Effectiveness of canal occlusion for intractable posterior canal benign paroxysmal positional vertigo: a systematic review

Maas, B.D.P.J.; Zaag-Loonen, H.J. van der; Benthem, P.P.G. van; Bruintjes, T.D.

AMERICAN ACADEMY OF OTOLARYNGOLOGY–HEAD AND NECK SURGERY FOUNDATION

Otolaryngology–Head and Neck Surgery
2020, Vol. 162(1) 40–49

DOI: 10.1177/0194599819881437
http://otojournal.org
SAGE

Effectiveness of Canal Occlusion for Intractable Posterior Canal Benign Paroxysmal Positional Vertigo: A Systematic Review

Britta D. P. J. Maas, MD[1], Hester J. van der Zaag-Loonen, MD, PhD[1], Peter Paul G. van Benthem, MD, PhD[2], and Tjasse D. Bruintjes, MD, PhD[1]

No sponsorships or competing interests have been disclosed for this article.

Abstract

Objectives. A last resort for therapy for intractable benign paroxysmal positional vertigo (BPPV) is mechanical occlusion of the posterior semicircular canal. The aim of this review was to assess the effect of posterior canal occlusion for intractable posterior canal BPPV on vertigo and to determine the risk of loss of auditory or vestibular function.

Data Sources. A systematic literature search according to the PRISMA statement was performed on PubMed, the Cochrane Library, Embase, Web of Science, and CINAHL. The last search was conducted in June 2018.

Review Methods. Cohort studies with original data and case reports describing >5 cases were included if they analyzed the effect of posterior semicircular canal obliteration in adults with intractable posterior BPPV on vertigo. Two authors screened titles and abstracts for eligibility. The first author screened full texts and analyzed the data.

Results. Eight retrospective studies met the eligibility criteria. The quality of all individual studies was rated fair. Canal occlusion was performed on 196 patients. All studies reported complete resolution of BPPV in all patients (100%). Among postoperatively tested patients, total loss of auditory function and vestibular function was reported in 2 of 190 (1%) and 9 of 68 (13%), respectively.

Conclusion. Posterior semicircular canal plugging resulted in 100% resolution of BPPV in patients with intractable BPPV in all studies. However, the strength of evidence was weak. Potential serious complications, such as deafness and loss of vestibular function, should be taken into account.

Keywords

benign paroxysmal positional vertigo, BPPV, posterior semicircular canal, canal occlusion

Received May 9, 2019; accepted September 19, 2019.

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular type of vertigo, with an estimated incidence of 64 cases per 100,000 population per year and a lifetime prevalence of 2.4%.[1,2] It is characterized by sudden and brief spinning sensations initiated by a change of head position.[3,4] A widely accepted hypothesis of the cause of BPPV is the detachment of degenerated otoconia from the otolithic membrane in the utricle, dislocating into the semicircular canals. Because of clotting of the otolithic fragments, the cupula in the semicircular canal becomes sensitive for gravity, and this leads to positional vertigo.[5] It can occur in the anterior, horizontal, and posterior semicircular canal.

BPPV may recover spontaneously in approximately 25% of patients after 1 month and up to 50% after 6 months of follow-up.[6-8] However, patients without spontaneous recovery are at increased risk for fall incidents and impairment for daily activities.[9,10]

In the case of posterior canal BPPV—the most common type of BPPV—patients can be treated successfully with a single canalith-repositioning maneuver (CRM) in 85% of the cases.[11] Before the introduction of a CRM for posterior canal BPPV in 1992, various surgical treatments were performed for intractable cases.[12-15] These included destruction of the labyrinth, nerve VIII transection, transection of the posterior ampullary nerve, and surgical occlusion of the posterior canal. Although not often performed, the last was accepted as an appropriate surgical option for patients with intractable posterior canal BPPV, since this technique was the least destructive and it was more easy for surgeons to perform than nerve transection. The first surgical occlusion of a posterior semicircular canal was described by Parnes

[1]Apeldoorn Dizziness Centre, Gelre Hospital, Apeldoorn, the Netherlands
[2]Department of Otorhinolaryngology and Head and Neck Surgery, Leiden University Medical Centre, Leiden, the Netherlands

Corresponding Author:
Britta D. P. J. Maas, MD, Apeldoorn Dizziness Centre, Gelre Hospital, Albert Schweitzerlaan 31, Apeldoorn, 7334 DZ, the Netherlands.
Email: b.maas@gelre.nl

and McClure in 1990.[16] They performed surgery on 2 patients with a history of episodic positional vertigo and severe ipsilateral hearing loss. The posterior semicircular canal was identified, blue lined, opened, and plugged with bone chips or bone paste. Postoperatively, both patients reported complete resolution of the positional vertigo, and no nystagmus and vertigo could be provoked by performing the Dix-Hallpike maneuver. However, caloric responses diminished, and because of the preoperative severe hearing loss, the effect of surgery on the auditory function was unclear. Nowadays, the CRM is the gold standard for treatment for BPPV. Surgical therapy, such as canal occlusion, is considered a last resort for patients who do not respond to repeated CRMs.[17]

Objectives

The aim of this review was to assess the effect of canal occlusion for intractable posterior canal BPPV on vertigo and to determine the risk of loss of the auditory or vestibular function by performing this surgery.

Methods

Protocol

We searched the literature using the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-analyses).[18]

Eligibility Criteria

Although we searched for retrospective and prospective studies, we included only retrospective cohort studies with original data as well as case reports describing >5 cases if they analyzed the effect of obliteration of the posterior semicircular canal in adults with intractable posterior BPPV on vertigo. Secondary outcome measures, such as auditory function, vestibular function, and complications, were recorded if presented in the data. No restrictions on language or publication year were applied.

Information Sources

We performed a systematic literature search to investigate the effectiveness of canal occlusion in intractable posterior canal BPPV on outcomes on vertigo, auditory function, and vestibular function. We searched the following electronic medical databases: the Cochrane Library, MEDLINE (PubMed), Embase, Web of Science, and CINAHL. The last search was performed on July 5, 2018.

Search

We searched the databases for abstracts, titles, and keywords containing suitable search terms from inception to June 2018. A detailed description of the search in each database is presented in Appendix 1 (available in the online version of the article).

Process of Study Selection and Data Collection

One reviewer excluded duplicates (B.D.P.J.M.). Two independent reviewers (B.D.P.J.M. and H.J.v.d.Z.-L.) screened titles and abstracts for eligibility for inclusion. Discrepancies were discussed. One reviewer (B.D.P.J.M.) screened full texts of the eligible articles and analyzed the data.

Data Items

From each full-text article, we extracted the following data items: study design, sample size, sex, age, indication for surgery, surgical technique, follow-up, lost to follow-up, and complications, as well as outcomes on vertigo, auditory function, and vestibular function.

Quality Assessment of Individual Studies

To assess the quality for each study, we used the quality assessment tool for case series studies developed by the National Heart, Lung, and Blood Institute of the US National Institutes of Health. This tool evaluates a total of 9 elements for each study. A polar (yes/no) scale was used to estimate the risk of bias for each element. In case one or more components could not be determined, were not reported, or were not applicable, this was noted. The evaluation was executed by 2 independent reviewers (B.D.P.J.M. and T.D.B.). If ratings differed, the reviewers discussed them to reach consensus.

We considered a research question or objective clearly stated if the authors defined the focus of their study and if they identified primary or secondary outcome variables. The study population was regarded as clearly and fully described if the authors presented a short explanation of patient characteristics, such as sex, mean age, and mean duration of symptoms. Comparability was based on the primary outcome measure, and subjects were considered comparable if they all had an equal indication for surgery, the same treatment prior to surgery, and identical outcomes on diagnostic tests. The outcome measure was considered clearly defined, valid, reliable and consistent if the authors clearly described that the primary outcome was objectively measured with the Dix-Hallpike maneuver and if this was implemented across all study patients. We defined an adequate length of follow-up as a period of at least 6 months after surgery.

Summary of Outcome Measures

We considered the outcome of BPPV the primary outcome measure. This outcome was subdivided into 2 outcome measures: an objective outcome and a subjective outcome. The Dix-Hallpike maneuver was considered an objective primary outcome measure. If the resolution of BPPV was assessed otherwise or if the method of measuring was not explained, we considered this to be a subjective primary outcome measure. The effects on the auditory function and vestibular function were considered secondary outcome measures. The following secondary outcome measures were considered valid: pure tone audiogram and caloric response.

Results

Study Selection

The search resulted in a total of 154 articles (**Figure 1**). After screening on title, abstract, and full text, we screened

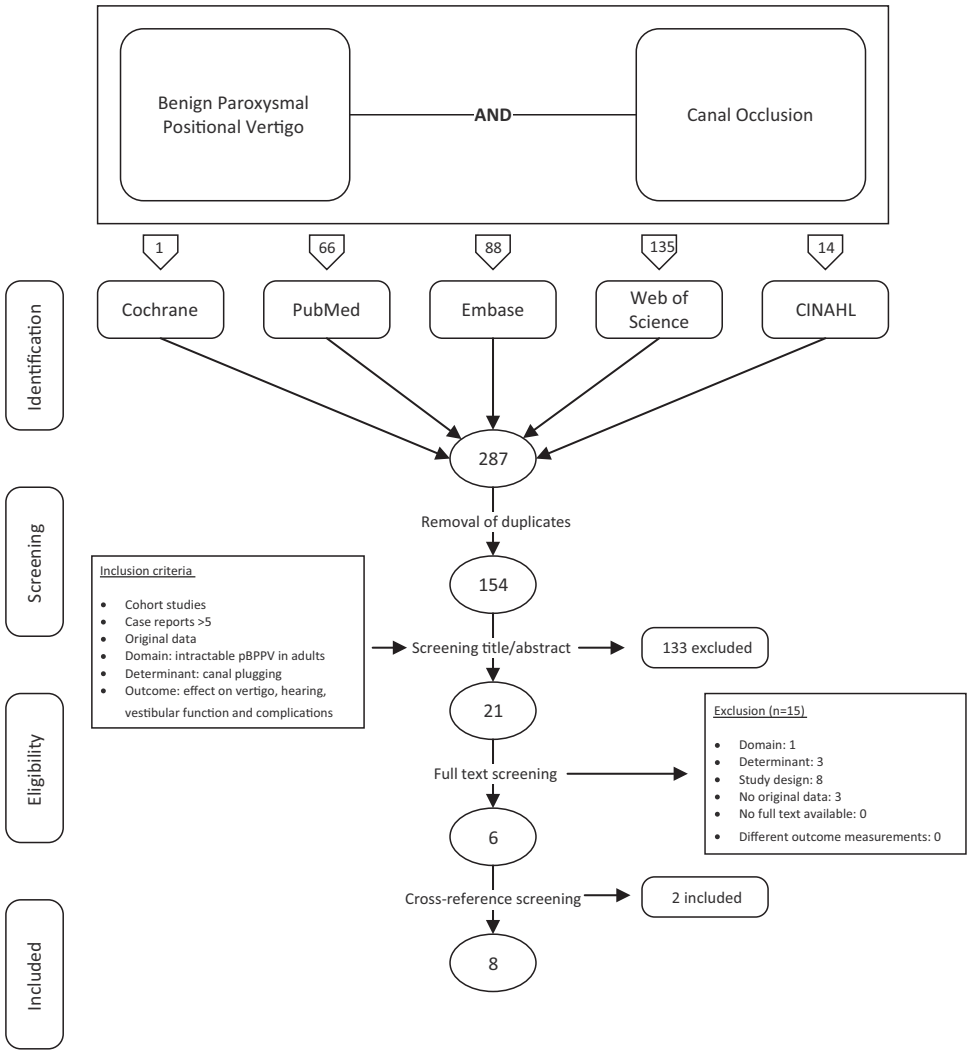

Figure 1. Flow diagram for study selection according to the PRISMA statement.

the reference lists of the remaining 6 articles. Eventually, we included a total of 8 retrospective studies in this review. Although we searched for retrospective and prospective studies, no eligible prospective cohort studies were retrieved from the literature.

Study Characteristics

Study Design, Sample Size, and Inclusion of Patients. All studies had a retrospective design (**Table 1**). Sample sizes were expressed in numbers of patients or procedures. The number of patients varied between 6 and 61. One study presented 12 procedures in 6 patients with bilateral disease.[19] All studies except for 1 reported the time span of inclusion of patients. The longest inclusion period was found in Beyea et al,[20] which lasted 22 years (1988-2010). Two studies included patients without a repositioning maneuver prior to surgery, as this was not the standard treatment at the time of these studies.[19,20]

Indication for Surgery and Surgical Technique. Before proceeding to canal occlusion, 4 studies clearly described a certain required duration of symptoms, and 7 studies considered failed conservative attempts, such as the Epley maneuver or Semont maneuver, as a criterion for plugging the posterior semicircular canal.

In all studies, a cortical mastoidectomy via a postauricular transmastoid approach was performed. If the posterior semicircular canal was identified, the canal was skeletonized, and the lumen of the canal was fenestrated. The membranous labyrinth was then mechanically compressed. In most studies, a mix of harvested bone tissue and fibrinogen sealant was used for plugging. Subsequently, the defect was covered up with fascia of the temporalis muscle.

Follow-up and Lost to Follow-up. Follow-up time was reported in 5 of 8 studies, and this time varied from 2 months to 17 years.

None of the studies reported loss of patients in follow-up. Three studies reported lost results for audiograms or caloric tests in follow-up.[21-23]

Quality Assessment of Individual Studies. The results of the quality assessment for each study are presented in **Table 2**. We rated the quality of all included studies as fair. According

Table 1. Study Characteristics.

First Author	Patients, n	Procedures, n	Sex, F:M	Age, y, Mean (Range)	Design	Time Span of Patient Inclusion	Indication for Surgery	Follow-up	Lost to Follow-up	Complications
Parnes (1991)[25]	6	6	5:1	66 (55-73)	Case series	Unknown	"BPPV for more than 1 year and with symptoms severe enough to significantly affect the patient's occupation or lifestyle"	11-21 mo	None	1 otitis media with effusion, 1 perilymph fistula
Hawthorne (1994)[22]	15	15	10:5	49.5 (31-64)	Retrospective cohort study	1990-1994	Intractable BPPV: "patients should have tried the Semont manoeuvre"	14-40 mo	Caloric results in 5 patients	Dislocation of the incus, causing 30-dB conductive hearing loss
Zappia (1996)[41]	8	8	7:1	32-48	Case series	1993-1995	"Failed attempts at conservative treatment"	2-27 mo	None	No serious complications
Pulec (1997)[42]	17	17	9:8	43 (18-72)	Retrospective cohort study	1991-1995	BPPV not responding to an Epley maneuver and "vestibular-suppressing medication"[a]	Unknown	Unknown	No serious complications
Kisilevsky (2009)[23]	30	32	24:8 procedures	46 (15-68)	Retrospective cohort study	1988-2006	Intractable BPPV: at least 12-mo duration, no response to physical therapy (Brandt-Daroff and Semont)	Mean, 63 mo; range, 2-205 mo	Caloric results in 9 patients	No serious complications
Ahmed (2012)[21]	53	55	35:18	59 (31-88)	Retrospective cohort study	1991-2011	Persistent or (monthly) relapsing, distressing or disabling, unilateral pBPPV; poor or no response to CRMs performed over a 6-mo period; hearing in the unaffected ear equal to or better than that in the affected ear	Mean, 4.3 y; range, 0.5-13 y	Audiograms in 6 patients, caloric results in 13 patients	1 wound infection, 1 perilymph fistula
Beyea (2012)[20]	61	65	Unknown	59.8	Retrospective cohort study	1988-2010	Intractable symptoms of BPPV; at least for 1 y; unresponsive to repeated CRM (43 of 65)[b]	Unknown	Unknown	No serious complications
Ramakrishna (2012)[19]	6	12	5:1	50-72	Case series	1989-2007	Bilateral intractable pBPPV; not responding to a single CRM; refractory to medical treatments[c]	Unknown	Unknown	No serious complications

Abbreviations: BPPV, benign paroxysmal positional vertigo; CRM, canalith-repositioning maneuver; F, female; M, male; pBPPV, posterior benign paroxysmal positional vertigo.
[a]"The Hallpike maneuver was performed by the author to document *horizontal-rotatory* nystagmus characteristic of BPPV of . . . the posterior semicircular canal."
[b]Between 1988 and 1991, the authors were not familiar with a repositioning maneuver; hence, in 12 patients, no repositioning maneuver prior to surgery was performed.
[c]One patient without a repositioning maneuver prior to surgery, as this was not standard treatment yet.

Table 2. Quality Assessment for Individual Case Series Studies.[a]

First Author	1	2	3	4	5	6	7	8	9
Parnes (1991)[25]	No	Yes	CD	Yes	Yes	No	Yes	NA	Yes
Hawthorne (1994)[22]	No	Yes	CD	No	Yes	Yes	Yes	NA	Yes
Zappia (1996)[41]	No	Yes	CD	No	Yes	Yes	No	NA	Yes
Pulec (1997)[42]	No	Yes	CD	No	Yes	No	NR	NA	Yes
Kisilevsky (2009)[23]	No	Yes	CD	No	Yes	Yes	No	NA	Yes
Ahmed (2012)[21]	Yes	Yes	CD	Yes	Yes	No	Yes	NA	Yes
Beyea (2012)[20]	Yes	Yes	CD	No	Yes	No	NR	NA	Yes
Ramakrishna (2012)[19]	No	Yes	CD	No	Yes	Yes	NR	NA	Yes

Abbreviations: CD, could not determine; NA, not applicable; NR, not reported.
[a](1) Was the study question or objective clearly stated? (2) Was the study population clearly and fully described? (3) Were the cases consecutive? (4) Were the subjects comparable? (5) Was the intervention clearly described? (6) Were the outcome measures clearly defined, valid, reliable, and implemented consistently across all study participants? (7) Was the length of follow-up adequate? (8) Were the statistical methods well described? (9) Were the results well described?

Table 3. Results of Individual Studies for Vertigo: Primary Outcome Measure.[a]

			Dix-Hallpike			
First Author	Patients, n	Procedures, n	Preoperative	Postoperative	Postoperative Point of Time	Measuring
Parnes (1991)[25]	6	6	Unknown[b]	Unknown	—	Subjective
Hawthorne (1994)[22]	15	15	Yes (15 of 15)[c]	Yes (15 of 15)	Unknown	Objective
Zappia (1996)[41]	8	8	Yes (8 of 8)[d]	Yes (8 of 8)	Unknown	Objective
Pulec (1997)[42]	17	17	Yes (17 of 17)	Unknown (17 of 17)[e]	Unknown	Subjective
Kisilevsky (2009)[23]	30	32	Yes (32 of 32)[f]	Yes (32 of 32)	"Immediate"	Objective
Ahmed (2012)[21]	53	55	Unknown[g]	Unknown	—	Subjective
Beyea (2012)[20]	61	65	Yes (65 of 65)[h]	Unknown	—	Subjective
Ramakrishna (2012)[19]	6	12	Unknown	Yes (6 of 6)	Unknown	Subjective

[a]Each study reported 100% resolution.
[b]One patient with preoperative ipsilateral Ménière's disease.
[c]Three patients with atypical nystagmus (no latency and/or no adaptation) and 1 without nystagmus on the preoperative Dix-Hallpike maneuver.
[d]One patient with a subjective response (sensation of vertigo without nystagmus) on the preoperative Dix-Hallpike.
[e]Postoperatively described as "the provocative position"; unclear whether this is a Dix-Hallpike maneuver.
[f]One patient tested negative on preoperative Dix-Hallpike maneuver; 2 patients with preoperative Ménière's disease.
[g]Seven patients with unilateral ear disease: 2 ipsilateral Ménière's, 1 previous vestibular neuronitis, 2 mastoid surgery, 1 previous herpes zoster, 1 vestibular schwannoma.
[h]Prior failed singular neurectomy in 2 patients.

to Shekelle et al, we graded the level of evidence as 4 and the level of recommendation as D.[24]

Results of Individual Studies per Outcome Measure

Primary Outcome: Vertigo. All studies discussed the effect of plugging on vertigo, and regardless of the method of measuring, all studies reported complete resolution of positional vertigo in all patients (100%; **Table 3**). Three of 8 studies objectively measured the effect of canal plugging on BPPV by performing the Dix-Hallpike maneuver pre- and postoperatively.

Secondary Outcomes

Auditory Function. For the evaluation of the auditory function, all studies stated to have performed audiometric tests before (n = 196) and after (n = 190) surgery (**Table 4**). Two patients (1%) of 1 study had a Barany deaf ear (>100-dB hearing loss).[21] Four studies reported a total of 15 patients (8%) with permanent sensorineural hearing loss due to surgery. Nine patients (5%) experienced persistent mild conductive hearing loss, and in 1 of them, this was caused by a dislocation of the incus.

Vestibular Function. Total loss of vestibular function was observed in 9 of 68 postoperatively tested patients (13%). Three of these patients had a preoperative reduction of their vestibular function on the side opposite to that of the operation.

A reduced caloric response was observed in 14 patients (21%) who underwent postoperative caloric testing. Not all patients who received preoperative caloric tests were subjected to postoperative testing. This was due to several reasons, as described in **Table 5**.

Table 4. Results of Individual Studies for Auditory Function: Secondary Outcome Measure.

First Author	Patients, n	Procedures, n	Audiogram			Hearing Outcome		
			Preoperative	Postoperative	First Postoperative Point of Time	Temporary Loss	Permanent Loss	Deafness
Parnes (1991)[25]	6	6	Yes (6 of 6)[a]	Yes (6 of 6)	Unknown	Transient mild to moderate mixed hearing loss (30-50 dB) in 3 of 5 patients with normal preoperative hearing	—	—
Hawthorne (1994)[22]	15	15	Yes (15 of 15)[b]	Yes (15 of 15)	6 wk	8 of 15 mild high-frequency SNHL (8-48dB)	1 of 15 with mean conductive hearing loss of 30 dB[c]	—
Zappia (1996)[41]	8	8	Yes (8 of 8)[d]	Yes (8 of 8)	3-6 wk	All patients with temporary hearing loss	None	None
Pulec (1997)[42]	17	17	Yes (17 of 17)	Yes (17 of 17)	Unknown	"All had conductive hearing loss for three to four weeks until there was resolution of the hemotympanum."	None	None
Kisilevsky (2009)[23]	30	32	Yes (32 of 32)[e]	Yes (32 of 32)	Unknown	"All patients had a temporary conductive hearing loss postoperatively."	2 of 32 with mild persistent SNHL at 8 kHz (15 of 25 dB); 8 of 32 with mild persistent conductive hearing loss	—
Ahmed (2012)[21]	53	55	Yes (55 of 55)[f]	Yes (49 of 55)	1 wk	15 of 49 audiograms with temporary SNHL >20 dB	9 of 49 patients with permanent change in SNHL >20 dB at low frequencies or >25 dB at high frequencies or both	2 of 9 with Barany deaf ear[g]
Beyea (2012)[20]	61	65	Yes (65 of 65)[h]	Yes (65 of 65)	Unknown	"Almost all patients with normal preoperative hearing have an initial transient postoperative hearing loss."	3 of 61 with delayed permanent SNHL; 1 severe loss at 3 mo postoperatively; 2 minor high-frequency loss at 1 y postoperatively	—
Ramakrishna (2012)[19]	6	12	Yes (6 of 6)	Yes (6 of 6)	Unknown	"All procedures were followed by transient postoperative mild to moderate hearing losses in the ear operated on."	1 of 6 with persistent very mild bilateral high-frequency SNHL	—

Abbreviation: SNHL, sensorineural hearing loss.

[a]Preoperatively 1 of 6 patients with profound SNHL due to Ménière's disease.

[b]Five patients with preoperative SNHL ranging from 30 dB to a total SNHL caused by central perforation, presbyacusis, postmastoidectomy, idiopathic origin, and genetic etiology.

[c]Due to a dislocation of the incus.

[d]Preoperatively 1 of 8 with mild SNHL.

[e]Preoperatively 8 of 32 with mild-moderate SNHL and 2 of 32 with severe-profound SNHL.

[f]Five patients with preoperative >20-dB hearing loss. Audiometric tests were conducted in only 24 procedures with an abnormal first postoperative audiogram (>20-dB SNHL)

[g]One patient with a change in hearing levels of >70 dB due to surgery; the other patient with a preoperative threshold of ±80 dB.

[h]Preoperatively 4 of 65 audiograms with profound SNHL.

Table 5. Results of Individual Studies for Vestibular Function: Secondary Outcome Measure.

			Calorics			Outcome	
First Author	Patients, n	Procedures, n	Preoperative	Postoperative	First Postoperative Time Point	Reduced Caloric Response	No Caloric Function
Parnes (1991)[25]	6	6	No	No	—	—	—
Hawthorne (1994)[22]	15	15	Yes (13 of 15)[a]	Yes (8 of 15)	Unknown	2 of 8 with a mild vestibular difference of 28% and 31%	1 of 8 without caloric response
Zappia (1996)[41]	8	8	No	No	—	—	—
Pulec (1997)[42]	17	17	Yes	Yes[b]	Unknown	No information	No information
Kisilevsky (2009)[23]	30	32	Yes (32 of 32)[c]	Yes (23 of 32)[d]	Unknown	7 of 23 with caloric deterioration	3 of 23 lost vestibular function (1 Ménière's disease, 2 with preoperative reduction)
Ahmed (2012)[21]	53	55	Yes (50 of 53)[e]	Yes (37 of 53)[f]	1 wk	5 of 37 with reduced caloric response (30%-78%)	5 of 37 without caloric response (98%-100%)
Beyea (2012)[20]	61	65	No	No	—	—	—
Ramakrishna (2012)[19]	6	12	No	No	—	—	—

[a]Three of 13 patients with preoperative vestibular hypofunction on the unaffected side.
[b]"Electronystagmography demonstrated a caloric response in the operated ear which was equal to or better than the preoperative test."
[c]Preoperatively, 6 patients with ipsilateral reduced caloric response; 1 patient with bilateral reduced caloric response.
[d]Due to logistical reasons (increased patient workload and a wider referral base), postoperative electronystagmography testing was performed in 23 of 32 cases.
[e]Preoperatively, 14 patients with canal paresis >25% on the affected side, of which 2 had Ménière's disease, 1 vestibular neuritis, and 1 herpes zoster.
[f]After posterior canal occlusion, all patients who reported vertigo or had a clinically positive lateral canal head impulse test result at 1 week had progress caloric testing.

Complications

All studies reported whether patients experienced complications. Five studies did not report any serious complication, which we defined as an unintended and undesired event or condition during or following medical treatment that causes incurable damage or that is so detrimental to the health of the patient that adjustment of the medical treatment is necessary. Two studies reported a postoperative perilymph fistula[21,25]; 1 study reported a wound infection[21]; another reported postoperative otitis media with effusion[25]; and 1 mentioned dislocation of the incus causing 30-dB conductive hearing loss.[22]

Discussion

Summary of the Evidence

This review shows the complexity of summarizing fair-quality retrospective studies.

One concise literature review has been published on this topic, and it concluded that the surgery delivers good results with minimal risks for hearing.[26] The authors included studies with different surgical techniques (eg, laser irradiation) for different types of BPPV, examining the results on vertigo and auditory function. Their outcomes are slightly different from our results, since they described a few patients with no complete resolution of BPPV and did not report any patients with postoperative deafness.

Although complete resolution of BPPV was reported in all studies included in our literature review, this has to be interpreted with caution, given that only 3 out of 8 studies included pre- and postoperative Dix-Hallpike results. Objective indications for surgery and for results of surgery are lacking in the majority of studies. Patient data are not entirely comparable across studies, because the subjects are not comparable with regard to indication for surgery and preoperative Dix-Hallpike responses. Moreover, patients are not consecutive; the outcomes are not uniformly reported; and follow-up data are inconsistent. Furthermore, 1 study presented 2 patients with a failed singular neurectomy prior to occlusion of the posterior semicircular canal. Since there was no resolution of BPPV after singular neurectomy, the diagnosis might have been incorrect. This emphasizes the importance of an objective diagnostic measurement, such as the Dix-Hallpike maneuver. Also, outcomes on resolution of BPPV might be inaccurate, given that caloric tests solely measure the lateral semicircular canal. In case of a postoperative total loss of vestibular function, stimuli from the otoconia will not be processed, because there is no vestibular function.

In contrast to the lacking objective results for surgery regarding the primary outcome, all studies reported pre- and postoperative audiometric results. Postoperatively, nearly all patients experienced transient conductive or sensorineural hearing loss. One study attributed the conductive loss to a hemotympanum. Other studies attributed the transient sensorineural loss to a perilymph fistula, labyrinthitis, or an infection of the mastoid. One patient had conductive hearing loss due to a dislocation of the incus, which is a possible but very rare complication of a canal occlusion with a transmastoid approach. Of the 2 patients who had a Barany deaf ear, 1 preoperatively experienced severe sensorineural hearing loss with a threshold >80 dB. The other patient became deaf due to the surgery.

Only 3 studies provided information on postoperative vestibular function measured by caloric responses. Hence, the overall percentage of patients with a reduced caloric response is unknown, since only a minority were tested. Among those patients tested, more than one-third had reduced vestibular function or had totally lost their caloric responses. In 2 studies, no further information on clinical symptoms of vestibular loss was provided. Ahmed et al[21] indicated that 2 patients reported a short-term period of acute vertigo and nausea and that vestibular loss resulted in mild permanent imbalance in 5 patients.

Although all studies reported whether patients experienced complications, some studies did not include secondary outcomes relating to vestibular function and hearing outcomes. Determining the true risk of the procedure according to data in this review is therefore difficult.

Limitations

Title and abstract screening was executed by the first and second authors, and the assessment of risk of bias for individual studies was done by the first and last authors. Furthermore, the reviewing process was limited to published articles only, and no correspondence was established with authors to obtain unpublished data. Hence, publication bias might have influenced the conclusion of this review.

The overall evidence was considered weak because the quality of individual studies was rated fair, due to the heterogeneity of indications for surgery and inconsistent follow-up data. Moreover, all studies carried the risk of selection and information bias because of the retrospective designs and the low numbers of included patients and because results were not compared with a control group. Given that not a single study included a control group, it remains unclear whether the resolution of vertigo may be from canal plugging, from a placebo effect of the surgery, or from spontaneous resolution.

Because of the shortage of objective criteria for surgery, other etiologies of intractable vertigo may be have been overlooked. Vertigo attacks could be the result of BPPV as well as other peripheral vestibular disorders. Recurrent vestibulopathy, vestibular paroxysm, vestibular migraine, and Ménière's disease are known causes of paroxysmal vertigo.[27-29] The duration of attacks is often longer, and attacks also occur without a change of position. In case of Ménière's disease, attacks are often accompanied by ear symptoms. Nevertheless, especially with subjective measurements, it can be difficult to distinguish BPPV from other peripheral vestibular disorders. In addition, vascular conditions, cerebral tumors, and, rarely, medical or psychiatric disorders can mimic BPPV.[30-33]

This review solely studied articles focusing on posterior canal plugging as a surgical therapy for BPPV. Other surgical therapies, such as singular neurectomy, were not

included in this review.[34,35] There are very few articles focusing on this particular operation. The large case series of Silverstein et al and Gacek et al showed complete resolution of vertigo between 80% and 97% of patients, with approximately 4% having deafness and severe sensorineural hearing loss.[36,37] However, smaller case series reported higher rates of sensorineural hearing loss, up to 42%, and reduced caloric responses.[38-40]

Conclusion

Posterior canal plugging resulted in 100% resolution of BPPV in patients with intractable BPPV in all studies. However, the true effectiveness and risks are difficult to determine because of the lack of objective data and the fair quality of studies. One should always take into account the possibility of serious complications, such as deafness and loss of vestibular function. In the future, prospective cohort studies should be conducted that describe the outcome measures and use comparable time spans between surgery and postoperative tests.

Author Contributions

Britta D. P. J. Maas, study design, literature search, title/abstract screening, full text screening, data analysis, writing article, final approval; **Hester J. van der Zaag-Loonen**, study design, title/abstract screening, revising manuscript, final approval; **Peter Paul G. van Benthem**, study design, revising manuscript, final approval; **Tjasse D. Bruintjes**, study design, data analysis, revising manuscript, final approval.

Disclosures

Competing interests: None.

Sponsorships: None.

Funding source: None.

Supplemental Material

Additional supporting information is available in the online version of the article.

References

1. Froehling DA, Silverstein MD, Mohr DN, Beatty CW, Offord KP, Ballard DJ. Benign positional vertigo: incidence and prognosis in a population-based study in Olmsted County, Minnesota. *Mayo Clin Proc*. 1991;66:596-601.
2. von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry*. 2006;78:710-715.
3. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology*. 1987;37:371-378.
4. Furman JM, Cass SP. Benign paroxysmal positional vertigo. *N Engl J Med*. 1999;341:1590-1596.
5. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). *CMAJ*. 2003; 169:681-693.
6. Lynn S, Pool A, Rose D, Brey R, Suman V. Randomized trial of the canalith repositioning procedure. *Otolaryngol Head Neck Surg*. 1995;113:712-720.
7. Burton MJ, Eby TL, Rosenfeld RM. Extracts from the Cochrane Library: modifications of the Epley (canalith repositioning) maneuver for posterior canal benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2012;147:407-411.
8. Bruintjes TD, Companjen J, van der Zaag-Loonen HJ, van Benthem PPG. A randomised sham-controlled trial to assess the long-term effect of the Epley manoeuvre for treatment of posterior canal benign paroxysmal positional vertigo. *Clin Otolaryngol*. 2014;39:39-44.
9. Oghalai JS, Manolidis S, Barth JL, Stewart MG, Jenkins HA. Unrecognized benign paroxysmal positional vertigo in elderly patients. *Otolaryngol Head Neck Surg*. 2000;122:630-634.
10. Lopez-Escamez JA, Gamiz MJ, Fernandez-Perez A, Gomez-Fiñana M. Long-term outcome and health-related quality of life in benign paroxysmal positional vertigo. *Eur Arch Otorhinolaryngol*. 2005;262:507-511.
11. Prokopakis E, Vlastos IM, Tsagournisakis M, Christodoulou P, Kawauchi H, Velegrakis G. Canalith repositioning procedures among 965 patients with benign paroxysmal positional vertigo. *Audiol Neurotol*. 2013;18:83-88.
12. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Neck Surg*. 1992;107:399-404.
13. Citron L, Hallpike CS. Observations upon the mechanism of positional nystagmus of the so-called benign paroxysmal type. *J Laryngol Otol*. 1956;70:253-259.
14. Citron L, Hallpike CS. A case of positional nystagmus of the so-called benign paroxysmal type and the effects of treatment by intracranial division of the VIIIth nerve. *J Laryngol Otol*. 1962;76:28-33.
15. Gacek RR. Transection of the posterior ampullary nerve for the relief of benign paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol*. 1974;83:596-605.
16. Parnes LS, McClure JA. Posterior semicircular canal occlusion for intractable benign paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol*. 1990;99(5, pt 1):330-334.
17. Bhattacharyya N, Gubbels SP, Schwartz SR, et al. Clinical practice guideline: benign paroxysmal positional vertigo (update). *Otolaryngol Neck Surg*. 2017;156(3):S1-S47.
18. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred Reporting Items for Systematic Reviews and Meta-analyses: the PRISMA statement. *J Clin Epidemiol*. 2009;62: 1006-1012.
19. Ramakrishna J, Goebel JA, Parnes LS. Efficacy and safety of bilateral posterior canal occlusion in patients with refractory benign paroxysmal positional vertigo: case report series. *Otol Neurotol*. 2012;33:640-642.
20. Beyea JA, Agrawal SK, Parnes LS. Transmastoid semicircular canal occlusion: a safe and highly effective treatment for benign paroxysmal positional vertigo and superior canal dehiscence. *Laryngoscope*. 2012;122:1862-1866.
21. Ahmed RM, Pohl DV, MacDougall HG, Makeham T, Halmagyi GM. Posterior semicircular canal occlusion for intractable benign positional vertigo: outcome in 55 ears in 53 patients operated upon over 20 years. *J Laryngol Otol*. 2012;126:677-682.
22. Hawthorne M, el-Naggar M. Fenestration and occlusion of posterior semicircular canal for patients with intractable

benign paroxysmal positional vertigo. *J Laryngol Otol*. 1994; 108:935-939.
23. Kisilevsky V, Bailie NA, Dutt SN, Rutka JA. Lessons learned from the surgical management of benign paroxysmal positional vertigo: the University Health Network experience with posterior semicircular canal occlusion surgery (1988-2006). *J Otolaryngol Head Neck Surg*. 2009;38:212-221.
24. Shekelle PG, Woolf SH, Eccles M, Grimshaw J. Developing clinical guidelines. *West J Med*. 1999;170:348-351.
25. Parnes LS, McClure JA. Posterior semicircular canal occlusion in the normal hearing ear. *Otolaryngol Head Neck Surg*. 1991; 104:52-57.
26. Leveque M, Labrousse M, Seidermann L, Chays A. Surgical therapy in intractable benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2007;136:693-698.
27. van Esch BF, van Wensen E, van der Zaag-Loonen HJ, Benthem PPG van, van Leeuwen RB. Clinical characteristics of benign recurrent vestibulopathy. *Otol Neurotol*. 2017;38: e357-e363.
28. Strupp M, Brandt T. Peripheral vestibular disorders. *Curr Opin Neurol*. 2013;26:81-89.
29. von Brevern M, Radtke A, Clarke AH, Lempert T. Migrainous vertigo presenting as episodic positional vertigo. *Neurology*. 2004;62:469-472.
30. Dunniway HM, Welling DB. Intracranial tumors mimicking benign paroxysmal positional vertigo. *Otolaryngol Neck Surg*. 1998;118:429-436.
31. Power L, Murray K, Drummond KJ, Trost N, Szmulewicz DJ. Fourth ventricle ependymoma mimicking benign paroxysmal positional vertigo. *Neurology*. 2018;91:327-328.
32. Heidenreich KD, Carender WJ, Heidenreich MJ, Telian SA. Strategies to distinguish benign paroxysmal positional vertigo from rotational vertebrobasilar ischemia. *Ann Vasc Surg*. 2010; 24:553.e1-553.e5.
33. Meurer WJ, Low PA, Staab JP. Medical and psychiatric causes of episodic vestibular symptoms. *Neurol Clin*. 2015;33: 643-659.
34. Nguyen-Huynh AT, Jackler RK, Pfister M, Tseng J. The aborted early history of the translabyrinthine approach. *Otol Neurotol*. 2007;28:269-279.
35. Cawthorne T. Labyrinthectomy. *Ann Otol Rhinol Laryngol*. 1960;69:1170-1178.
36. Silverstein H, White DW. Wide surgical exposure for singular neurectomy in the treatment of benign positional vertigo. *Laryngoscope*. 1990;100:701-706.
37. Gacek RR, Gacek MR. Results of singular neurectomy in the posterior ampullary recess. *ORL J Otorhinolaryngol Relat Spec*. 2002;64:397-402.
38. Meyerhoff WL. Surgical section of the posterior ampullary nerve. *Laryngoscope*. 1985;95:933-935.
39. Epley JM. Singular neurectomy: hypotympanotomy approach. *Otolaryngol Head Neck Surg*. 1979;88:304-309.
40. Gacek MR, Gacek RR, Martell R. Effect of singular neurectomy on the caloric response. *Am J Otolaryngol*. 1995;16:362-366.
41. Zappia JJ. Posterior semicircular canal occlusion for benign paroxysmal positional vertigo. *Am J Otol*. 1996;17:749-754.
42. Pulec JL. Ablation of posterior semicircular canal for benign paroxysmal positional vertigo. *Ear Nose Throat J*. 1997;76:17-22, 24.