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REVIEW

Surgical therapy in intractable benign paroxysmal positional vertigo

Marianne Leveque, MD, Marc Labrousse, MD, Laurent Seidermann, MD, André Chays, MD, Reims, France

OBJECTIVES: Benign paroxysmal positional vertigo (BPPV) is the most frequent vestibular disorder. Although it is easily cured with canal repositioning maneuvers for the majority of patients, it can be disabling in rare cases. For these patients, surgical solutions may be proposed. The aim of this article is to review the techniques used, the reported cases in the literature, and to discuss their indication in intractable BPPV.

STUDY DESIGN: Literature review.

MATERIALS AND METHODS: All the articles from 1972 to 2005 that discussed a specific surgical therapy in BPPV were reviewed. Many of them reported cases of operated patients and described original techniques. Some others are anatomic studies that discussed the two techniques used: singular neurectomy and posterior semicircular canal occlusion.

RESULTS: Singular neurectomy (posterior ampullary nerve transsection) and posterior semicircular canal occlusion are the 2 specific techniques used in intractable BPPV surgery. The numbers of operated cases are 342 and 97, respectively. These small numbers indicate that the procedures are difficult and risk compromising hearing and that a very small population of patients require surgical treatment of BPPV. The operated cases have been decreasing since the early 1990s because of improved management in BPPV. This article summarizes the techniques and their results and proposes a currently recommended practice of surgical therapy in BPPV as well as new insights into intractable BPPVs' physiopathology.

First described in 1921 by Barany,[1] benign paroxysmal positional vertigo (BPPV) is a very common vestibular disorder that affects nearly 40% of patients with complaints of vertigo. Although it has been regarded as a disease in the posterior semicircular canal since the histopathologic studies of Schuknecht and Ruby in 1969,[2] its physiopathology remains uncertain. Nevertheless, it is generally accepted that cupulolithiasis, canalithiasis, or both are related to the cause of BPPV. The symptoms of BPPV, precipitated by head movements, are brief (from 15 to 60 seconds) and the cause of typical forms is rarely serious, but the high intensity and frequency of attacks may be seriously disabling.

Physical therapy has been attempted since 1954.[3,4] Its aim was to make vestibular system adapted to stimulation produced by provocative positions. More recently, in the early 80s, canal repositioning maneuvers were developed that aimed to free lithiasis from the canal. Such maneuvers were described by Epley[5] and Semont et al.[6] These maneuvers are very effective, with 98% success after three maneuvers. Furthermore, spontaneous resolution is usual and takes from 6 to 8 weeks. Thus BPPV is generally considered a benign disease.

However, a small fraction of patients experience highly disturbing levels and frequency of BPPV attacks even after repeated canal repositioning maneuvers. Chronically disabling BPPV is defined as refractory vertigo persistent for 1 year with physical therapy. Some of those patients may be affected in their social, professional, and family activities and may be regarded as handicapped. Surgical solutions have been proposed for chronically disabled BPPV patients. These are based on ultraselective surgery that must be limited to the posterior canal system and are reserved for intractable patients with posterior BPPV after complete vestibular and imaging explorations.

Two types of interventions have been attempted for this indication to date: singular neurectomy (transsection of the

From the Department of ENT, Robert Debre Hospital, CHU Reims, France.

Reprint requests: Marianne Leveque, MD, 125 avenue du general Koenig, 51092 Reims cedex, France.

E-mail address: marilvq@aol.com.

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posterior ampullary nerve) and occlusion of the posterior semicircular canal. The goal of this article is to review each technique, its variants, treatment efficacy, and current trend in treatment plans.

SINGULAR NEURECTOMY

Richard R Gacek[7] proposed the first surgical solution in 1974. After several animal experimentations, he began to perform transsection of the posterior ampullary nerve (singular neurectomy); this nerve, also called singular nerve, innervates the posterior canal, whose dysfunction is responsible for the symptoms of BPPV. In the last 30 years, 342 singular neurectomies have been performed, 252 by Gacek himself.[8]

Anatomy of the Posterior Ampullary Nerve[9-13]

The posterior ampullary nerve runs from the posterior ampulla to the saccular nerve (forming the inferior vestibular nerve) through the singular canal of Morgagni in temporal bone. It is in the shape of an inverted "J" and is divided into three segments: canalicular (joining the internal auditory canal: straight line of the "J"), intermediate (curve of the "J"), and cribiform (entering the ampulla). The singular canal is approximately 4.5 mm long and 1 mm wide. In the intermediate segment, near the bottom of the "J" curve, the singular nerve lies very close to the round window membrane, which can be located 1 to 2 mm deep to its posteroinferior margin.

The embryologic genesis of the round window area explains the position of the singular canal.[12] The nerve grows in the middle of two ossification centers. Depending on the predominant ossification center, the level of the nerve in comparison with the round window may vary. Three types of positions are described. Type 1 is the lowest position: the nerve is located under the inferior part of the scala tympani. Type 3 is the highest position: the nerve is located higher than the superior part of the round window (this type goes straight from the internal auditory canal to the posterior ampulla). Type 2 is an intermediate form. Difficulty level in singular neurectomy depends on the course of the singular nerve. Type 1 is the easiest to operate on with the lowest risk, whereas type 2 and particularly type 3 are riskier in terms of damage to the round window membrane. The aim of the singular neurectomy is to expose and section the intermediate part of the singular nerve in the round window niche.

Technique

Gacek[7,8,14-19] described the technique with the patient under local anesthesia in order to control the patient's hearing and sensation of vertigo. A transcanal approach is made. The entire surface of the round window niche must be exposed, which sometimes implies drilling the posterior canal wall. Then, the bony overhang of the membrane is removed until the scala tympani is entirely visualized. Drilling is performed under the posterior margin of the membrane at a depth of 1 to 2 mm to locate the singular nerve. The singular nerve is transsectioned with a hook, which induces an attack of vertigo and a vertical downbeat nystagmus, and confirms the ablation of the semicircular canal input. In some cases, the singular canal may not be located. For those patients, Gacek recommends opening the posterior ampulla in order to section its neural ending. A slight flow of cerebrospinal fluid can occur when opening the singular canal. To prevent perilymph fistula, a piece of gelfoam should be placed on the drilling site at the end of the operation.

Variants

A few surgeons have attempted this intervention; five of them have published their results in literature and have proposed variants in the technique described by Gacek with relation to anesthesia and surgical approach: Silverstein and White,[20] Epley,[21] and Fernandes[22] preferred the use of general anesthesia; Meyerhoff[23] and Haüsler and Pampurik[24] operated under local anesthesia.

To facilitate exposure of the round window membrane, wide surgical approaches have been used. Silverstein and White performed a posterior incision that allowed them to posteriorly enlarge the external bony canal until the vertical portion of the facial nerve was identified. Epley used a hypotympanum approach by removing the inferior wall of the external bony canal.

RESULTS

The patient is tested 1 day after the operation with the Dix Hallpike maneuver. A successful treatment is indicated by the absence of vertigo and nystagmus while a provocative position is maintained. Complete relief was seen in 75% to 96% of cases according to the test performed by the surgeon. Partial relief occurred in 1.5% to 17% of patients, which is defined by reduction but not absence of vertigo and nystagmus triggered by a provocative maneuver.

When a report included more than 15 patients, there were always some patients whose symptoms did not improve after surgery. Causes were mostly incorrect diagnoses (horizontal and superior canal BPPV, bilateral BPPV), failure to locate the singular nerve, and incomplete transsection of the posterior ampullary nerve due to the presence of an accessory branch.[25] Patients experienced dizziness for a few days after the intervention; for that period, a vertical downbeat nystagmus can be seen that is known to be of cerebellar origin.[19]

Silverstein and White[20] are the only authors who explored the vestibular function on operated patients. They found that 41% of them had vestibular dysfunction with no clinical consequences whereas there were only 14% before operation. They concluded that there might be frequent

degenerative phenomena after such surgery (known as labyrinthitis) that may be responsible for that vestibular dysfunction and some loss of audition.

An important factor in the evaluation of the results is the loss of audition. Drilling very close to the round window membrane exposes the basal turn of the cochlea. As opening of the perilymphatic spaces seems to be a major cause of labyrinthitis, audition is at high risk. Sensorineural hearing loss depends on the surgeon's experience and represents from 3.7% of cases for Gacek[8] (252 neurectomies) to 41% of cases for Epley[21] (12 neurectomies). It ranges from a 30 dB hearing loss to total deafness. Most of those sensorineural hearing losses can be explained by the surgical act (injury to the scala tympani, to the perilymphatic space or, worse, to the endolymphatic space). Sensorineural hearing losses that remain unexplained may be due to secondary labyrinthitis. The risk increases when the singular canal is classified type 2 or 3, when there is otosclerosis, and in cases of reintervention. No hearing loss was observed in the 16 patients who underwent the posterior ampulla approach.

POSTERIOR SEMICIRCULAR CANAL OCCLUSION

Parnes and McClure[26] introduced posterior canal occlusion for BPPV in 1990. On the basis of the study by Money and Scott,[27] which proved that ablation of a semicircular canal could be performed while preserving function of other canals, they attempted their first posterior canal occlusion on guinea pigs. They found that the normal hearing can be retained unless the membranous labyrinth is damaged.

Obstruction of the semicircular canal lumen is thought to prevent endolymph flow and to fix the cupula in order to render it unresponsive to angular acceleration and gravitation. Parnes and McClure performed 42 cases of the total 97 posterior canal occlusions since 1990.[28,29]

Technique

Parnes and McClure[26] performed this operation with the patient under general anesthesia. The operation begins with a mastoidectomy that exposes the osseous posterior semicircular canal. Occlusion must be made at the farthest point from the ampulla and the vestibule to prevent damage. This target area is at or just inferior to the area bisected by the lateral semicircular canal, approximately 3 mm posterior to the facial nerve. A 3 to 4 mm segment is skeletonized 180 degrees around down to the endosteum with a diamond bur. If the endosteum is damaged and the perilymph leaks, drilling and suction must stop. Otherwise, the endosteal bone is removed with a hook, which exposes the membranous canal. A plug formed with the dry bone chips from the mastoidectomy and human fibrinogen glue is prepared and gently and firmly inserted to completely fill the canal, compressing the membranous labyrinth. Secondary fibrosis phenomenon will complete the occlusion. At the end of the procedure, a piece of fascia temporalis is placed over the canal to prevent perilymph fistula.

Variants

To minimize the risk of sensorineural hearing loss, some authors[30-33] proposed the use of lasers. When the canal is blue-lined, three to four bursts of argon or CO2 laser irradiation are made, before or after perforating endosteum with a pick. A plug is also used to occlude the canal. Those authors (Anthony,[30] Nomura et al,[31,32] Kartush and Sargent[33]) used laser irradiation to prevent tearing of the endolymphatic membrane when inserting the plug, but its advantage is not proven.

RESULTS

Eleven authors have reported 97 cases in the literature.[28-40] Only 3 patients among those 97 presented partial relief; vertigo symptoms recurred in 2 (3 months and several years later). In one patient, vertigo persisted when the head was moved rapidly. The 94 other patients were completely cured. All of those operated patients experienced post-operatively a period of dizziness and vertigo that lasted from 48 hours to a few weeks.

There were four cases of hearing losses reported. The most severe case was due to infectious labyrinthitis in a woman who had already undergone surgery twice for intractable BPPV (singular neurectomy).[29] She experienced an 85 dB sensorineural hearing loss with less than 8% speech discrimination. Parnes[29] reported another case of sensorineural hearing loss of 20 dB without affecting speech discrimination. The other two cases were conductive hearing losses; one of these was due to an inadvertent drilling on the incus.[34]

DISCUSSION

Current Practice for Singular Neurectomy

Many surgeons, mainly in the United States, have attempted transsection of the posterior ampullary nerve. However, only a few of them have published their results. Since 1993, only Gacek has continued to perform this procedure (252 neurectomies of the 342 reported), and only Gacek is reported to preserve hearing (97% of patients).

This operation seems to be largely superseded by posterior semicircular canal occlusion and the head positional maneuvers proposed by Epley[5] and Semont.[6] Haüsler and Pampuric[24] admit that cases of intractable BPPV in their unit have clearly decreased since they began to perform liberatory maneuvers.

Current Practice for Posterior Semicircular Occlusion

Despite excellent results with minimal risks for hearing, this operation does not seem to receive unanimous agreement.

Since 1990, only 97 instances of this procedure have been reported, mostly in Canada (however, there might be more unpublished cases). Eleven surgeons reported their experience in the literature; only four articles were published between 1999 and 2002.

Some authors attempted occlusion of other semicircular canals: there is 1 case of anterior semicircular occlusion[35] and another case of lateral semicircular occlusion.[32] At last, the technique of posterior canal occlusion has also been applied to the treatment of other inner ear pathologies.[28] For example, anterior canal dehiscence can be treated by anterior canal plugging and difficult-to-access skull base lesion can benefit from partial labyrinthectomy by plugging some of the canals to preserve hearing.

Comparison Between the Two Procedures

Thirty years after the first case of surgical treatment of intractable BPPV, those two procedures are still debated. The comparison is not straightforward because the number of operated cases and the follow-up time are quite different: 342 singular neurectomies versus 97 posterior semicircular occlusion and 14 to 30 years of follow-up.

They are both difficult procedures but it seems that posterior canal occlusion is the preferred route when intractable BPPV is surgically treated. This is because hearing is at high risk in singular neurectomy, except for cases by Gacek, where hearing loss is rare in posterior semicircular occlusion. This is explained by the various locations of the singular nerve, which is more difficult to localize than the posterior canal. The singular nerve is difficult to identify when drilling and the access to the round window niche area is complicated because it lies under the level of the inferior wall of the external bony canal. Many authors emphasized the great difficulty of singular neurectomy by analyzing the anatomy of the singular nerve. Ohmichi et al,[10] Mills et al,[9] and Leuwer and Westhofen[13] called into question the excellent results obtained by Gacek and concluded that in 14% to more than 30% of cases (corresponding to all type 3 nerve and some type 2) singular nerve cannot be found without opening the basal turn of cochlea, making Gacek's good results very difficult to explain (Gacek performs ampullary nerve transsection without identifying the nerve's type preoperatively, and therefore he cannot exclude type 2 and 3 nerve). Leuwer and Westhofen[13] hypothesized that its good results might depend on other mechanisms, maybe vibrations due to drilling.

Current View on Surgical Management for Intractable BPPV

Two observations can be made with respect to the practice for intractable BPPV. First, this kind of surgery does not seem to be popular outside North America: 413 of the 439 operations were performed in Canada and the United States. Singular neurectomy was mainly performed in the United States, and posterior semicircular canal occlusion in Canada. These techniques seem to be confined to where they were first developed. This is likely because of the difficulty of the procedure as well as the training necessary for new practitioners.

Second, the number of cases of surgical therapy has decreased since the early 1990s. Today, surgical treatment of BPPV appears to be limited to exceptional cases. This is likely because of improved management of patients with BPPV in the following areas: lateral, anterior, and conjugated canals' BPPV symptoms were first diagnosed at the end of the 1980s. Before that time, many intractable BPPV were misdiagnoses of posterior canal BPPV. The use of videonystagmography, which is much more precise than electronystagmography, improved the accuracy of diagnoses of difficult cases.

Differential diagnosis of atypical and intractable BPPV can easily be made by cerebellar fossa imaging (MRI). Physical management for BPPV has also advanced, and liberatory maneuvers can now cure most posterior BPPV, including repetitive ones. Furthermore, other liberatory maneuvers and re-education techniques have been elaborated to help patients in which the lateral, anterior, or associated canal is involved.

Is Intractable BPPV Still BPPV?

Despite our knowledge on BPPV, the question remains: why are some people still suffering from intractable BPPV? It is obvious that in the 1970s and the 1980s many intractable BPPVs were a misdiagnosis of lateral, anterior, bilateral BPPV, or posterior fossa tumors. Nowadays, well-established physiopathologic theories of cupulolithiasis and canalithiasis allow clinicians to make very precise diagnoses of the different forms of BPPV and then to give their patients the best available treatment.

As good examination and appropriate treatment fail to cure patients with intractable BPPV, pathophysiological models of cupulolithiasis and canalithiasis may be called into question in those particular patients. Many similarities have been reported between the symptoms of BPPV and some neurologic symptoms such as migraine or posterior fossa tumors, and it is clearly established that neurologic troubles can mimic BPPV. Furthermore, there is a strong association between neuronitis and secondary BPPV, which are more difficult to treat. We can hypothesize that intractable BPPV might represent a neurologic vestibular disorder rather than the mechanical disease responsible for the common BPPV. In the 1970s, Fluur and Siegborn[41-43] described interactions between otolith and semicircular canal inputs in oculomotor muscles control, especially interaction of the saccula with the posterior canal. In a recent study, Gacek[44] considered that saccular nerve dysfunction might be able to cause BPPV, as he observed virally induced saccular nerve degeneration in five temporal bones with BPPV but without canalithiasis. He also reported MRI examination during BPPV attacks show high signal intensity localized in the vestibular nerve, and he concluded that BPPV may be a particular form of neuronitis.

We believe that such a theory could apply to intractable BPPV. That could explain that this particular form does not behave as a canalithiasis or a cupulolithiasis. To test this hypothesis, further vestibular examinations must be made in those patients, including otoliths' function testing such as subjective, visual, vertical, and vestibular evoked myogenic potentials.

CONCLUSION: INDICATIONS OF BPPV'S SURGERY TODAY

Two surgical techniques discussed in this article are exceptional treatment, recently practiced with decreasing frequency. Intractable BPPV is, in fact, not benign, and the symptoms, although similar to BPPV, might involve another physiopathology probably more neurologic. Therefore, intractable BPPV deserves a more accurate name. Patients with this disease should be tested with precise vestibular examination and posterior fossa imaging to confirm the diagnosis of "posterior canal system disease," but also to elucidate its physiopathology. Until the physiopathology of the intractable BPPV is adequately identified, our best treatment option for severely affected patients is a surgical therapy, preferably posterior semicircular canal occlusion. In our experience of dissecting several temporal bones with the two techniques, we found that posterior canal occlusion presents far fewer technical issues and far lower risk of hearing loss, compared with transsection of the posterior ampullary nerve. However, intractable BPPV is a very rare disease and its surgical therapy should be applied to exceptional cases only, necessitating only a few surgeons in the world.

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