The fate of cranial neuropathy after flow diversion for carotid aneurysms

Benjamin L. Brown, MD,¹ Demetrius Lopes, MD,² David A. Miller, MD,¹ Rabih G. Tawk, MD,¹ Leonardo B. C. Brasiliense, MD,¹ Andrew Ringer, MD,³ Eric Sauvageau, MD,⁴ Ciarán J. Powers, MD, PhD,⁵ Adam Arthur, MD,⁶ Daniel Hoit, MD,⁶ Kenneth Snyder, MD, PhD,⁷ Adnan Siddiqui, MD, PhD,⁷ Elad Levy, MD,⁷ L. Nelson Hopkins, MD,⁷ Hugo Cuellar, MD,⁸ Rafael Rodriguez-Mercado, MD,⁹ Erol Veznedaroglu, MD,¹⁰ Mandy Binning, MD,¹⁰ J Mocco, MD,¹¹ Pedro Aguilar-Salinas, MD,¹² Alan Boulous, MD,¹³ Junichi Yamamoto, MD, PhD,¹³ and Ricardo A. Hanel, MD, PhD⁴

¹Department of Neurosurgery, Mayo Clinic, Jacksonville, Florida; ²Department of Neurosurgery, Rush University Medical Center, Chicago, Illinois; ³Department of Neurosurgery, University of Cincinnati, Ohio; ⁴Lyerly Neurosurgery, Baptist Health, Jacksonville, Florida; ⁵Department of Neurological Surgery, The Ohio State University, Columbus, Ohio; ⁶Semmes Murphey Neurologic and Spine Institute, Semmes Murphey Clinic, Memphis, Tennessee; ⁷Department of Neurosurgery, University of Buffalo, New York; ⁸Department of Neurosurgery, Louisiana State University, Shreveport, Louisiana; ⁹Department of Neurosurgery, University of Puerto Rico Medical Sciences, San Juan, Puerto Rico; ¹⁰Capital Institute for Neurosciences, Capital Health, Trenton, New Jersey; ¹¹Department of Neurological Surgery, Vanderbilt University, Nashville, Tennessee; ¹²National Institute of Medical Sciences and Nutrition, Salvador Zubiran, Mexico City, Mexico; and ¹³Department of Neurosurgery, Albany Medical Center, Albany, New York

OBJECTIVE The authors sought to determine whether flow diversion with the Pipeline Embolization Device (PED) can approximate microsurgical decompression in restoring function after cranial neuropathy following carotid artery aneurysms.

METHODS This multiinstitutional retrospective study involved 45 patients treated with PED across the United States. All patients included presented between November 2009 and October 2013 with cranial neuropathy (cranial nerves [CNs] II, III, IV, and VI) due to intracranial aneurysm. Outcome analysis included clinical and procedural variables at the time of treatment as well as at the latest clinical and radiographic follow-up.

RESULTS Twenty-six aneurysms (57.8%) were located in the cavernous segment, while 6 (13.3%) were in the clinoid segment, and 13 (28.9%) were in the ophthalmic segment of the internal carotid artery. The average aneurysm size was 18.6 mm (range 4–35 mm), and the average number of flow diverters placed per patient was 1.2. Thirty-eight patients had available information regarding duration of cranial neuropathy prior to treatment. Eleven patients (28.9%) were treated within 1 month of symptom onset, while 27 (71.1%) were treated after 1 month of symptoms. The overall rate of cranial neuropathy improvement for all patients was 66.7%. The CN deficits resolved in 19 patients (42.2%), improved in 11 (24.4%), were unchanged in 14 (31.1%), and worsened in 1 (2.2%). Over time, the rate of cranial neuropathy improvement was 33.3% (15/45), 68.8% (22/32), and 81.0% (17/21) at less than 6, 6, and 12 months, respectively. At last follow-up, 60% of patients in the isolated CN II group had improvement, while in the CN III, IV, or VI group, 85.7% had improved. Moreover, 100% (11/11) of patients experienced improvement if they were treated within 1 month of symptom onset, whereas 44.4% (12/27) experienced improvement if they treated after 1 month of symptom onset; 70.4% (19/27) of those with partial deficits improved compared with 30% (3/10) of those with complete deficits.

CONCLUSIONS Cranial neuropathy caused by cerebral aneurysm responds similarly when the aneurysm is treated with the PED compared with open surgery and coil embolization. Lower morbidity and higher occlusion rates obtained with the PED may suggest it as treatment of choice for some of these lesions. Time to treatment is an important consideration regardless of treatment modality.

<http://thejns.org/doi/abs/10.3171/2015.4.JNS142790>

KEY WORDS aneurysm; cranial neuropathy; flow diversion; Pipeline Embolization Device; vascular disorders

ABBREVIATIONS CN = cranial nerve; PED = Pipeline Embolization Device.

SUBMITTED December 8, 2014. **ACCEPTED** April 8, 2015.

INCLUDE WHEN CITING Published online October 16, 2015; DOI: 10.3171/2015.4.JNS142790.

MICROSURGERY remains the gold standard for decompression of cranial nerves (CNs) affected by aneurysmal mass effect. Over the past 2 decades, coil embolization has gained rapid acceptance as a viable alternative. For the treatment of large unruptured aneurysms of the proximal internal carotid artery, flow diversion has grown in popularity as an alternative to both microsurgery and coil embolization. Cranial nerve compression is not an uncommon presenting symptom for these aneurysms. For cavernous aneurysms, the presenting symptom is diplopia in about 65% of patients, while decreased visual acuity affects approximately 16% of patients.³⁰

The question of whether endovascular flow diversion can approximate microsurgical decompression in restoring function in cranial neuropathy remains unanswered. To our knowledge, there have been no studies focusing on the results of cranial neuropathy after Pipeline Embolization Device (PED; Covidien) flow diversion. We set out to review this subject in the following multiinstitutional study.

Methods

This study was approved by each participating institution's review board. The medical records of patients presenting with cranial neuropathy (CNs II, III, IV, and VI) due to cerebral aneurysm treated with flow diversion between November 2009 and October 2013 were retrospectively reviewed. Ten institutions from North America, led by the Endovascular Neurosurgery Research Group, contributed patient data. Antiplatelet agents were used for all patients in the pre- and postoperative period; however, the antiplatelet agent and dosage varied per each institution's standard. All outcomes were assessed during routine office visits and were based on the clinical judgment of each physician at the treating institutions. All patients were treated using the PED. The PED was sized according to standard protocol to be larger than the maximum diameter of the target vessel to ensure good wall apposition. Additional devices were deployed at the surgeon's discretion to increase stasis within the aneurysm or to ensure complete coverage of a lesion if a longer construct was required.

Patient demographics, presentation deficit, duration of deficit prior to treatment, aneurysm demographics, number of flow diverters used, presence/absence of coils, cranial neuropathy outcome (resolved, improved, unchanged, or worse), clinical complications, and radiographic outcome (Raymond classification²⁸) were recorded. Subanalyses were performed for CN II and CNs III, IV, and VI. Cranial nerves III, IV, and VI were grouped together because of their similar function. Some patients involved in the study were also reported on in the Pipeline for Uncoilable or Failed Aneurysms Study.²

Statistical Analysis

The statistical analysis of this study was largely descriptive. Descriptive statistics such as mean and percentage were used to characterize the data. Univariate analysis was performed to understand which factors were associated with better outcome. Factors that were studied included duration of the deficit prior to treatment, complete

palsy, use of coils, follow-up time, Raymond classification (complete occlusion vs partial occlusion), and aneurysm size. Statistically significant difference in outcomes based on treatment parameters was determined by Fisher's exact test. A p value of < 0.05 was determined to be statistically significant. Odds ratios were computed when possible with their corresponding 95% confidence intervals. Statistical analysis was performed using JMP version 10.0.0 (SAS Institute).

Results

Forty-five patients met inclusion criteria for the study, representing 45 treated aneurysms of the proximal intracranial internal carotid artery. The demographic information for the patients is included in Table 1. There were 42 females (93%) and 3 males (7%). The mean age was 64.7 years (range 33–89 years). Of the 45 aneurysms, 26 (57.8%) were located in the cavernous segment, while 6 (13.3%) were in the clinoid segment, and 13 (28.9%) were in the ophthalmic segment. The average aneurysm size was 18.6 mm (range 4–35 mm). There were 19 aneurysms larger than 20 mm, 19 aneurysms between 10 and 20 mm, and 7 aneurysms smaller than 10 mm. The average number of flow diverters placed per patient was 1.2. Thirty-eight patients had available information regarding duration of cranial neuropathy prior to treatment. Eleven (28.9%) were treated within 1 month of symptom onset, while 27 (71.1%) were treated after having symptoms for 1 month.

TABLE 1. Patient demographics

Variable	Value*
No. of patients	45
Sex	
Male	3 (6.67)
Female	42 (93.33)
Age in yrs	
Mean	64.7
Range	33–89
Cranial neuropathy	
II	10 (22.2)
III	16 (35.6)
VI	11 (24.4)
III & IV	1 (2.2)
III & VI	4 (8.9)
III, IV, & VI	1 (2.2)
II, III, IV, & VI	2 (4.4)
Aneurysm location	
Cavernous segment	26 (57.8)
Paraclinoid segment	6 (13.3)
Ophthalmic segment	13 (28.9)
Comorbidities	
Tobacco use	12 (26.7)
Diabetes	2 (4.4)

* Values are number of patients (%) unless otherwise noted.

TABLE 2. Treatment and clinical demographics

Variable	Value*
Follow-up in mos	
Mean	8.4
Range	0.1–21.5
Raymond class (last follow-up; n = 40)	
I	26 (65)
II	9 (22.5)
III	5 (12.5)
Raymond class (12 mos; n = 13)	
I	11 (84.6)
II	2 (15.4)
III	0
30-day overall morbidity & mortality (n = 45)	
Total	8 (17.8)
Ischemic stroke	3 (6.7)
Hemorrhagic stroke	2 (4.4)
GI bleed	1 (2.2)
New CN deficit	1 (2.2)
MI	1 (2.2)
30-day major stroke or death	1 (2.2)

GI = gastrointestinal; MI = myocardial infarction.

* Values are number of patients (%) unless otherwise noted.

The treatment information is summarized in Table 2. The average follow-up was 8.4 months (range 0.1–21.5 months). Forty patients had available follow-up imaging. Radiographically, 26 aneurysms (65%) were completely occluded, 9 (22.5%) had a neck residual, and 5 (12.5%) had some residual dome filling at last follow-up. Thirteen patients had 12-month follow-up imaging, with 11 (84.6%) showing complete occlusion and the remaining 2 (15.4%) showing a neck remnant. The 30-day morbidity was 17.8%, including 3 ischemic strokes, 2 hemorrhagic strokes, 1 gastrointestinal bleed, 1 myocardial infarction, and 1 new-onset cranial neuropathy. There was 1 ischemic stroke with lasting morbidity, making the 30-day rate of major stroke or death 2.2%.

At last follow-up, the overall rate of cranial neuropathy improvement for all patients was 66.7% (30/45). Cranial nerve deficits resolved in 19 patients (42.2%), improved in 11 (24.4%), were unchanged in 14 (31.1%), and worsened in 1 (2.2%). The length of follow-up varied for each patient. Over time, the rate of cranial neuropathy improvement was 33.3% (15/45), 68.8% (22/32), and 81.0% (17/21) at less than 6, 6, and 12 months, respectively. At less than 6 months, 15.6% (7/45) had worsening of their cranial neuropathy. Between 6 and 12 months, the number decreased to 3.1% (1/32) and 4.8% (1/21), respectively. These data are summarized as a chart in Fig. 1. At 12 months, there were 5 patients with isolated CN II deficits and 14 patients with CN III, IV, or VI (or some combination) deficits. At last follow-up, 60% of patients with CN II deficits had improved, whereas 85.7% of those with CN III, IV, or VI deficits had improved. These results, shown over time, are summarized in Fig. 2.

Thirty-eight patients had a known duration of symptoms prior to treatment. At last follow-up, 100% (11/11) of patients who were treated within 1 month of symptom onset experienced improvement, while 44.4% (12/27) of those treated after 1 month of symptom onset experienced improvement ($p = 0.002$, Fisher's exact test). Thirty-seven patients had information on the status of their deficit (either partial or complete) on initial workup. At last follow-up, 70.4% (19/27) of those with partial deficits improved compared with 30% (3/10) of those with complete deficits ($p = 0.056$, OR 5.54 [95% CI 1.14–27.03]). In 40 patients with available follow-up imaging, we analyzed whether aneurysm occlusion resulted in improved recovery of cranial neuropathy. If the aneurysm was completely occluded, there was a trend toward better clinical outcome ($p = 0.12$, RR 1.44 [95% CI 0.84–2.48]). Forty-four patients had information on the use of coils in addition to flow diversion for treatment of their aneurysm. At last follow-up, 66.7% (24/36) versus 62.5% (5/8) of patients improved with no coils or with coils, respectively ($p = 1.0$, OR 1.2 [95% CI 0.24–5.89]). With regard to aneurysm size, if an aneurysm was smaller than 20 mm, 61.5% of patients (16/26) improved, whereas if the aneurysm was larger than 20 mm versus 73.7% (14/19) improved ($p = 0.53$, OR 0.57 [95% CI 0.16–2.08]). Patients were stratified based on age younger

FIG. 1. Cranial neuropathy outcome by follow-up time.

FIG. 2. Comparison of CN II deficit versus CN III, IV, and VI deficit improvement over time.

than 60 years. Those patients in the younger cohort improved 50% of the time (8/16), and those in the older cohort improved 75.9% of the time (22/29) ($p = 0.1$, OR 0.32 [95% CI 0.09–1.16]). These findings are summarized in Table 3.

Discussion

Our study demonstrates a comparable rate of cranial neuropathy improvement when using flow diversion compared with rates reported in the literature for both microsurgical and endovascular techniques. The bulk of available data involve CNs II and III. A literature search for series of 5 or more patients reporting on resolution of CNs II and III after surgical and endovascular intervention of cerebral aneurysm was performed. Tables 4 and 5 summarize the literature for CN II and III deficit outcomes, respectively. The improvement rates for CN II deficits range between 50% and 100% (mean 68.6%) for microsurgery and between 37.5% and 60% (mean 46.2%) for coiling. Our series found an improvement rate of 60% at 12 months for those patients with CN II neuropathy, placing flow diversion slightly behind microsurgery and ahead of coil embolization. The improvement rates for CN III deficits range between 77% and 100% (mean 93.6%) for

microsurgery and between 80% and 100% (mean 92.2%) for coil embolization. In the present series, we found an 85% improvement rate for deficits CNs III, IV, and VI at 12 months, placing flow diversion slightly behind both microsurgery and coil embolization.

As indicated in Tables 4 and 5, the rates of cranial neuropathy recovery after coiling or surgical clipping of cerebral aneurysms vary widely in published literature. There are a few retrospective studies that analyze predictors of improvement aside from method of treatment. Most of these studies deal with cranial neuropathy due to posterior communicating artery aneurysms. Factors considered are age, duration of symptoms prior to treatment, cranial neuropathy associated with subarachnoid hemorrhage, and cardiovascular risk factors (tobacco use, obesity, dyslipidemia, hypertension, and diabetes). Stiebel-Kalish et al.³¹ reported on 11 patients treated with coiling and noted that age and microvascular risk factors were observed more often in those cases with residual cranial nerve palsy. Ahn et al.¹ reported on 10 patients who were treated with coiling and 7 patients who underwent microsurgical clipping. In the coiling group, older age, presence of diabetes, complete third nerve palsy at presentation, and delayed treatment were associated with poor prognosis in recovery, whereas in the clipping group, only older age was related to decreased cranial nerve recovery. On the other hand, Kassis et al.¹⁸ reported outcomes for 20 patients, noting that early management (within 7 days of cranial nerve palsy onset) and clinical presentation with subarachnoid hemorrhage influenced recovery, but cardiovascular risk had no effect. Recently, Chalouhi et al.³ observed recovery of nearly 90% of CN III deficits after endovascular treatment in 37 patients, and partial deficit at presentation was the only statistically significant predictor of complete recovery. Due to the design of previously published studies, it is unclear whether there was an association of recovery prognosis with factors other than the method of treatment, but time to treatment and CN deficit at presentation are consistently reported.

In the present study, we tested 4 parameters in our secondary analysis: time from onset of neuropathy to treatment, presence of coils, aneurysm size, partial or complete cranial neuropathy, and age. The only factor that reached

TABLE 3. Secondary analysis

Factor	% Improved	p Value	OR (95% CI)
Treated w/in 1 mo of symptoms	100 (11/11)	0.002	NA
Treated after 1 mo of symptoms	44.4 (12/27)		
Partial deficit	70.4 (19/27)	0.056	5.54 (1.14–27.03)
Complete deficit	30 (3/10)		
No coils used	66.7 (24/36)	1.0	1.2 (0.24–5.89)
Coils used	62.5 (5/8)		
Aneurysm <20 mm	61.5 (16/26)	0.53	0.57 (0.16–2.08)
Aneurysm ≥20 mm	73.6 (14/19)		
Age <60 yrs	50 (8/16)	0.1	0.32 (0.09–1.16)
Age ≥60 yrs	75.9 (22/29)		

TABLE 4. Literature review for improvement of oculomotor nerve palsies after surgical clipping and coil embolization

Authors & Year	No. of Patients	No. Improved	%
Surgical clipping			
Khan et al., 2013	8	8	100
Park et al., 2011	13	13	100
Javalkar et al., 2010	26	20	76.9
Nam et al., 2010	8	7	87.5
Chen et al., 2006	7	7	100
Ahn et al., 2006	7	7	100
Dimopoulos et al., 2005	5	5	100
Fujiwara et al., 1989	26	22	84.6
Mean			93.6
Coil embolization			
Khan et al., 2013	9	8	88.9
Ko & Kim, 2011	10	8	80
Nam et al., 2010	6	5	83.3
Kassis et al., 2010	20	19	95
Hanse et al., 2008	21	19	90.5
Chen et al., 2006	6	6	100
Ahn et al., 2006	10	10	100
Stiebel-Kalish et al., 2003	11	11	100
Mean			92.2

TABLE 5. Literature review for improvement of ophthalmic nerve palsies after surgical clipping and coil embolization

Authors & Year	No. of Patients	No. Improved	%
Surgical clipping			
Mattingly et al. 2013	14	11	78.6
Dehdashti et al., 2012	12	9	75
Schuss et al., 2011	12	9	75
Park et al., 2009	10	8	80
de Oliveira et al., 2009	14	14	100
Nonaka et al., 2007	7	3	42.9
Hoh et al., 2001	12	8	66.7
Date et al., 1998	6	3	50
Day, 1990	23	17	73.9
Norwood et al., 1986	8	5	62.5
Ferguson & Drake, 1981	14	7	50
Mean			68.6
Coil embolization			
Drazin et al., 2013	15	8	53.3
Schuss et al., 2011	8	3	37.5
Heran et al., 2007	15	7	46.7
Malisch et al., 1998	6	2	33.3
Halbach et al., 1994	5	3	60
Mean			46.2

statistical significance was time from onset of neuropathy to treatment (< 1 month); all 11 patients had improvement in their deficit. Presence of partial versus complete neuropathy at presentation nearly reached statistical significance ($p = 0.56$, OR 5.54), which is consistent with prior literature, indicating that the degree of deficit has prognostic value regardless of treatment technique.^{1,3} Interestingly, the absence or presence of coils had almost no effect on the improvement rate ($p = 1$, OR 1.2), which supports the theory in coiling literature that the cranial neuropathy depends less on mass effect than it does on the pulsatility of the aneurysm sac.

It is important to note that in 15% of our patients, cranial neuropathy worsened in the short term after placement of a flow diverter. This number decreased to 4.8% (representing 1 patient) by 12 months. This is important to note for the counseling of patients if flow diversion is to be used.

This study is limited by its retrospective nature as well as the relatively small sample size. Data were extracted from multiple institutions and therefore depend on each institution's records. Formal neuroophthalmological testing was not available for many patients. Another confounding factor in our study is that we examined all recovery of function of all cranial nerves related to visual function (CNs II, III, IV, and VI). CNs III, IV, and VI were grouped together because of their similar function. It is possible that some of these nerves are more likely to improve than others.

The average follow-up time was 8.4 months. In a disease that clearly improves with time, it would be helpful to have longer follow-up times.

Conclusions

Cranial neuropathy caused by cerebral aneurysm responds similarly when the aneurysm is treated with PED in comparison with open surgery and coil embolization. Lower morbidity and higher occlusion rates obtained with the PED may suggest it as treatment of choice for some of these lesions. Time to treatment is an important consideration regardless of treatment modality. A prospective study with formal pre- and posttreatment ophthalmological examinations is warranted to verify these findings.

Acknowledgments

Dr. Siddiqui receives research grants from the NIH and the University at Buffalo (not related to the current work).

References

- Ahn JY, Han IB, Yoon PH, Kim SH, Kim NK, Kim S, et al: Clipping vs coiling of posterior communicating artery aneurysms with third nerve palsy. **Neurology** 66:121–123, 2006
- Becske T, Kallmes DF, Saatci I, McDougall CG, Szikora I, Lanzino G, et al: Pipeline for uncoilable or failed aneurysms: results from a multicenter clinical trial. **Radiology** 267:858–868, 2013
- Chalouhi N, Theofanis T, Jabbour P, Dumont AS, Gonzalez LF, Starke RM, et al: Endovascular treatment of posterior communicating artery aneurysms with oculomotor nerve palsy: clinical outcomes and predictors of nerve recovery. **AJNR Am J Neuroradiol** 34:828–832, 2013
- Chen PR, Amin-Hanjani S, Albuquerque FC, McDougall C, Zabramski JM, Spetzler RF: Outcome of oculomotor nerve palsy from posterior communicating artery aneurysms:

- comparison of clipping and coiling. **Neurosurgery** 58:1040–1046, 2006
5. Date I, Asari S, Ohmoto T: Cerebral aneurysms causing visual symptoms: their features and surgical outcome. **Clin Neurol Neurosurg** 100:259–267, 1998
 6. Day AL: Aneurysms of the ophthalmic segment. A clinical and anatomical analysis. **J Neurosurg** 72:677–691, 1990
 7. de Oliveira JG, Borba LA, Rassi-Neto A, de Moura SM, Sanchez-Júnior SL, Rassi MS, et al: Intracranial aneurysms presenting with mass effect over the anterior optic pathways: neurosurgical management and outcomes. **Neurosurg Focus** 26(5):E3, 2009
 8. Dehdashti AR, Le Roux A, Bacigaluppi S, Wallace MC: Long-term visual outcome and aneurysm obliteration rate for very large and giant ophthalmic segment aneurysms: assessment of surgical treatment. **Acta Neurochir (Wien)** 154:43–52, 2012
 9. Dimopoulos VG, Fountas KN, Feltes CH, Robinson JS, Grigorian AA: Literature review regarding the methodology of assessing third nerve paresis associated with non-ruptured posterior communicating artery aneurysms. **Neurosurg Rev** 28:256–260, 2005
 10. Drazin D, Choulakian A, Nuño M, Gandhi R, Edgell RC, Alexander MJ: Improvement in visual symptomatology after endovascular treatment of cavernous carotid aneurysms: a multicenter study. **J Vasc Interv Neurol** 6:15–21, 2013
 11. Ferguson GG, Drake CG: Carotid-ophthalmic aneurysms: visual abnormalities in 32 patients and the results of treatment. **Surg Neurol** 16:1–8, 1981
 12. Fujiwara S, Fujii K, Nishio S, Matsushima T, Fukui M: Oculomotor nerve palsy in patients with cerebral aneurysms. **Neurosurg Rev** 12:123–132, 1989
 13. Halbach VV, Higashida RT, Dowd CF, Barnwell SL, Fraser KW, Smith TP, et al: The efficacy of endosaccular aneurysm occlusion in alleviating neurological deficits produced by mass effect. **J Neurosurg** 80:659–666, 1994
 14. Hanse MC, Gerrits MC, van Rooij WJ, Houben MP, Nijssen PC, Sluzewski M: Recovery of posterior communicating artery aneurysm-induced oculomotor palsy after coiling. **AJNR Am J Neuroradiol** 29:988–990, 2008
 15. Heran NS, Song JK, Kupersmith MJ, Niimi Y, Namba K, Langer DJ, et al: Large ophthalmic segment aneurysms with anterior optic pathway compression: assessment of anatomical and visual outcomes after endosaccular coil therapy. **J Neurosurg** 106:968–975, 2007
 16. Hoh BL, Carter BS, Budzik RF, Putman CM, Ogilvy CS: Results after surgical and endovascular treatment of paraclinoid aneurysms by a combined neurovascular team. **Neurosurgery** 48:78–90, 2001
 17. Javalkar V, Cardenas R, Nanda A: Recovery of third nerve palsy following surgical clipping of posterior communicating artery aneurysms. **World Neurosurg** 73:353–356, 2010
 18. Kassir SZ, Jouanneau E, Tahon FB, Salkine F, Perrin G, Turjman F: Recovery of third nerve palsy after endovascular treatment of posterior communicating artery aneurysms. **World Neurosurg** 73:11–16, e2, 2010
 19. Khan SA, Agrawal A, Hailey CE, Smith TP, Gokhale S, Alexander MJ, et al: Effect of surgical clipping versus endovascular coiling on recovery from oculomotor nerve palsy in patients with posterior communicating artery aneurysms: A retrospective comparative study and meta-analysis. **Asian J Neurosurg** 8:117–124, 2013
 20. Ko JH, Kim YJ: Oculomotor nerve palsy caused by posterior communicating artery aneurysm: evaluation of symptoms after endovascular treatment. **Interv Neuroradiol** 17:415–419, 2011
 21. Malisch TW, Guglielmi G, Viñuela F, Duckwiler G, Gobin YP, Martin NA, et al: Unruptured aneurysms presenting with mass effect symptoms: response to endosaccular treatment with Guglielmi detachable coils. Part I. Symptoms of cranial nerve dysfunction. **J Neurosurg** 89:956–961, 1998
 22. Mattingly T, Kole MK, Nicolle D, Boulton M, Pelz D, Lownie SP: Visual outcomes for surgical treatment of large and giant carotid ophthalmic segment aneurysms: a case series utilizing retrograde suction decompression (the “Dallas technique”). **J Neurosurg** 118:937–946, 2013
 23. Nam KH, Choi CH, Lee JJ, Ko JG, Lee TH, Lee SW: Unruptured intracranial aneurysms with oculomotor nerve palsy: clinical outcome between surgical clipping and coil embolization. **J Korean Neurosurg Soc** 48:109–114, 2010
 24. Nonaka T, Haraguchi K, Baba T, Koyanagi I, Houkin K: Clinical manifestations and surgical results for paraclinoid cerebral aneurysms presenting with visual symptoms. **Surg Neurol** 67:612–619, 2007
 25. Norwood EG, Kline LB, Chandra-Sekar B, Harsh GR III: Aneurysmal compression of the anterior visual pathways. **Neurology** 36:1035–1041, 1986
 26. Park J, Kang DH, Chun BY: Superciliary keyhole surgery for unruptured posterior communicating artery aneurysms with oculomotor nerve palsy: maximizing symptomatic resolution and minimizing surgical invasiveness. **J Neurosurg** 115:700–706, 2011
 27. Park JH, Park SK, Kim TH, Shin JJ, Shin HS, Hwang YS: Anterior communicating artery aneurysm related to visual symptoms. **J Korean Neurosurg Soc** 46:232–238, 2009
 28. Raymond J, Guilbert F, Weill A, Georganos SA, Juravsky L, Lambert A, et al: Long-term angiographic recurrences after selective endovascular treatment of aneurysms with detachable coils. **Stroke** 34:1398–1403, 2003
 29. Schuss P, Güresir E, Berkefeld J, Seifert V, Vatter H: Influence of surgical or endovascular treatment on visual symptoms caused by intracranial aneurysms: single-center series and systematic review. **J Neurosurg** 115:694–699, 2011
 30. Stiebel-Kalish H, Kalish Y, Bar-On RH, Setton A, Niimi Y, Berenstein A, et al: Presentation, natural history, and management of carotid cavernous aneurysms. **Neurosurgery** 57:850–857, 2005
 31. Stiebel-Kalish H, Maimon S, Amsalem J, Erlich R, Kalish Y, Rappaport HZ: Evolution of oculomotor nerve paresis after endovascular coiling of posterior communicating artery aneurysms: a neuro-ophthalmological perspective. **Neurosurgery** 53:1268–1274, 2003

Disclosure

The authors report the following: Dr. Lopes: consultant for Covidien. Dr. Rawk: stock ownership in Blockade Medical. Dr. Ringer: consultant for Covidien, MicroVention, and Stryker. Dr. Arthur: consultant for Covidien, Codman, Stryker, MicroVention, Penumbra, and Sequent. Dr. Hoyt: consultant for Covidien. Dr. Snyder: support of non-study-related clinical or research effort from Boston Scientific, Cordis, Endtex, Medtronic, Abbott Vascular, ev3, Toshiba, Micrus, Zimmer, EPI, Guidant, Kerberos and Primus; stock ownership in Boston Scientific, Access Closure, and Niagara Gore Medical; financial interest in Cordis, Endotex, Micrus, and EPI; and consultant for Medtronic, Abbott Vascular, ev3, Toshiba, Micrus, and Zimmer; and Primus. Dr. Siddiqui: stock ownership in Hotspur, Intratech Medical, StimSox, Valor Medical, Blockade Medical, Lazarus Effect, Pulsar Vascular, and Medina Medical; consultant for Codman & Shurtleff, Inc., Covidien Vascular Therapies, Guidepoint Global Consulting, Penumbra Inc., Stryker, Pulsar Vascular, MicroVention, Lazarus Effect, Blockade Medical, and Reverse Medical; speakers' bureau for Codman & Shurtleff; advisory board of Codman & Shurtleff, Covidien Neurovascular, ICAVL, and Medina Medical; honoraria from Abbott Vascular and Codman & Shurtleff. Dr. Levy:

ownership in Intratech Medical Ltd. and Blockade Medical LLC; National PI for SWIFT Prime Trials for Covidien and Carotid Training Sessions for Physicians for Abbott; and legal opinion as an expert witness for Renders Medical. Dr. Hopkins: consultant for Boston Scientific, Cordis, Abbott Vascular, Covidien; direct stock ownership in Boston Scientific, Valor Medical, Claret Medica, Inc., Augmenix, Endomation, Silk Road, Ostial, Apama, StimSox, Photolitec, ValnTx, Ellipse, Axria, NextPlain, and MedinaMed; research grant from Toshiba; speakers' bureau for Abbott Vascular and Toshiba; honoraria from Cordis, Memorial HealthCare System, Complete Conf. Management, and Covidien; and board position for Claret Medical. Dr. Mocco: Consultant for Lazarus effect, Reverse, Pulsar, Edge Therapeutics, and Medina; and investor in Blockade Medical and Medina; and advisory board for Codman Neurovascular. Dr. Hanel: consultant for Covidien, Stryker, and Codman; stock ownership in Blockade; and scientific advisory board for Medina.

Author Contributions

Conception and design: Brown, Hanel. Acquisition of data: Brown, Lopes, Miller, Tawk, Brasiliense, Ringer, Sauvageau, Powers, Arthur, Hoit, Snyder, Siddiqui, Levy, Hopkins, Cuellar, Rodriguez-Mercado, Veznedaroglu, Binning, Mocco, Boulos, Yamamoto, Hanel. Analysis and interpretation of data: Brown, Aguilar-Salinas, Hanel. Drafting the article: Brown. Critically revising the article: Brown, Hanel. Reviewed submitted version of manuscript: Brown, Brasiliense, Hanel. Approved the final version of the manuscript on behalf of all authors: Brown. Statistical analysis: Brown, Aguilar-Salinas. Administrative/technical/material support: Brasiliense. Study supervision: Hanel.

Correspondence

Benjamin Brown, Department of Neurosurgery, Mayo Clinic, 4500 San Pablo Rd., Jacksonville, FL 32224. email: brown.benjamin1@mayo.edu.