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Hearing Loss following Posterior Fossa Microvascular Decompression: A Systematic Review

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Abstract

Objectives.—(1) Determine the prevalence of hearing loss following microvascular decompression (MVD) for trigeminal neuralgia (TN) and hemifacial spasm (HFS). (2) Demonstrate factors that affect postoperative hearing outcomes after MVD.

Data Sources.—PubMed-NCBI, Scopus, CINAHL, and PsycINFO databases from 1981 to 2016.

Review Methods.—Systematic review of prospective cohort studies and retrospective reviews in which any type of hearing loss was recorded after MVD for TN or HFS. Three researchers extracted data regarding operative indications, procedures performed, and diagnostic tests employed. Discrepancies were resolved by mutual consensus.

Results.—Sixty-nine references with 18,233 operations met inclusion criteria. There were 7093 patients treated for TN and 11,140 for HFS. The overall reported prevalence of hearing loss after MVD for TN and HFS was 5.58% and 8.25%, respectively. However, many of these studies relied on subjective measures of reporting hearing loss. In 23 studies with consistent perioperative audiograms, prevalence of hearing loss was 13.47% for TN and 13.39% for HFS, with no significant difference between indications ($P = .95$). Studies using intraoperative brainstem auditory evoked potential monitoring were more likely to report hearing loss for TN (relative risk [RR], 2.28; $P < .001$) but not with HFS (RR, 0.88; $P = .056$).

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Author Contributions

Matthew Bartindale, design, data acquisition/interpretation, writing and revising manuscript, approval; **Matthew Kircher**, conception and design, writing and revising manuscript, approval; **William Adams**, data analysis and interpretation, revising manuscript, approval; **Neelam Balasubramanian**, data analysis and interpretation, revising manuscript, approval; **Jeffrey Liles**, data acquisition, revising manuscript, approval; **Jason Bell**, data acquisition, revising manuscript, approval; **John Leonetti**, conception and design, manuscript revision and approval.

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Supplemental Material

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

Conclusion.—Conductive and sensorineural hearing loss are important complications following posterior fossa MVD. Many studies have reported on hearing loss using either subjective measures and/or inconsistent audiometric testing. Routine perioperative audiogram protocols improve the detection of hearing loss and may more accurately represent the true risk of hearing loss after MVD for TN and HFS.

Keywords

microvascular decompression; cerebellopontine angle; posterior fossa; complications; hearing loss; neurotology

Dandy[1] first proposed the neurovascular conflict theory in 1934 when he suggested that ectatic vessels could cause clinical syndromes. In the late 1960s, Jannetta[2] developed microvascular decompression (MVD), where the offending artery is transposed away from the cranial nerve root exit zone. MVD has been found to be an effective and safe treatment for several pathologies, most commonly trigeminal neuralgia (TN) and hemifacial spasm (HFS).[2–5]

Trigeminal neuralgia is a clinical syndrome manifesting as a lancinating unilateral facial pain in the sensory distribution of the trigeminal nerve.[6] The annual incidence of TN is 4 to 13 cases per 100,000 people per year.[7,8] First-line treatments are medical, but for those patients with symptoms intractable to medical treatment or with intolerable medication side effects, MVD is an effective surgical option.[3] HFS is characterized by irregular, involuntary, and recurring contractions of unilateral facial muscles.[4] The annual incidence of HFS is 9.8 to 11.0 cases per 100,000 people per year.[9] Medical therapy and botulinum toxin injection are less invasive treatment alternatives, but MVD has been shown to provide consistent long-lasting relief.[5] Other indications for MVD surgery include glossopharyngeal neuralgia,[10] occipital neuralgia,[11] tinnitus,[12] and vagal palsy.[13] Figure 1 shows the view the neurosurgeon has when performing a microvascular decompression surgery.

An important complication of posterior fossa MVD is hearing loss. Potential causes of sensorineural hearing loss (SNHL) previously discussed include stretching of cranial nerve (CN) VIII while retracting the cerebellum, direct trauma to CN VIII, manipulation of the labyrinthine artery or the anteroinferior cerebellar artery, neocompression of the nerve by the spacer material, and acoustic trauma from drill noise.[14–16] Conductive hearing loss (CHL) is usually caused by a middle ear effusion secondary to fluid entering the mastoid air cells during craniotomy.[16] Brainstem auditory evoked potential monitoring (BAEP) is used at many institutions and has previously been shown to decrease the prevalence of postoperative hearing loss.[17]

While numerous articles have been written regarding hearing loss as a complication of MVD, no meta-analysis or systematic review has investigated the true prevalence of hearing loss as a postoperative complication. Surprisingly, many centers report on hearing outcomes after MVD without the use of perioperative audiograms. In patients requiring MVD for TN or HFS, we aimed to determine the frequency of hearing loss as a complication and evaluate the impact of the indication for surgery, perioperative audiogram protocols, use of BAEP, and use of fully endoscopic technique by performing a systematic literature review.

Methods

Eligibility Criteria

(1) Patients must have been treated with MVD, (2) the reference had to report hearing loss, (3) the indication for included surgeries had to be either TN or HFS, (4) the complications had to be matched with the indication for MVD, and (5) the patients in the study could not be selected based on specific factors related to hearing loss (eg, intraoperative BAEP changes).

Search Strategy

To identify relevant studies, searches were performed in PubMed-NCBI, Scopus, PsycINFO, and CINAHL by an academic librarian. Search strategies are included in the appendix (available in the online version of the article). Only articles printed in English were included, and no year filter was used. The search was conducted according to PRISMA guidelines.[18] An institutional review board exemption was granted because human subjects were not involved in this study.

Study Selection and Validation

Two reviewers both screened the abstracts and then evaluated the remaining full articles for eligibility. Discrepancies were resolved by a third reviewer.

Data Abstraction

Information extracted from each study included author, year of publication, number of patients who underwent MVD for each indication, number of patients with each indication with hearing loss, number of patients with transient vs permanent hearing loss, usage of a perioperative audiogram protocol, use of BAEP, whether revision surgeries were included, whether the study was retrospective or prospective, and the study's level of evidence based on the Oxford Centre for Evidence Based Medicine 2011 criteria.[19] If perioperative audiograms were performed, the type of hearing loss, laterality of hearing loss, and hearing loss threshold used were documented if recorded. If audiograms were not mentioned, it was assumed that there was no audiogram protocol in place. The data were entered into an electronic research database (REDCap).[20]

Assessment of Bias of Individual Studies

The National Institutes of Health's (NIH's) Quality Assessment of Case Series Studies[21] was used to evaluate bias of individual studies. Figure 2 shows the criteria used in this assessment.

Statistical Methods

All statistical analyses were conducted using SAS software version 9.4 (SAS Institute, Cary, North Carolina). Raw prevalence estimates for each article were first transformed using a double arcsine method to free them from their 0 to 1 range. These prevalence estimates were weighted using the inverse of the summation of their between- and within-study variances as described by Hedges and Veva.[22] Cochran's Q statistic was used as an assessment of the

heterogeneity between articles, and the pooled prevalence of hearing loss was estimated using a weighted linear random-effects model with a back transformation to the original 0 to 1 raw prevalence scale as described by Barendregt et al.[23]

A weighted linear random-effects model was also used to evaluate moderators of hearing loss, including whether hearing loss varied by studies using BAEP, routine audios, or endoscopic procedures. In these models, the transformed estimates were weighted as described above, and random intercepts were allowed for each article. Model-derived pooled estimates were back transformed to the raw 0 to 1 prevalence scale as described by Barendregt et al,[23] and the relative ratio of hearing loss among articles with and without the moderator was estimated as described by Agresti.[24] Publication bias was evaluated visually using Light and Pillemer[25] funnel plots.

Results

Study Selection

A total of 481 studies were identified from the 4 databases searched. There were 50 duplicate studies removed. After implementation of our selection criteria, 309 studies were excluded based on their abstracts. The remaining 122 full articles were reviewed, and 69 studies fulfilled all inclusion criteria. The reasons for exclusion of studies are listed in Figure 3.

Study Characteristics

The studies that met inclusion criteria were published in English between 1981 and 2016. A total of 18,233 patients were described, with 7093 for TN and 11,140 for HFS. Twenty-two of the studies had a perioperative audiogram protocol in place (preoperative and postoperative audiograms), comprising 6530 of the patients. The threshold for what was considered hearing loss in those studies was inconsistent and often unreported (Table 1). Of these 23 studies, 8 reported whether the hearing loss was conductive or sensorineural, 9 reported whether the hearing loss was transient or permanent, and 10 reported laterality of the hearing loss. Forty-six studies had no audiogram protocol and relied on subjective complaints of hearing loss (Table 2). Intraoperative BAEP monitoring was consistently used in 21 studies. Four studies used fully endoscopic techniques for performing the surgery. Fourteen studies included some revisions surgeries, and the remaining 55 only included primary MVD. The assessment of bias for each study was recorded in Table 3.

True Prevalence Estimates of Hearing Loss across All Studies

There was a significant amount of heterogeneity in prevalence across the sampled TN articles ($Q = 262.33$, $P < .001$) and HFS articles ($Q = 388.29$, $P < .001$). Compared with HFS articles, the risk of hearing loss was lower among TN studies (relative risk [RR], 0.67; 95% confidence interval [CI], 0.60–0.76; $P < .001$), and consequently, a decision was made to stratify the results by indication.

Among TN studies ($\kappa = 35$), the estimated true prevalence of hearing loss in patients who underwent MVD was 5.58% (95% CI, 3.85%–7.61%). Figure 4 displays the forest plot of

individual study prevalence estimates as well as the overall pooled estimate for TN studies. Conversely, the estimated true prevalence of hearing loss among patients treated for HFS (κ = 47) is 8.25% (95% CI, 6.51%–10.18%). Figure 5 displays the forest plot for these estimates. Supplemental Tables S1 and S2 (available in the online version of the article) report the individual study sample sizes, prevalence estimates, and article weights.

Impact of Perioperative Audiograms on Hearing Loss Detection

Studies with routine perioperative audiograms detected significantly more hearing loss. TN studies that used perioperative audiograms were 3.64 times more likely to report hearing loss compared to TN studies not using routine audiograms (95% CI, 2.98–4.44; $P < .001$). Similarly, HFS studies using perioperative audiograms were 2.27 times more likely to report hearing loss compared to HFS studies not using routine audiograms (95% CI, 2.01–2.57; $P < .001$). An Altman and Bland test for interaction[26] confirmed routine audiograms had a larger impact on detection of hearing loss among patients with TN than HFS (z = 3.93, $P < .001$).

True Prevalence Estimates of Hearing Loss in Studies with Perioperative Audiograms

The estimated true prevalence of hearing loss in patients who underwent MVD for TN in studies with consistent perioperative audiograms (κ = 10) was 13.47% (95% CI, 7.79%–20.35%), while the estimated true prevalence for HFS (κ = 16) was 13.39% (95% CI, 9.39%–17.96%). In these studies, there was no significant difference in prevalence when comparing the 2 indications ($P = .95$). Of the studies that made these distinctions, 45.64% reported that hearing loss was conductive (κ = 8) and 67.12% indicated that it was transient (κ = 9). In the studies where the laterality of hearing loss was reported (κ = 10), 4.59% had a bilateral hearing loss and 1.53% had only a contralateral loss (κ = 10).

Impact of BAEP on Hearing Loss

Compared to TN articles not using BAEP, patients were 2.29 times more likely to report hearing loss when BAEP was used (95% CI, 1.70–3.08; $P < .001$). There was no meaningful difference between articles using or not using BAEP among HFS studies (RR, 0.88; 95% CI, 0.77–1.00; $P = .056$). Perioperative audiograms were performed more frequently in studies where BAEP was used. When BAEP was used, 58% of TN studies and 69% of HFS studies performed consistent audiograms. However, when BAEP was not used, consistent audiograms were only performed in 13% of TN studies and 21% of HFS studies.

Additional Findings

There was no difference in hearing loss between studies using or not using a fully endoscopic technique among TN articles (RR, 0.47; 95% CI, 0.10–2.12; $P = .32$) as well as among HFS articles (RR, 0.91; 95% CI, 0.45–1.84; $P = .81$). Studies that included revision surgeries showed a decreased prevalence of hearing loss among TN studies (RR, 0.69; 95% CI, 0.49–0.97; $P = .03$). However, HFS studies that included revision surgeries demonstrated a significantly higher prevalence of hearing loss compared to studies that excluded revision surgeries (RR, 1.80; 95% CI, 1.45–2.23; $P < .001$).

Regarding publication bias, Light and Pillemer[25] funnel plots revealed a high level of publication bias for both TN and HFS (Figures 6 and 7). As such, these results should be treated as nascent estimates of hearing loss following microvascular decompression for TN and HFS.

Discussion

Microvascular decompression has been consistently shown to be an effective treatment option for TN and HFS. However, as examined in this study, hearing loss is a relatively common complication.[3] Hearing loss can have a significant impact on quality of life,[27] particularly in the auditory frequencies critical to discriminating daily speech (500–2000 Hz).[28]

While many studies do mention hearing loss as a complication after MVD, standards used in the testing and reporting of hearing loss vary significantly between studies. In this review, nearly two-thirds of all reviewed studies (46 of 69 articles) relied solely on subjective assessment of hearing loss. This type of reporting is obviously inadequate yet surprisingly common. We found that studies with consistent perioperative audiograms are over 3 times more likely to detect hearing loss among patients with TN and over 2 times more likely with HFS. Shah et al[29] have shown that the sensitivity of patients subjectively reporting hearing loss is only 56.3% for class C or D hearing in the American Academy of Otolaryngology—Head and Neck Surgery (AAO-HNS) hearing classification system[30] and 17.2% for CHL. The timing of the audiogram, type of hearing loss, and hearing loss threshold are also important factors to consider in the diagnosis and management of these patients.

The distinction between conductive and sensorineural hearing loss is critical when the audiogram is performed shortly after surgery. This is because new-onset CHL after posterior fossa surgery is almost uniformly transient, as it results from a middle ear effusion secondary to fluid entering the mastoid air cells during craniotomy.[16] Timing of the audiogram then becomes important because an observation period of 2 to 3 months will allow for middle ear and mastoid fluid to resolve and conductive hearing to normalize in most patients.[31] SNHL, on the other hand, is more likely permanent and can result from a number of factors as previously mentioned earlier.

Hearing loss following MVD is not necessarily specific to the ipsilateral ear. Noise level exposures ranging from 117 to 122 dB in sound pressure level have been reported from drilling the cortical and mastoid bone.[32] It has been suggested that this acoustic trauma could lead to auditory threshold shifts.[33–35] These shifts are more likely to occur at high frequencies due to increased susceptibility to sound stimulation of the short outer hair cells in the high-frequency cochlear region compared to the less susceptible taller receptor cells in the low-frequency regions.[36,37] In animal models, this threshold shift has been found to be more likely to occur in older animals.[35] Some patients will recover their hearing over a period of time; therefore, long-term follow-up audiograms would be needed to detect these changes. Other theories for the cause of contralateral hearing loss after MVD include intraoperative brainstem shift, brainstem edema, labyrinthine fluid imbalance secondary to cerebrospinal fluid release, or venous congestion at the ipsilateral inferior colliculus.[38,39]

There is also considerable inconsistency in what qualifies as hearing loss across studies (Table 1). Many studies either did not report auditory thresholds or used variable end points. Too small of a threshold could lead to false positives due to testing error,[40] and too large of a threshold allows patients who have significant hearing loss to go unreported. It is important that speech discrimination as well as pure-tone average is involved in consideration of hearing loss. The most frequently used threshold in past studies includes a greater than 15 dB pure-tone average increase and/or 20% speech discrimination score decrease (Table 1). More detailed audiometry reporting standards are well established, including the 1995 AAO-HNS hearing classification system[30] and the more recently developed scattergram plots.[41]

Across all studies, MVD of CN VII had a significantly higher risk of hearing loss than MVD of CN V. When only studies with consistent audiograms were included, there was no significant difference between indications. Given the facial nerve's closer proximity to the vestibulocochlear nerve and important inner ear vasculature, a higher rate of hearing loss is not surprising. The facial nerve's location necessitates more high-risk manipulation as well as a higher possibility of neocompression from the spacer material. However, with the discrepancy in the 2 data analyses, no conclusion can definitively be made.

Surgeons have employed BAEP in the interest of detecting and avoiding hearing loss in MVD.[42,43] Interestingly, the risk of hearing loss increased when BAEP was used among TN articles, whereas it had no impact among HFS articles. It is not immediately clear why the prevalence of hearing loss was higher in TN studies using BAEP. It is possible that this is because authors who used BAEP were more likely to obtain audiograms for TN; however, that is also the case for studies focusing on HFS. It is possible that BAEP leads to overconfidence when surgeons decompress CN V, leading to less caution in cerebellar retraction. The actual cause of this observed difference is a possible topic for future study. The studies that used fully endoscopic technique showed no significant difference in rates of hearing loss compared to those that did not use fully endoscopic technique; however, this analysis was limited by the small number of these studies.

Perioperative audiograms should consist of a preoperative hearing test within 6 months of surgery and a postoperative hearing test approximately 2 to 3 months after surgery. This timing of the preoperative audiogram provides an up-to-date preoperative assessment and a baseline value to establish reasonable patient expectations. In patients with previously undetected preoperative hearing loss, a preoperative audiogram becomes an important medicolegal consideration when the patient has a postoperative hearing complaint. Appropriate timing of a postoperative audiogram allows time for a potential mastoid effusion to resolve and avoids false-positive hearing loss.

All studies have some limitations, and Light and Pillemer[25] funnel plots revealed a high level of publication bias in this meta-analysis. This may be because neurosurgeons who have higher than normal rates of hearing loss are less likely to publish. Worse, when they publish, they may be less likely to publicize their complication rates. The variability in reporting standards in the body of literature may also contribute to this uncertainty. Only additional original research will help improve the reported estimates of hearing loss among patients with MVD.

It is important that all measures are taken to document and protect hearing during MVD for TN and HFS. Even the most experienced surgeon taking all necessary precautions may still be faced with some cases of hearing loss after MVD. This hearing loss in many cases is likely a multifactorial pathologic process that is more complicated than simple nerve stretching or trauma and may represent patient susceptibility to a number of surgical factors.[44] Thus, it is critically important that patients are counseled regarding the risk to hearing after MVD for TN and HFS. In the 23 studies using routine perioperative audiograms, the estimated true prevalence of hearing loss was 13.47% and 13.39% among patients with TN and HFS, respectively. We believe these numbers are likely the most accurate representation of the prevalence of postoperative hearing loss and would be appropriate to quote when counseling patients regarding the risk of hearing loss following MVD.

Conclusion

Hearing loss is a potential complication following MVD for TN and HFS. In general, reporting on hearing loss after MVD has been of poor quality, with many studies relying on patient-reported subjective hearing loss and/or inadequate audiometric measures. Distinctions should be made between types of hearing loss (sensorineural or conductive), timing of audiometry, and standardizing audiometric reporting thresholds between studies. Routine perioperative audiogram protocols improve the detection of hearing loss and may better represent the true risk of hearing loss after MVD for TN and HFS.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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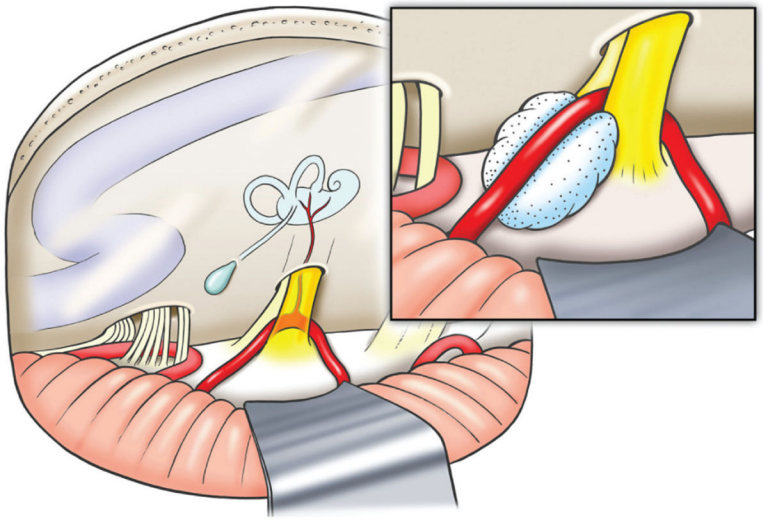

Figure 1.

The view the neurosurgeon has while performing a microvascular decompression in the posterior fossa. ©Jackler RK. *Atlas of Skull Base Surgery and Neurotology*. Stuttgart, Germany: Thieme; 2017.[105]

1. Was the study question or objective clearly stated?
2. Was the study population clearly and fully described, including a case definition?
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?

Figure 2.
Criteria for the National Institutes of Health's Quality Assessment of Case Series Studies. Source: National Heart, Lung, and Blood Institute, National Institutes of Health.[21]

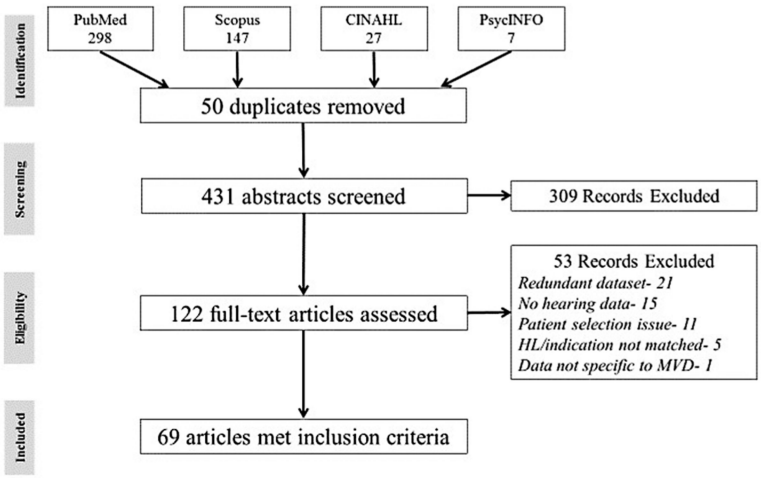

Figure 3.

Study selection process and reasons for exclusion. HL, hearing loss; MVD, microvascular decompression.

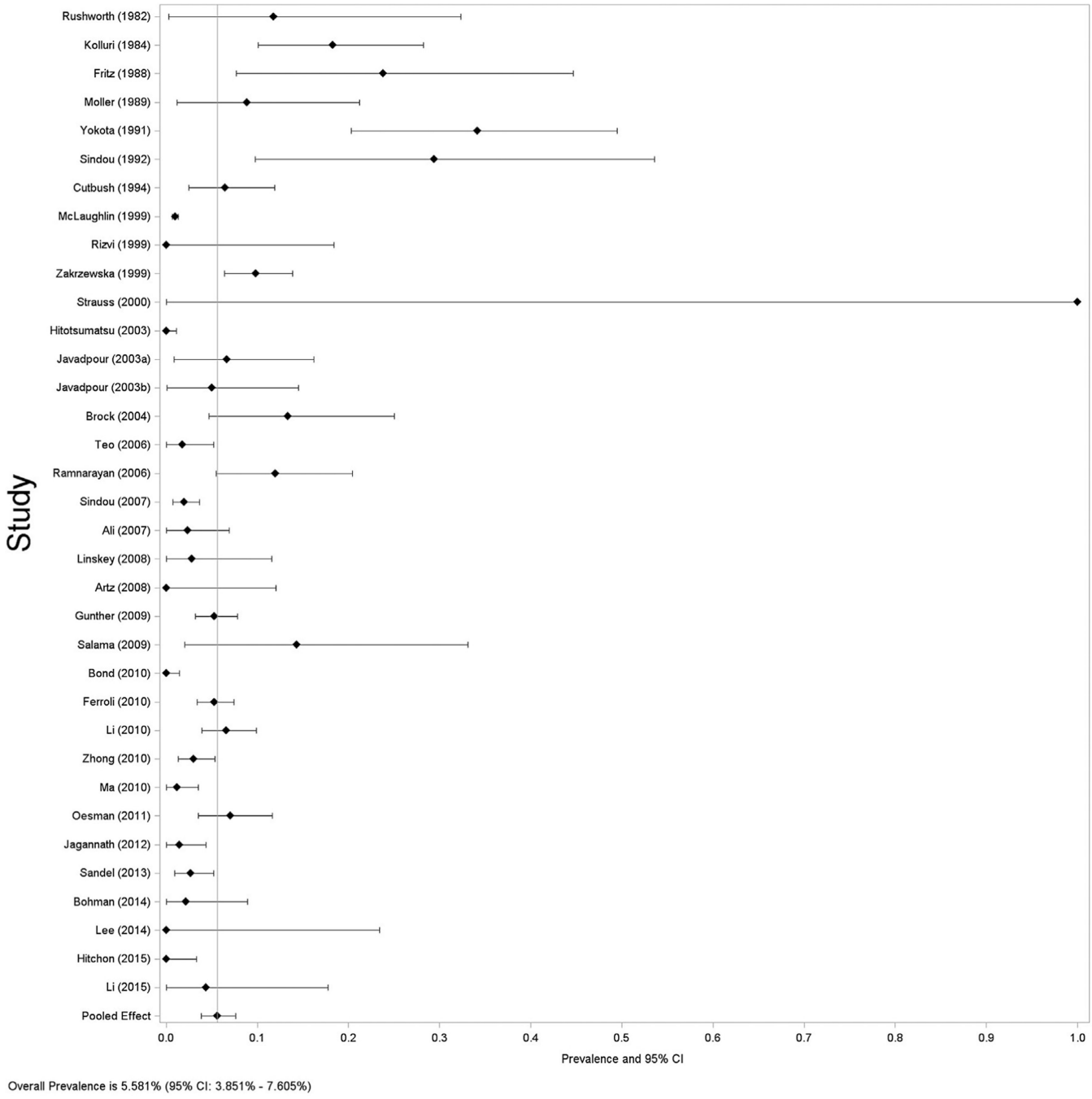

Figure 4.

Forest plot for hearing loss in patients who underwent microvascular decompression for trigeminal neuralgia across all studies.

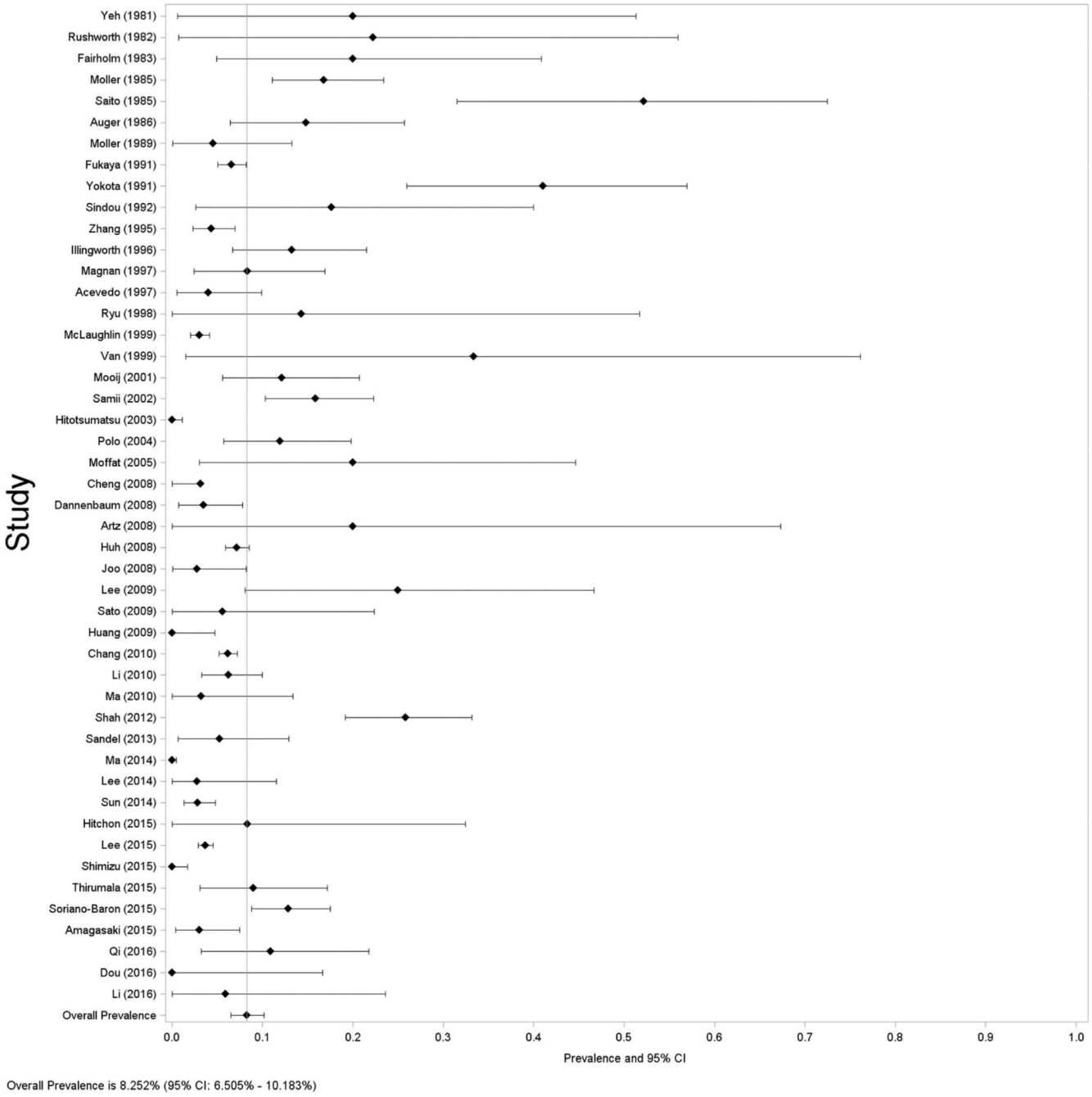

Figure 5.

Forest plot for hearing loss in patients who underwent microvascular decompression for hemifacial spasm across all studies

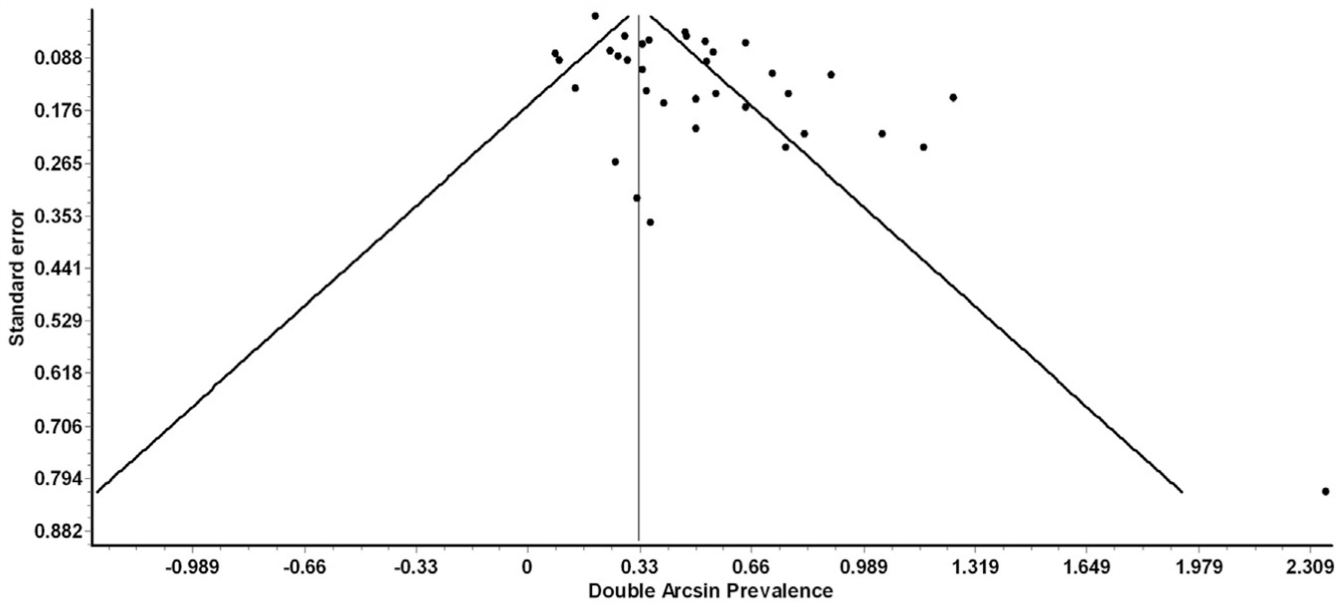

Figure 6.
Publication bias in trigeminal neuralgia studies.

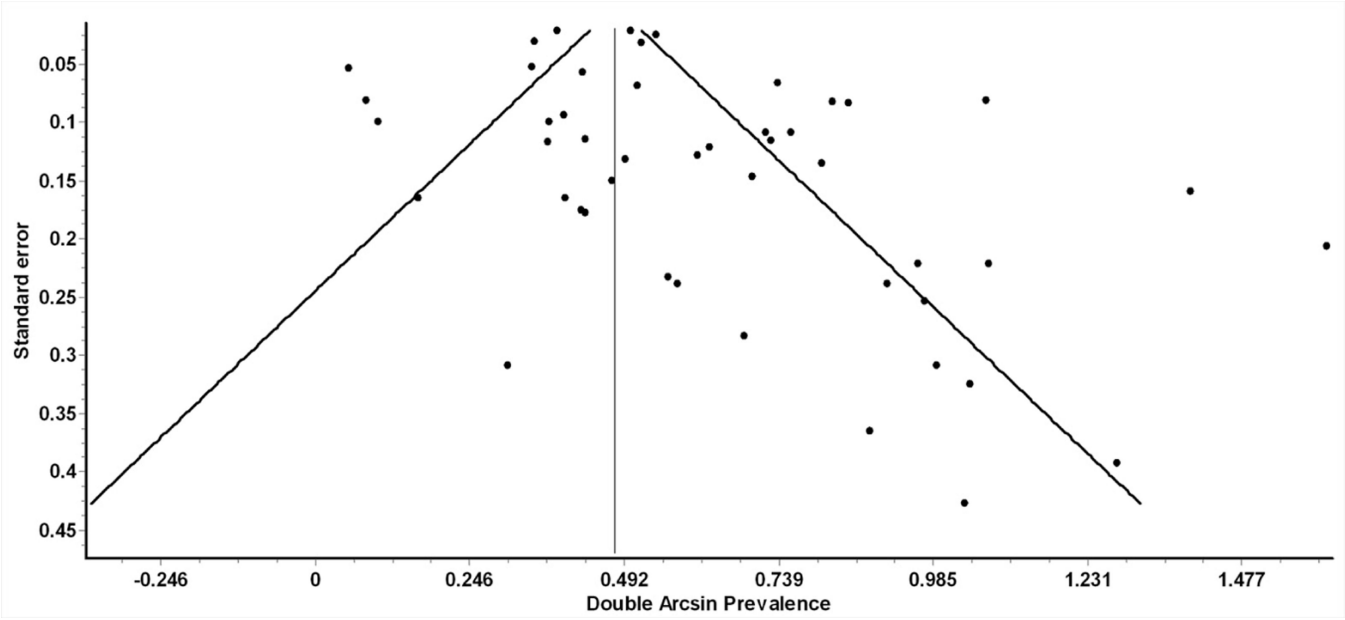

Figure 7.
Publication bias in hemifacial spasm studies.

Table 1.

Studies That Used Perioperative Audiometry and Their Hearing Loss Thresholds.

First Author	PY	Cases	HL Threshold	BAEP	Rev.	C/SN	R/P	CEBM
Lee[15]	2015	2027	>15 dB PTA or 20% SDS	Yes	No	Yes	R	4
Thirumala[44]	2015	67	>15 dB PTA	Yes	Yes	No	R	4
Shimizu[45]	2015	100	NR	Yes	No	No	P	4
Soriano-Baron[46]	2015	226	NR	No	Yes	No	R	4
Shah[29]	2012	151	>15 dB PTA or 20% SDS	Yes	No	Yes	R	4
Ferroli[47]	2010	476	NR	No	No	No	R	4
Gunther[48]	2009	362	NR	No	No	No	R	4
Lee[49]	2009	20	>10 dB PTA	No	No	Yes	P	4
Huh[50]	2008	1524	>25 dB PTA	Yes	No	Yes	R	4
Ramnarayan[51]	2006	75	>20 dB PTA	Yes	No	Yes	P	4
Moffat[52]	2005	15	NR	No	No	Yes	R	4
Brock[53]	2004	45	>20 dB PTA	Yes	Yes	No	R	4
Polo[17]	2004	84	>20 dB PTA	Yes	No	No	P	4
Strauss[38]	2000	1	>20 dB PTA	Yes	No	No	R	4
Van[54]	1999	6	>35 dB PTA	No	No	Yes	P	4
Rizvi[55]	1999	9	NR	Yes	No	No	P	4
Sindou[43]	1992	34	>20 dB PTA	Yes	No	No	R	4
Fukaya[56]	1991	963	NR	No	No	Yes	R	4
Yokota[57]	1991	80	>15 dB PTA	Yes	No	No	P	4
Moller[42]	1989	78	>10 dB PTA or 15% SDS	Yes	Yes	No	R	4
Fritz[58]	1988	21	>15 dB PTA or 20% SDS	No	No	Yes	R	4
Moller[59]	1985	143	>10 dB PTA	No	No	No	R	4
Saito[60]	1985	23	>10 dB PTA	No	No	Yes	R	4

Abbreviations: BAEP, brainstem auditory evoked potential monitoring; C/SN, distinguishes conductive and sensorineural hearing loss; CEBM, Centre for Evidence-Based Medicine Level of Evidence; HL, hearing loss; NR, not reported; PTA, pure-tone average; PY, publication year; Rev., revisions included; R/P, retrospective or prospective; SDS, speech discrimination score.

Table 2.

Studies That Did Not Use Perioperative Audiometry.

First Author	PY	Cases, No.	BAEP	Rev.	R/P	CEBM
Qi[4]	2016	46	No	No	R	4
Dou[61]	2016	10	No	No	R	4
Li[62]	2016	17	No	No	R	4
Hitchon[5]	2015	63	No	No	R	4
Li[63]	2015	23	No	No	R	4
Amagasaki[64]	2015	100	Yes	No	R	4
Bohman[65]	2014	47	No	Yes	R	4
Ma[66]	2014	348	No	No	R	4
Lee[67]	2014	43	Yes	No	R	4
Sun[68]	2014	356	No	No	R	4
Sandel[69]	2013	283	No	No	R	4
Jagannath[70]	2012	137	No	Yes	P	4
Oesman[71]	2011	156	No	No	R	4
Bond[72]	2010	119	Yes	Yes	R	4
Chang[73]	2010	2137	No	No	R	4
Li[74]	2010	481	No	No	R	4
Ma[75]	2010	200	No	No	R	4
Zhong[76]	2010	298	No	No	R	4
Sato[77]	2009	18	No	No	R	4
Huang[78]	2009	36	Yes	No	P	4
Salama[79]	2009	21	No	No	P	4
Linskey[80]	2008	35	Yes	No	P	4
Cheng[81]	2008	32	No	No	R	4
Dannenbaum[82]	2008	114	No	No	R	4
Artz[83]	2008	19	No	No	R	4
Joo[84]	2008	72	Yes	No	R	4

First Author	PY	Cases, No.	BAEP	Rev.	R/P	CEBM
Ali[85]	2007	86	No	No	P	4
Sindou[86]	2007	362	No	Yes	R	4
Teo[87]	2006	114	No	No	R	4
Zakrzewska[88]	2005	245	No	No	R	4
Hitotsumatsu[89]	2003	300	No	No	R	4
Javadpour[90]	2003	85	Yes	Yes	R	4
Samii[91]	2002	145	No	Yes	R	4
Mooij[92]	2001	74	No	Yes	R	4
McLaughlin[93]	1999	4265	No	No	R	4
Ryu[94]	1998	7	No	No	R	4
Magnan[95]	1997	60	No	No	R	4
Acevedo[96]	1997	75	No	No	R	4
Illingworth[97]	1996	83	No	No	P	4
Zhang[98]	1995	300	No	No	R	4
Cutbush[99]	1994	109	No	No	R	4
Auger[100]	1986	54	No	Yes	R	4
Kolluri[101]	1984	71	No	No	R	4
Fairholm[102]	1983	20	No	No	R	4
Rushworth[103]	1982	26	No	Yes	R	4
Yeh[104]	1981	10	No	No	R	4

Abbreviations: BAEP, brainstem auditory evoked potential monitoring; CEBM, Centre for Evidence-based Medicine Level of Evidence; PY, publication year; Rev., revisions included; R/P, retrospective or prospective.

Table 3.

Standardized Risk Assessment of Individual Studies Based on the NIH Quality Assessment Tool for Case Series Studies.[21,a]

First Author	PY	1. Objective?	2. Population Description?	3. Consecutive Cases?	4. Comparable Subjects?	5. Intervention Described?	6. Outcomes Defined?	7. Adequate Follow-up?	8. Well-Described Statistical Methods?	9. Well-Described Results?
Dou[61]	2016	Y	Y	N	Y	Y	N	Y	Y	Y
Li[62]	2016	Y	Y	Y	Y	Y	N	Y	NA	Y
Qi[4]	2016	Y	Y	CD	Y	Y	N	Y	Y	Y
Amagasaki[64]	2015	Y	Y	Y	Y	Y	N	CD	Y	Y
Hitchon[5]	2015	Y	Y	Y	Y	Y	N	Y	Y	Y
Lee[15]	2015	Y	Y	Y	Y	Y	Y	Y	Y	Y
Li63	2015	Y	Y	Y	Y	Y	N	Y	NA	Y
Shimizu[45]	2015	Y	Y	Y	Y	Y	N	CD	NA	Y
Soriano-Baron[46]	2015	Y	Y	CD	Y	Y	N	Y	Y	Y
Thirumala[44]	2015	Y	Y	Y	Y	Y	Y	N	Y	Y
Bohman[65]	2014	Y	Y	Y	Y	Y	N	Y	Y	Y
Lee[67]	2014	Y	Y	CD	Y	Y	N	Y	Y	Y
Ma[66]	2014	Y	Y	CD	Y	Y	N	Y	NA	Y
Sun[68]	2014	Y	Y	Y	Y	Y	N	Y	Y	Y
Sandel[69]	2013	Y	Y	Y	Y	Y	N	Y	Y	Y
Jagannath[70]	2012	Y	Y	Y	Y	Y	N	Y	NA	Y
Shah[29]	2012	Y	Y	CD	Y	Y	Y	N	Y	Y
Oesman[71]	2011	Y	Y	Y	Y	Y	N	Y	Y	Y
Bond[72]	2010	Y	Y	CD	Y	Y	N	Y	N	Y
Chang[73]	2010	Y	Y	CD	Y	Y	N	Y	Y	Y
Ferroli[47]	2010	Y	Y	CD	Y	Y	N	Y	Y	Y
Li[74]	2010	Y	Y	CD	Y	Y	N	Y	NA	Y
Ma[75]	2010	Y	Y	Y	Y	Y	N	Y	N	Y
Zhong[76]	2010	Y	Y	Y	Y	Y	N	CD	Y	Y

First Author	PY	1. Objective?	2. Population Description?	3. Consecutive Cases?	4. Comparable Subjects?	5. Intervention Described?	6. Outcomes Defined?	7. Adequate Follow-up?	8. Well-Described Statistical Methods?	9. Well-Described Results?
Gunther[48]	2009	Y	N	Y	Y	Y	N	Y	Y	Y
Huang[78]	2009	Y	N	Y	Y	Y	N	Y	N	Y
Lee[49]	2009	Y	Y	Y	Y	Y	Y	Y	Y	Y
Salama[79]	2009	Y	Y	Y	Y	Y	N	Y	Y	Y
Sato[77]	2009	Y	Y	N	Y	Y	N	CD	Y	Y
Artz[83]	2008	Y	N	CD	Y	Y	N	Y	Y	Y
Cheng[81]	2008	Y	Y	Y	Y	Y	N	Y	NA	Y
Dannenbaum[82]	2008	Y	Y	Y	Y	Y	N	Y	Y	Y
Huh[50]	2008	Y	Y	Y	Y	Y	Y	Y	Y	Y
Joo[84]	2008	Y	Y	CD	Y	Y	N	Y	Y	Y
Linskey[80]	2008	Y	Y	Y	Y	Y	N	Y	Y	Y
Ali[85]	2007	Y	Y	Y	Y	Y	N	CD	NA	Y
Sindou[86]	2007	Y	Y	Y	Y	Y	N	Y	Y	Y
Ramnarayan[51]	2006	Y	Y	CD	Y	Y	Y	Y	Y	Y
Teo[87]	2006	Y	Y	Y	Y	Y	N	Y	NA	Y
Moffat[52]	2005	Y	Y	CD	Y	Y	N	Y	NA	Y
Zakrzewska[88]	2005	Y	Y	N	Y	Y	N	Y	Y	Y
Brock[53]	2004	Y	N	Y	Y	Y	Y	Y	N	Y
Polo[17]	2004	Y	Y	Y	Y	Y	Y	Y	Y	Y
Hitotsumatsu[89]	2003	Y	N	CD	CD	Y	N	CD	N	Y
Javadpour[90]	2003	Y	N	Y	Y	Y	N	Y	Y	Y
Samii[91]	2002	Y	Y	Y	Y	Y	N	Y	NA	Y
Mooij[92]	2001	Y	Y	CD	Y	Y	N	Y	N	Y
Strauss[38]	2000	Y	Y	NA	Y	Y	Y	Y	NA	Y
McLaughlin[93]	1999	Y	N	Y	Y	Y	N	CD	N	Y
Rizvi[55]	1999	Y	N	CD	Y	Y	N	CD	N	Y
Van[54]	1999	Y	N	Y	Y	Y	Y	Y	N	Y
Ryu[94]	1998	Y	Y	N	Y	Y	N	Y	NA	Y

First Author	PY	1. Objective?	2. Population Description?	3. Consecutive Cases?	4. Comparable Subjects?	5. Intervention Described?	6. Outcomes Defined?	7. Adequate Follow-up?	8. Well-Described Statistical Methods?	9. Well-Described Results?
Acevedo[96]	1997	N	Y	Y	Y	Y	N	CD	NA	Y
Magnan[95]	1997	Y	Y	Y	Y	Y	N	Y	NA	Y
Illingworth[97]	1996	Y	Y	CD	Y	Y	N	Y	NA	Y
Zhang[98]	1995	N	Y	CD	Y	Y	N	Y	NA	Y
Cutbush[99]	1994	Y	Y	Y	Y	Y	N	Y	N	Y
Sindou[43]	1992	Y	Y	N	Y	Y	Y	Y	N	Y
Fukaya[56]	1991	Y	N	N	Y	Y	N	Y	N	Y
Yokota[57]	1991	Y	Y	CD	Y	Y	Y	Y	NA	Y
Moller[42]	1989	Y	N	Y	Y	Y	Y	Y	N	Y
Fritz[58]	1988	Y	N	N	Y	Y	Y	N	NA	Y
Auger[100]	1986	Y	Y	Y	Y	Y	N	Y	Y	Y
Moller[59]	1985	Y	Y	Y	Y	Y	Y	Y	NA	Y
Saito[60]	1985	Y	N	Y	Y	Y	Y	Y	NA	Y
Kolluri[101]	1984	Y	Y	Y	Y	Y	N	Y	N	Y
Fairholm[102]	1983	Y	Y	Y	Y	Y	N	Y	NA	Y
Rushworth[103]	1982	N	N	Y	Y	Y	N	CD	NA	Y
Yeh[104]	1981	N	Y	Y	Y	Y	N	Y	NA	Y

Abbreviations: CD, cannot determine; N, no; NA, not applicable; NIH, National Institutes of Health; PY, publication year; Y, yes.

[a] Full questions are listed in Figure 2.