Intracranial Arterial Compression of the Anterior Visual Pathway

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ABSTRACT

Compression of anterior visual pathway (AVP) structures by intracranial arteries is observed not infrequently on neuroimaging. Whether or not such compression results in damage to these structures, however, remains unclear. This information is important to define as AVP compression by intracranial arteries may be a causative factor in patients with otherwise unexplained visual dysfunction. In a single centre, 37 patients with evidence of intracranial artery AVP compression demonstrated on magnetic resonance imaging were identified by retrospective review of case records over the period 2011–2017. Variables were collected, including patient demographics, visual acuity, visual fields, pupillary reactions and optic disc appearance for patients in the case series. Visual field deficits correlated with compression sites in the 37 patients examined. Internal carotid artery-optic nerve compression was the most frequent (unilateral compression $n = 9$, bilateral compression $n = 14$), followed by chiasmal compression by the anterior cerebral artery ($n = 8$) and a combination of optic nerve and chiasmal compression ($n = 5$). Visual acuity and visual fields were stable on follow-up (mean 4 years) in 24 of 26 cases (93%). We conclude that AVP compression by intracranial arteries may be a causative factor in unexplained visual dysfunction. The visual defects are largely non-progressive.

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Introduction

Cranial nerve dysfunction due to compression occurs most commonly secondary to lesions such as intracranial tumours or aneurysms.^{1,2} However, it is also recognized that intracranial arteries may similarly compress adjacent cranial nerves, leading to nerve dysfunction.^{3,4} Pressure on the 5th, 7th, 9th, and 11th cranial nerves by adjacent arteries has been reported, respectively, in association with trigeminal neuralgia, hemifacial spasm, glossopharyngeal neuralgia, and spasmodic torticollis.^{4–14} However, the occurrence of dysfunction of the anterior visual pathway (AVP) due to vascular compression remains controversial.^{15–17} While compression of the optic nerves, chiasm, or optic tracts by intracranial arteries has been previously observed in radiological studies, there is a paucity of studies examining in detail whether or not this compression may result in visual dysfunction.^{15,17–22}

The present study aimed primarily to correlate clinical and radiological features in a cohort of

patients with vascular compression of the AVP in order to determine if this entity could be a cause of unexplained visual dysfunction in these patients. A secondary aim was to examine the natural history of visual deficits in patients with vascular compression of the AVP.

Methods

This study was approved by the University of New South Wales Human Research Ethics Committee (approval number HC16250) and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all patients included.

Patients for the retrospective case series were identified through consecutive retrospective file review of all patients who had undergone magnetic resonance imaging (MRI) of the brain in a single-centre suburban Ophthalmology practice over the period January 2011–January 2017. Patients were

included in the study if they had vascular compression of the AVP demonstrated on MRI of the brain, in combination with visual field defects, but without another defined cause.

The patients had initially been referred with visual dysfunction to the ophthalmology practice. The reasons for referral included visual loss, visual field defects, or optic nerve changes. The patients had MRI of the brain requested to investigate a Neuro-ophthalmic cause for their visual loss as they had features atypical of glaucoma or another ophthalmological condition.

Features which were not typical of glaucoma included visual field defects or optic disc appearances uncharacteristic of glaucoma, the presence of a relative afferent pupillary defect (RAPD) and poor anatomical correlation between visual field defects and retinal nerve fibre layer (RNFL) analysis. Specifically, the pattern of the visual field defect and focal neuroretinal rim loss on direct visualization of the optic nerve needed to correspond in order to be consistent with a diagnosis of glaucomatous optic neuropathy. In borderline cases, spectral-domain optical coherence tomography (OCT) imaging (Cirrus HD-OCT, Carl Zeiss Meditec Inc., Jena, Germany) with the latest commercially available software was used to define these focal RNFL deficits (signal strength was ≥ 7 in all images utilized).

Patient data recorded at presentation and last-recorded follow-up included age, gender, past medical and surgical histories, treatments for glaucoma, and neuro-ophthalmological examination findings. The neuro-ophthalmological examination included best-corrected distance visual acuity (CDVA), visual fields as determined by static automated perimetry (SAP) with the Humphrey visual field analyser (24-2 or 30-2), the presence of a RAPD, the optic disc appearance, and intraocular pressures (IOPs) as measured by Goldmann Applanation Tonometry.

T2-weighted MRI sequences (3.0 T) in the coronal and axial planes were assessed by two assessors: a neurosurgeon (M.Y.S.K.) and a neuroradiologist (A.N.). The assessors were masked to the patients' clinical details. 'Compression' of the AVP was

designated by assessors only if there was visible contact and deformation of the AVP by adjacent arteries.

Results

Thirty-seven patients with visual dysfunction and MRI of the brain demonstrating neurovascular compression were identified for inclusion in the study. Patients did not have histories or examination findings consistent with conditions which could result in vision loss such as retinal pathology, optic disc anomalies such as in optic disc hypoplasia or high myopia, features suggestive of inflammatory or ischaemic optic neuropathy, or previous stroke. While some patients had tilted discs, the combination of visual fields and OCT findings did not suggest this to be a cause of their visual field deficits. MRI of the brain (with gadolinium contrast wherever possible) confirmed that no patients in this study had any other intracranial pathology affecting the AVP that could have accounted for the observed visual field defects, such as cerebral infarction, intracranial tumours, or aneurysms.

Table 1 documents the characteristics of the 19 males and 18 females in the cohort, ranging in age from 37 to 91 years. CDVA was largely preserved (median 6/5, range 6/4 to 6/18). Thirteen patients (35%) had systemic hypertension and three patients (9%) had Type 2 diabetes. Nineteen patients (51%) were receiving medical treatment for glaucoma. Twenty-four of the 26 cases (92%) with follow-up data available demonstrated no progressive loss of visual acuity or visual fields. The mean follow-up interval was 4 years.

The AVP compression site and field defects for each patient are presented in Table 2. No patients with bilateral optic nerve compression had monocular visual defects. Of the seven patients with a positive RAPD, six demonstrated ipsilateral compression of AVP structures and the remaining patient demonstrated bilateral compression, potentially reflecting asymmetric optic nerve compression.

A summary of AVP compression sites and involved arteries is presented in Table 3. Internal carotid artery (IAC)-optic nerve compression was the most frequent, followed by chiasmal

Table 1. Characteristics of study group.

No.	Gender/age	CDVA	Systemic health disorders	Intraocular pressure (mmHg, R/L)	Glaucoma treatment	Progression (years at follow-up)
1.	M/80	6/5 OU	Hypertension, diabetes, renal cancer	10/14	Yes	None (2)
2.	M/71	6/4 OU	Hypertension	12/16	Yes	None (5)
3.	F/77	6/6 OD, 6/5 OS	–	15/17	Yes	None (3)
4.	M/62	6/4 OU	–	18/19	Yes	N/A
5.	M/56	6/9 OU	Uveitis	16/16	No	None (4)
6.	M/84	6/4 OD, 6/9 OS	Hypertension, diabetes	13/11	Yes	L VA, VF stable (6)
7.	F/69	6/6 OD, 6/5 OS	–	17/14	No	N/A
8.	M/74	6/5 OD, 6/4 OS	–	14/13	Yes	None (4)
9.	F/83	6/18 OU	Chronic myeloid leukaemia	14/13	Yes	R VA, VF stable (3)
10.	F/69	6/4 OU	Hypertension	18/16	No	None (2)
11.	M/68	6/6 OD, 6/9 OS	–	15/14	No	None (2)
12.	M/37	6/4 OU	Lupus	12/9	No	None (3)
13.	F/73	6/4 OU	Hemifacial spasm	18/12	Yes	None (1)
14.	M/91	6/4 OU	Diabetes	20/18	Yes	None (6)
15.	M/62	6/6 OU	–	25/25	No	N/A
16.	F/71	6/4 OU	–	16/17	Yes	None (6)
17.	F/59	6/4 OU	–	21/21	No	N/A
18.	F/66	6/4 OD, 6/5 OS	–	13/17	Yes	None (2)
19.	M/76	6/5 OD, 6/4 OS	Hypertension	10/11	Yes	None (2)
20.	M/75	6/4 OU	Hypertension	18/18	No	None (1)
21.	M/72	6/9 OU	Hypertension	21/16	Yes	N/A
22.	M/63	6/5 OD, 6/6 OS	Hypertension	16/19	No	None (2)
23.	F/85	6/5 OD, 6/6 OS	Hypertension	13/21	Yes	None (0)
24.	M/63	6/4 OU	–	15/14	No	
25.	M/51	6/4 OU	–	10/12	Yes	None (10)
26.	F/70	6/6 OD, 6/4 OS	Hypertension	14/12	No	N/A
27.	F/78	6/4 OD, 6/6 OS	Hypertension	25/16	Yes	N/A
28.	F/85	6/6 OD, 6/9 OS	–	11/12	Yes	N/A
29.	F/73	6/4 OU	–	10/10	Yes	None (2)
30.	M/71	6/9 OD, 6/4 OS	–	14/13	No	None (2)
31.	F/82	6/4 OD, 6/5 OS	Hypertension	10/12	No	N/A
32.	M/75	6/4 OU	–	16/16	Yes	None (2)
33.	M/68	6/6 OU	Hypertension	11/15	No	None (2)
34.	F/88	6/5 OD, 6/6 OS	High cholesterol	14/14	No	None (8)
35.	F/73	6/4 OU	–	10/12	No	N/A
36.	F/74	6/5 OU	–	13/12	No	None (2)
37.	F/74	6/6 OU	–	17/18	No	N/A

Notes. OU: both eyes; OD: right eye; OS: left eye; RAPD: relative afferent pupil defect; VF: visual field.

compression and a combination of optic nerve and chiasmal compression.

Selected case reports

Patient 28: An 85-year-old woman was found to have a bilateral superior visual field defects on her Humphrey SAP (Figure 1). She had a CDVA of 6/6 OD and 6/9 OS, cup-to-disc ratios of 0.6 and 0.5, and IOPs of 11 and 12 mmHg in the right eye and in the left eye, respectively. There was superior and inferior thinning bilaterally on RNFL imaging. The visual field defect and OCT findings corresponded with imaging demonstrating compression of both optic nerves inferiorly by the supraclinoid ICAs with deformation of the nerves and elevation such that

the superior surfaces of the optic nerves also abutted the inferior frontal lobes.

Patient 5: A 56-year-old man was found to have CDVA of 6/5 OU and a cup-to-disc ratio of 0.5 bilaterally. A binasal field defect was found on SAP (inferiorly > superiorly) (Figure 2). This correlated with the bilateral compression of the superolateral surfaces of the chiasm by the ACAs. Bilateral superior and inferior RNFL thinning on OCT imaging was further observed. Visual acuity, field defects, and optic disc cupping were non-progressive over 4 years.

Patient 3: A 77-year-old woman was found to have predominantly a right inferonasal visual field defect on her Humphrey SAP (Figure 3). Her CDVA was 6/5 OU, her IOPs were 15 and

Table 2. Visual field defects and corresponding compression sites.

No.	Field defect	Compression site	Vessel	Cup/disc ratio (R/L)	RAPD (graded as 0/4 absent – 4/4)
1.	R inferior	R temporal optic tract from above	P Comm	0.5/0.4	R 2/4
2.	L inferior				
3.	L superonasal	L temporal optic nerve from below	L ICA	0.8/0.7	Absent
4.	R superior	Bilateral optic nerves	Bilateral ICA	0.6/0.5	Absent
5.	L superior				
6.	R superior	R and L optic nerves from below	Bilateral ICA	0.8/0.8	Absent
7.	L superior				
8.	R & L inferonasal	Bilateral optic chiasm from above	Bilateral ACA	0.7/0.6	Absent
9.	L & R inferotemporal	Central optic chiasm from above	R ACA	0.8/0.7	Absent
10.	Inferior bitemporal quadrantanopia	Central optic chiasm from above (L optic nerve near optic chiasm from below)	L ACA	0.1/0.0	Absent
11.	R superonasal	Bilateral optic nerves from below	Bilateral ICA	0.8/0.8	R 2/4
12.	L inferotemporal	R optic chiasm	R ACA		
13.	L temporal	L ACA compressing central chiasm	L ACA	0.9/0.8	Absent
14.	R inferonasal	Bilateral optic nerves from below	Bilateral ICA	0.6/0.7	Absent
15.	L inferior				
16.	R temporal	R optic nerve on lateral side	R ICA	0.9/0.6	R 2/4
17.	R inferotemporal	Central optic chiasm from above	R ACA	0.4/0.3	Absent
18.	R superior	Bilateral optic nerves from below	Bilateral ICA	0.3/0.3	Absent
19.	L inferior				
20.	R & L superior	Bilateral optic nerves from below	Bilateral ICA	0.6/0.3	Absent
21.	L superotemporal	L medial optic nerve	L ACA	0.3/0.2	Absent
22.	L inferior and R nasal	R medial optic nerve	R ACA	0.7/0.9	Absent
23.		Central chiasm from above	L ACA		
24.	L superonasal	L optic nerve	L ICA	0.6/0.7	Absent
25.	R & L inferior arcuate scotomas	Bilateral optic nerves from below	Bilateral ICA	0.8/0.8	Absent
26.			R ACA		
27.	Bitemporal field	Central optic chiasm from above	L ACA	0.8/0.7	Absent
28.	R superotemporal & L superonasal	Bilateral lateral optic nerves from below	Bilateral ICA	0.3/0.4	Absent
29.	R & L superonasal	Bilateral optic nerves from below	Bilateral ICA	0.4/0.2	Absent
30.	R & L superotemporal	Bilateral optic nerves from below (R optic nerve from medial)	Bilateral ICA	0.3/0.4	Absent
31.			R ACA		
32.	R superior	Central chiasm from above	Bilateral ACA	0.6/0.5	Absent
33.	R & L inferior	Bilateral optic nerve from above and below	Bilateral ICA	0.2/0.2	Absent
34.			Bilateral ACA		
35.	R inferior	R optic nerve from below	R Ophthalmic artery	0.9/0.7	R 2/4
36.		R chiasm from above	R ACA		
37.	R & L superior	Central chiasm from above	R ACA	0.1/0.1	Absent
38.	L nasal crossing midline & R superonasal	Bilateral optic nerves	Bilateral ICA	0.8/0.9	Absent
39.	R inferonasal	R temporal optic nerve	R ICA terminus	0.7/0.6	R 3/4
40.	Binasal, L > R	Bilateral optic nerves from temporal sides, L > R	Bilateral ICA	0.4/0.5	Absent
41.	R inferior	R optic nerve from below	R ICA	0.6/0.5	Absent
42.	R inferior	R optic nerve from below	R ICA	0.3/0.3	R 3/4
43.	L inferonasal	Bilateral optic nerves, L worse than R	Bilateral ICA	0.6/0.7	Absent
44.	R nasal				
45.	R superior	R optic nerve and chiasm from temporal side	R ICA, ACA and recurrent artery of Heubner	0.5/0.8	Absent
46.	R inferotemporal	R optic nerve from below	R ICA	0.3/0.5	Absent
47.	L inferonasal	L optic nerve from underside	L ICA	0.3/0.7	L 2/4
48.	L & R inferior	Bilateral lateral optic nerves	L ICA	0.3/0.3	Absent
49.			R ICA		
50.	R inferonasal	Superior aspect of chiasm	R ACA	0.2/0.3	Absent

Notes. ICA: internal carotid artery; ACA: anterior cerebral artery; R: right; L: left; P Comm: posterior communicating.

17 mmHg and her cup-to-disc ratios were 0.7 and 0.6 in her right eye and left eye, respectively. She had a right RAPD, associated with impaired

colour saturation on the right. A right superior and inferior nerve fibre layer thinning was seen on OCT imaging as well as inferior RNFL

Table 3. Compression of AVP structures and involved arteries.

Unilateral optic nerve	
ICA	<i>n</i> = 9
Bilateral optic nerves	
ICA	<i>n</i> = 11
ICA + ACA	<i>n</i> = 3
Chiasm	
ACA	<i>n</i> = 8
Optic nerves + chiasm	
Combination of ICA and/or ACA ^a	<i>n</i> = 5
Unilateral optic tract	
Posterior communicating artery	<i>n</i> = 1

Notes. ICA: internal carotid artery; ACA: anterior cerebral artery; AVP: anterior visual pathway.

^aNote the ophthalmic artery and recurrent artery of Heubner were involved in one case each.

thinning in the left eye. An MRI of the brain demonstrated compression of the right optic nerve by the supraclinoid portion of the ICA.

Discussion

The role of vascular compression of the AVP in causing visual defects has thus far been unclear.^{23–25} The present study described a cohort of 37 patients with vascular compression of the AVP and correlated patients' radiological and clinical features. The study supports the notion that AVP-artery compression may explain observed visual field defects and optic nerve head changes in some patients without a hitherto-defined cause of their visual dysfunction. It further suggests that visual dysfunction observed in patients with AVP-artery compression is only slowly progressive.

While it is known that mass lesions are an important consideration in the evaluation and workup of unexplained optic neuropathies,²³ relatively few studies have examined the incidence of AVP-artery compression in patients with visual dysfunction.^{24,25} Jacobson et al. had described a series of 18 patients with symptomatic optic nerve compression by the ICA. However, a third of the patients in the study did not have visual field testing and the patients were not followed up.¹⁵ Purvin et al. more recently also described a series of 10 patients with compression of the AVP by vessels.¹⁷ The present study represents the largest cohort of patients with potential vascular compression of the AVP thus far. Additionally, it provides more detailed clinical and radiological correlations in comparison with previous studies, as well as longitudinal data.

While compression of the optic nerve by the ICA has been observed more commonly in radiological studies,^{15,18–22,26–29} the phenomenon of chiasmal compression by normal intracranial arteries has been described previously only in isolated case reports.³⁰ In the present study, however, 13 patients had compression of the optic chiasm by the ACAs. In one patient, the optic tract was compressed by the posterior communicating artery, and the optic nerve by the ophthalmic artery in another patient. Both cases had visual field defects consistent with the pattern of compression, adding to the body of recent case reports describing AVP compression involving the posterior circulation and ophthalmic arteries.^{31,32} Hence, the current study indicates that compression of structures throughout the AVP may contribute to visual field defects and supplements the existing literature by outlining an extended range of potential nerve–artery interactions.

The phenomenon of AVP compression can potentially be confused with normal pressure glaucoma (NPG), as IOP may not be raised and visual field defects can be paracentral even upon presentation or in early disease. Therefore, further investigations including imaging should be considered for patients with features atypical of NPG, as is accepted practice to exclude conditions that can masquerade as glaucomatous optic neuropathy.³³ This includes careful history with special attention given to neuro-ophthalmological presentations. This includes signs such as impaired visual acuity, loss of colour vision, loss of central vision, pallor of the neuroretinal rim and the presence of a RAPD, which are generally considered classic of compressive rather than glaucomatous damage.

The patients in the current and previous series, however, did not, in general, exhibit these features,¹⁵ implying AVP compression may present with clinical features atypical of compressive injury. It has recently been proposed that RNFL thinning of nasal and temporal quadrants of the optic nerve may suggest the presence of a compressive aetiology rather than glaucomatous damage.³⁴ Indeed, nerve fibre analysis loss, such as demonstrated by OCT, especially with the newer modality of macular ganglion cell within the inner plexiform layer (GCIP) analysis, may prove to be a useful new adjunct in distinguishing possible compressive damage of the AVP by intracranial arteries from glaucomatous

Figure 1. T2-weighted MRI in coronal plane demonstrating compression of the right and left optic nerves inferiorly by the supraclinoid ICAs. Bilateral superior field defects on Humphrey 24-2 SAP. Bilateral inferior and superior retinal nerve fibre layer thinning on OCT.

damage.³⁵ In chiasmal compression, for example, binasal hemiretinal thinning may be appreciated on macular GCIP, while glaucoma would tend to produce thinning of the macular GCIP which respects the horizontal meridian.³⁶ Macular GCIP thinning may further precede peripapillary RNFL loss and perimetric changes.³⁶

The mechanism underlying nerve damage resulting from artery–nerve contact remains controversial. Damage has been proposed to result from either the direct impact of vessel pulsatility on nerve fibres, or from ischaemic injury, since the local blood supply of nerve fibres is compromised due to compression.²⁰ It

is further possible that factors other than nerve impingement may predispose to visual field defects in cases of AVP compression. These include circulatory disorders such as hypertension and diabetes, which were present in the 37 patients examined in the current study (35% and 9%, respectively).

Defining the role of AVP compression in producing visual field defects may well prove to be worthwhile, as options such as microvascular decompression have been suggested to improve symptoms and signs in a selected cohort of patients.^{18,26–28} Nevertheless, visual loss in AVP compression was shown to be only slowly

Figure 2. T2-weighted MRI in coronal planes demonstrates compression of the optic chiasm superolaterally by both ACAs. Binasal defect on Humphrey 24-2 SAP. Bilateral superior and inferior retinal nerve fibre layer thinning on OCT.

progressive in the current study, as well as in the review by Purvin et al. of 10 patients¹⁷ This suggests that conservative management with detailed follow-up with repeat visual field evaluation might be suitable for a subcohort of the patients, especially the older ones. Furthermore, optic nerve-ICA and optic chiasm-ACA deformation has also

been described as a relatively frequent radiological finding. A recent study by Tsutsumi et al. of 183 patients suggested an 11% incidence while the previous study by Jacobson et al. of 100 patients suggested a 17% incidence.^{37,38} This would suggest other aetiologies of visual dysfunction must be excluded before interventions to alleviate vascular

Figure 3. T2-weighted MRI in coronal planes demonstrates compression of the right optic nerve by the right supraclinoid ICA. Right lower nasal defect on Humphrey 24-2 SAP. Right inferior and left superior and inferior retinal nerve fibre layer thinning on OCT.

compression are considered. However, patients in the Tsutumi et al. radiological study did not undergo full visual testing, so it is possible that patients had unidentified visual dysfunction.³⁷ This feature also highlights that there is an incomplete understanding of the exact mechanism of loss of function in artery–nerve compression and interaction. As yet unidentified factors may be involved in leading AVP compression to manifest with symptomatic visual dysfunction.

This study includes the largest cohort to date of patients with potential AVP compression examined utilizing the high-resolution MRI methodology now available. In contrast to previous MRI studies, which

may not have allowed complete clarity as to the possibility of direct nerve compression,^{15,19,39} there was clear MRI evidence of compression in all patients in this study. Limitations of the present study include its retrospective design and inherent limitations of being a case series in which is represented a selected group of patients. Colour vision was also not routinely tested in the patients. As previously mentioned, such vascular compression of AVP structures may occur with a relatively high frequency in asymptomatic individuals,^{37,38} rendering its role in causing unexplained visual field defects still difficult to define. Our group plans to undertake a study with 250 prospective consecutive MRIs from otherwise normal

patients in order to establish the frequency of contact or compression of the AVP by any of the four arteries cited and assess for any ophthalmological findings. It is difficult to eliminate glaucoma completely as a contributor to visual dysfunction in this cohort of patients and indeed it is plausible that NPG could coexist with AVP-compression phenomena. Glaucoma therapy was continued in any patient in whom this remained a possibility. Furthermore, although T2 signal changes within the AVP in the setting of compression have previously been suggested to correlate with the degree of observed visual dysfunction,^{40,41} T2 signal changes were not observed on imaging in our study.

Despite these limitations, as the largest cohort of patients described hitherto with symptomatic AVP-artery compression, this report provides evidence of a correlation between vascular compression and visual field deficits. Recognizing neurovascular compression when present is beneficial, as it may provide an explanation for otherwise unexplained visual deficits to patient and clinicians and may potentially help to prevent unnecessary further investigations.

Conclusion

While compression of cranial nerves by intracranial arteries is a well-accepted phenomenon and incidental finding, the occurrence of damage to the AVP is potentially under-recognized. The present study supports the hypothesis that vascular compression of the AVP may be a causative factor in some cases of unexplained visual dysfunction. This has potential clinical ramifications in providing reassurance to patients and clinicians. Indeed, if visual deficits correspond with the pattern of neurovascular compression, these deficits may well be non-progressive. This phenomenon may be confused with glaucomatous optic neuropathy, suggesting that investigation of possible AVP vascular compression with MRI should be considered in patients who have features atypical of classical glaucomatous disease, including defined neuro-ophthalmological signs.

Declaration of interest

The authors report no conflicts of interest.

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References

1. Rush JA, Younge BR. Paralysis of cranial nerves III, IV, and VI: cause and prognosis in 1,000 cases. *Arch Ophthalmol.* 1981;99:76–79.
2. Hyland H, Barnett H. The pathogenesis of cranial nerve palsies associated with intracranial aneurysms. *Proc R Soc Med.* 1954;47:141.
3. Jannetta PJ, McLaughlin MR, Casey KF. Technique of microvascular decompression. Technical note. *Neurosurg Focus.* 2005;18:E5.
4. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. *J Neurosurg.* 1999;90(1):1–8. doi:10.3171/jns.1999.90.1.0001.
5. Fukuda H, Ishikawa M, Okumura R. Demonstration of neurovascular compression in trigeminal neuralgia and hemifacial spasm with magnetic resonance imaging: comparison with surgical findings in 60 consecutive cases. *Surg Neurol.* 2003;59:93–99.
6. Meaney JF, Eldridge PR, Dunn LT, Nixon TE, Whitehouse GH, Miles JB. Demonstration of neurovascular compression in trigeminal neuralgia with magnetic resonance imaging: comparison with surgical findings in 52 consecutive operative cases. *J Neurosurg.* 1995;83(5):799–805. doi:10.3171/jns.1995.83.5.0799.
7. Nagahiro S, Takada A, Matsukado Y, Ushio Y. Microvascular decompression for hemifacial spasm: patterns of vascular compression in unsuccessfully operated patients. *J Neurosurg.* 1991;75(3):388–392. doi:10.3171/jns.1991.75.3.0388.
8. Ohtsuka K, Sone A, Igarashi Y, Akiba H, Sakata M. Vascular compressive abducens nerve palsy disclosed by magnetic resonance imaging. *Am J Ophthalmol.* 1996;122(3):416–419. doi:10.1016/S0002-9394(14)72068-9.
9. Jho H, Jannetta PJ. Microvascular decompression for spasmodic torticollis. *Acta Neurochir.* 1995;134:21–26.
10. Barker FG 2nd, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD. Microvascular decompression for hemifacial spasm. *J Neurosurg.* 1995;82(2):201–210. doi:10.3171/jns.1995.82.2.0201.
11. Resnick DK, Jannetta PJ, Bissonette D, Jho HD, Lanzino G. Microvascular decompression for glossopharyngeal neuralgia. *Neurosurgery.* 1995;36:64–68.
12. Sekula RF, Marchan EM, Fletcher LH, Casey KF, Jannetta PJ. Microvascular decompression for trigeminal neuralgia in elderly patients. *J Neurosurg.* 2008;108(4):689–691. doi:10.3171/JNS/2008/108/4/0689.
13. Barker FG, Jannetta PJ, Bissonette DJ, Larkins MV, Jho HD. The long-term outcome of microvascular decompression for trigeminal neuralgia. *N Engl*

- J Med.* 1996;334(17):1077–1084. doi:10.1056/NEJM199604253341701.
14. Gardner WJ. Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. *J Neurosurg.* 1962;19:947–958. doi:10.3171/jns.1962.19.11.0947.
 15. Jacobson DM. Symptomatic compression of the optic nerve by the carotid artery: clinical profile of 18 patients with 24 affected eyes identified by magnetic resonance imaging. *Ophthalmology.* 1999;106(10):1994–2004. doi:10.1016/S0161-6420(99)90414-1.
 16. Spetzler RF, Koos WT, Richling B, Lang J. *Color Atlas of Microneurosurgery: Microanatomy, Approaches, Techniques.* 2nd ed. Stuttgart: Thieme; 1997.
 17. Purvin V, Kawasaki A, Zeldes S. Dolichoectatic arterial compression of the anterior visual pathways: neuro-ophthalmic features and clinical course. *J Neurol Neurosurg Psychiatry.* 2004;75:27–32.
 18. Colapinto EV, Cabeen MA, Johnson LN. Optic nerve compression by a dolichoectatic internal carotid artery: case report. *Neurosurgery.* 1996;39:604–606.
 19. Golnik KC, Hund III PW, Stroman GA, Stewart WC. Magnetic resonance imaging in patients with unexplained optic neuropathy. *Ophthalmology.* 1996;103:515–520.
 20. Gutman I, Melamed S, Ashkenazi I, Blumenthal M. Optic nerve compression by carotid arteries in low-tension glaucoma. *Graefes Arch Clin Exp Ophthalmol.* 1993;231:711–717.
 21. Ogata N, Imaizumi M, Kurokawa H, Arichi M, Matsumura M. Optic nerve compression by normal carotid artery in patients with normal tension glaucoma. *Br J Ophthalmol.* 2005;89(2):174–179. doi:10.1136/bjo.2004.047035.
 22. Umihara J, Oguchi K, Kurimoto Y, Matsuno K, Fukasaku K, Yoshimura N. Optic nerve compression by the internal carotid artery in patients with normal tension and high tension glaucoma. *Br J Ophthalmol.* 1999;83(11):1306. doi:10.1136/bjo.83.11.1306b.
 23. Ahmed II, Feldman F, Kucharczyk W, Trope GE. Neuroradiologic screening in normal-pressure glaucoma: study results and literature review. *J Glaucoma.* 2002;11:279–286.
 24. Bergaust B. Unusual course of internal carotid artery accompanied by bitemporal hemianopia. *Acta Ophthalmol.* 1963;41(3):270–274. doi:10.1111/j.1755-3768.1963.tb02437.x.
 25. Nishioka T, Kondo A, Suga K. Progressive blurring of vision in both eyes. *Lancet.* 1995;346(8987):1402. doi:10.1016/S0140-6736(95)92410-8.
 26. Fargen KM, Blackburn S. Surgical decompression for optic neuropathy from carotid artery ectasia: case report with technical considerations. *World Neurosurg.* 2014;82(1–2):239.e9–12. doi:10.1016/j.wneu.2013.06.023.
 27. Uchino M, Nemoto M, Ohtsuka T, Kuramitsu T, Isobe Y. [Unilateral visual field defect due to optic nerve compression by sclerotic internal carotid artery: a case report]. *No Shinkei Geka (Neurological Surgery).* 1999;27:189–194.
 28. Nonaka T, Uede T, Ohtaki M, Tanabe S, Hashi K. [Vascular compression neuropathy of the optic nerve by sclerotic internal carotid artery]. *No Shinkei Geka (Neurological Surgery).* 1987;15:861–866.
 29. Rebollada G, Corcóstegui J, Arruabarrena C, Martínez SMJ, Muñoz-Negrete F. Optociliary shunt vessels in compressive optic neuropathy by the intracranial internal carotid artery. *Eur J Ophthalmol.* 2007;18(2):316–319. doi:10.1177/112067210801800227.
 30. McLaughlin N, Bojanowski MW. Microvascular decompression of the optic chiasm. Case report. *J Neurosurg.* 2011;114(3):857–860. doi:10.3171/2009.9.JNS081658.
 31. Guirgis MF, Lam BL, Falcone SF. Optic tract compression from dolichoectatic basilar artery. *Am J Ophthalmol.* 2001;132(2):283–286. doi:10.1016/S0002-9394(01)00869-8.
 32. Gire J, Hoffart L, Zanin E, Girard N, Ridings B, Conrath J. Optic nerve compression by an intraorbital megadolichoectatic ophthalmic artery. *Orbit.* 2010;29(1):60–62. doi:10.3109/01676830903294677.
 33. Heijl A, Patella V. *Essential Perimetry: The Field Analyzer Primer.* 3rd ed. Jena: Carl Zeiss Meditec, Inc.; 2002.
 34. Danesh-Meyer HV, Yap J, Frampton C, Savino PJ. Differentiation of compressive from glaucomatous optic neuropathy with spectral-domain optical coherence tomography. *Ophthalmology.* 2014;121(8):1516–1523. doi:10.1016/j.ophtha.2014.02.020.
 35. Sergott RC, Balcer LJ. The latest on optical coherence tomography. *J Neuroophthalmol.* 2014;34(Suppl):S1–2. doi:10.1097/WNO.0000000000000160.
 36. Chen JJ. The role of optical coherence tomography in neuro-ophthalmology. *Ann Eye Sci.* 2018;3(6). doi:10.21037/aes.2018.05.08.
 37. Tsutsumi S, Ono H, Yasumoto Y. Vascular compression of the anterior optic pathway: a rare occurrence? *Can Assoc Radiol J.* 2017. doi:10.1016/j.carj.2017.02.001.
 38. Jacobson DM, Warner JJ, Broste SK. Optic nerve contact and compression by the carotid artery in asymptomatic patients. *Am J Ophthalmol.* 1997;123:677–683.
 39. Stroman GA, Stewart WC, Golnik KC, Cure JK, Olinger RE. Magnetic resonance imaging in patients with low-tension glaucoma. *Arch Ophthalmol.* 1995;113(2):168. doi:10.1001/archophth.1995.01100020050027.
 40. Saeki N, Uchino Y, Murai H, Kubota M, Isobe K, Uno T, Sunami K, Yamaura A. MR imaging study of edema-like change along the optic tract in patients with pituitary region tumors. *Am J Neuroradiol.* 2003;24(3):336–342.
 41. Tokumaru AM, Sakata I, Terada H, Kosuda S, Nawashiro H, Yoshii M. Optic nerve hyperintensity on T2-weighted images among patients with pituitary macroadenoma: correlation with visual impairment. *Am J Neuroradiol.* 2006;27:250–254.