patient showed no clinical signs of additional posterior canal affection, which we interpret as the dominance of the heavy cupula of the horizontal canal, subduing symptoms which might have been caused by the pathology we found in the posterior canal. The patient was symptom-free after 4 days of practicing Brandt-Daroff exercises. This confirms our hypothesis and supports the original conception of Semont et al. [20] and Brandt and Daroff [1], who developed their maneuvers to liberate the cupula from attached debris. The sudden movements from the sitting to the lateral position probably dislodged the debris from the cupula.

Although in the presented cases HR-3D-MR offers a new parameter for the diagnostic procedure for BPPV, one should be aware that clinical examination using positional maneuvers is still the key in diagnosing BPPV [28]. Direction and characteristic provocation of the nystagmus is still the most important feature in determining the involved canal. Duration, velocity, latency of onset of the nystagmus and severity of the concomitant vertigo are of no topodiagnostic value [28]. Neither are caloric irrigation or electronystagmography [56]. Paroxysmal vertigo and nystagmus can even be caused by an ear with absent caloric response, because the neurovascular supply of the posterior and superior semicircular canals is separated from the supply of the horizontal canal [7]. In our opinion, a patient who presents with the features of typical BPPV and responds favorably to bedside treatment does not need further specialized evaluation. If the symptoms and nystagmus completely resolve with or without treatment, it is highly probable that the sole cause has been canalolithiasis.

We recommend the use of HR-3D-MR in combination with cranial MR for the assessment or exclusion of concomitant neurological diseases, in patients who do not respond to physical treatment, in patients with recurrent atypical BPPV and in patients with posttraumatic BPPV. This method proves to be highly sensitive in detecting labyrinthine obstruction. Like Held et al. [36], who compared three-dimensional MR reconstructions of the inner ear in three groups of patients of different ages, we hold that filling defects in the membranous labyrinth depicted in HR-3D-MR are definitely pathological features and allow the clinician to draw diagnostic conclusions and to understand the failure of therapeutic measurements like canalith repositioning maneuvers and Brandt-Daroff exercises.

Conclusions

We believe that the unique findings in the patients presented in this article further substantiate the theory that 'typical' BPPV arises from free-floating particles within the endolymph of one or more semicircular canals. Considering our results, we come to the conclusion that there is no pathophysiological mechanism which would offer a better alternative explanation. The 10 patients with typical BPPV included in this study did not show any morphological changes in the vestibular organ. This does not contradict the canalolithiasis theory because, considering the difference in diameter between ampulla and semicircular canal, cupula displacement can be effected by a small mass of loose otoconia which are beyond the resolution capacity of HR-3D-MR and act as a leaky piston [7]. On the other hand, we could also demonstrate the existence of a 'clot' which occurs under certain circumstances in one of the canals and acts exactly as predicted by the canalolithiasis theory.

Our investigations confirm the theory of Hall et al. [13], who proposed that the permanent, therapy-resistant or atypical forms of BPPV may have a different pathophysiological mechanism than the self-limiting form of this disease. In each of our 5 cases with atypical BPPV we found evidence of structural changes of the vestibular labyrinth we did not find in cases with typical BPPV.

The canalolithiasis and cupulolithiasis theories do not contradict each other, but the latter is very rare indeed. In our own cases consisting of over 350 patients we found only 1 with a heavy cupula. Although the existence of cupular deposits seems to be a physiological process increasing with age [39, 40] without causing positional vertigo, these findings are no evidence against the existence of a heavy cupula causing positional vertigo. In our opinion it is the quantity of debris attached to the cupula which is the decisive factor for the cupula becoming a gravity-sensitive organ causing positional vertigo. HR-3D-MR of this patient supports the hypothesis of Baloh et al. [37] that the otoconial debris originally forms in the posterior semicircular canal and becomes attached to the horizontal cupula when traversing the common crus into the utricle. Cupulolithiasis causes a not fatigable *positional* rather than *positioning* nystagmus. Our patient (No. 5) could be cured after repeated performances of Brandt-Daroff exercises. Considering the HR-3D-MR findings, the conception of Semont and Brandt-Daroff maneuvers, originally aimed at freeing the cupula from attached debris, has been up to the point, although in most cases they prove also effective in the cure of typical BPPV by

dispersing conglomerations of debris in any canal. When performing cross-sections through the utricle and the horizontal canal ampulla we found a round filling defect of 1 mm in diameter in the left horizontal ampulla. In our opinion however, this finding offers no sufficient proof to be regarded as the radiological equivalent of a heavy cupula.

Affection of the superior horizontal canal is considered to be the rarest feature causing BPPV, because with the head in the upright position it is the highest location of the vestibular labyrinth, therefore it is impossible for loose otoconia to collect in it. For this reason there should always be a morphological anomaly which can be detected with HR-3D-MR when this canal is involved. Especially patients with 'typical' BPPV who do not respond to canalith repositioning maneuvers should be suspected of suffering from an affection of the superior semicircular canal of the contralateral side. Considering our investigations, involvement of the superior horizontal canal may be more frequent than is generally expected [38].

Examination using positional maneuvers remains the most important tool in diagnosing BPPV and determining the involved canal. Diagnostic problems arise when patients present with a concomitant ear disease or with symptoms or signs that suggest a more widespread balance disorder. Especially when dealing with multicanal disease and horizontal canal BPPV, the assumption that the side with the more intense nystagmus is the abnormal side may not always be correct. Many problems may arise when dealing with BPPV following head trauma and whiplash lesions, vestibular neuritis, Ménière's disease, stapes surgery and very old, immobile patients who cannot be examined properly. Here HR-3D-MR can be an important parameter to define the exact underlying pathology.

For the rare patients with severe, intractable symptoms that are unresponsive to bedside treatment maneuvers, surgery such as singular neurectomy and canal occlusion is an option [57, 58]. Especially in these patients, although being of crucial importance, it may be difficult to determine the exact side and canal, as a high percentage of patients suffering from incurable BPPV also show atypical nystagmus.

For the reasons mentioned above, we suggest the performance of HR-3D-MR of the inner ear under the following circumstances:

(1) in all patients suffering from the intractable form of positional vertigo as there is a high probability to find out the underlying pathophysiological mechanism;

(2) in cases of BPPV showing atypical nystagmus and prolonged symptoms to develop an effective therapy by possibly identifying the correct side and canal;

(3) after BPPV following head trauma, because radiological evidence of structural changes in the inner ear may give support to a medical expertise;

(4) in patients with positional vertigo suffering from multiple balance disorders or from a concomitant neurological disease because HR-3D-MR is usually performed in combination with MR of the brain and brainstem and thus a nonbenign variant can be excluded;

(5) in patients designated for operative procedures, because HR-3D-MR may be helpful to determine the pathological changes in the labyrinth.

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