

Bulbar Compression by the Vertebral Artery: Four Atypical Cases

Miran Skrap¹, Marco Vindigni¹, Francesco Tuniz²

1. Department of Neurosurgery – Azienda Ospedaliero Universitaria di Udine, Piazzale S.Maria della Misericordia 33100 Udine – Italy 2. Department of Neurosurgery, Azienda Ospedaliero Universitaria di Udine, Piazzale S.Maria della Misericordia 33100 Udine – Italy

✉ **Corresponding author:** Francesco Tuniz, tuniz.francesco@gmail.com

Disclosures can be found in Additional Information at the end of the article

Abstract

Background: Symptomatic compression of the medulla oblongata and the pons by the vertebral artery is thought to be a rare condition; the literature pertaining to this condition consists merely of isolated case studies. Recent clinical experience suggests that this condition may be clinically under-appreciated and that patients with vascular compression of the brainstem are being misdiagnosed.

Methods: Herein, we report on four surgical cases. All cases had a neuroradiological picture characterized by a megadolichovertebral artery pressing on the anterior bulbar surface. It should be emphasized that in all cases, the symptoms were initially attributed to associated pathologies (Chiari malformation, cervical spondylomyelopathy, cavernoma of the fourth ventricle, and hydrocephalus).

Results: In the first few weeks after microvascular decompression of the bulb, the neurological picture of the patients showed clear signs of improvement with a considerable reduction in pre-operative symptoms. After 12 months, the patients with the Chiari malformation, cervical spondylomyelopathy and hydrocephalus continued to improve, whereas the patient suffering from a cavernoma of the fourth ventricle roof suffered partial symptom relapse.

Conclusions: Currently, the major body of literature on compression of the medulla oblongata and the pons by the vertebral artery is sparse and consists only of "case reports". However, we believe that for patients displaying a series of symptoms indicating bulbar compression in the presence of a megadolichovertebral artery, the microvascular decompression of the brainstem should also be considered.

Categories: Neurosurgery

Keywords: megadolichovertebral artery, neurovascular conflict, compression, medulla oblongata, microvascular decompression, mvd

Published 06/15/2011

© Copyright 2011

Skrap et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 3.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

The concept of a neurovascular conflict relating to nerve structures, such as the trigeminal, facial and glossopharyngeal nerves, is today unanimously accepted since it is well-documented from the physiopathological point of view and also clinically well defined [1-3]. The same concept of a neurovascular conflict also exists for the vestibular nerve; however, in this case, indications for surgical treatment are more problematic as clinical indication and preoperative findings are not as well-defined [4-6].

How to cite this article

Skrap M, Vindigni M, Tuniz F (June 15, 2011) Bulbar Compression by the Vertebral Artery: Four Atypical Cases. Cureus 3(6): e28. DOI 10.7759/cureus.28

Compression of the medulla oblongata and the pons by the megadolichovertebral artery is probably not a rare condition. We may think that it is an underestimated situation which rarely comes to the attention of a neurosurgeon. Consequently, in terms of neurosurgical treatment, it has been given relatively little consideration [7-10].

Here, we report on four cases of patients with compression on the anterior bulbar surface caused by a megadolichovertebral artery, for which microvascular decompression surgery was performed.

A peculiarity of these cases lies in the fact that the symptoms were attributed to pre-diagnosed pathologies (a brainstem cavernoma, a Chiari's malformation, a cervical spondylomyelopathy and a hydrocephalus) and not to the direct vascular compression on the medulla oblongata, even though the symptoms did not really fit the initial diagnosis.

Informed consent regarding the benefits and possible surgical risks was given by all the patients before treatment.

Case Presentation

Case 1

Case 1 was a 59-year-old male, known to have a cavernoma of the roof of the fourth ventricle on the left side for the past 12 years (Figure 1a). The patient was indicated as being a high surgical risk and was therefore treated conservatively. His clinical history was characterised by progressive and ingravescent equilibrium disturbances, i.e. diplopia, dysphagia mainly due to fluids, dysphonia with tingling paresthesia of the left hemisoma with sparing of the face, and asthenia to the right with easy fatigability as well as arterial hypertension under drug therapy.

The neurological impression resulted in diagnosis of Wallenberg's syndrome: right facial hypoesthesia; alterations in the temperature and pain sensitivity in the left half of the body, muscular paresis of the soft palate, the throat muscles and the vocal cords on the left side, right lateropulsion (Romberg test), without worsening of eye closure and atactic gait. There was a modest disalignment on the right (Mingazzini test), difficulty in performing fine movements with the right hand, right dysmetria, hypopallesthesia in the left hemisoma, deficiency in the abducens nerve on the left, and psychomotor retardation.

Various instrument tests were carried out such as a Hess screen, the somatosensory evoked potentials (SSEP) and the brainstem activating evoked potentials (BAEP). The Hess screen resulted in a deficiency of the left external rectus being identified. The somatosensory evoked potentials in the upper limbs indicated a bilateral retardation in the afferent conduction at the cord and lemniscus level. The brainstem activating evoked potentials, by stimulation on the left, demonstrated a partial block of the mesencephalic activation associated with partial retardation of the ponto-mesencephalic conduction.

The encephalic MRI and Angio-CT highlighted a long tortuous hyperplastic vertebral artery on the right, and a clear compression on the anterolateral portion of the right medulla oblongata. The contralateral vertebral artery was absent (Figure 1b) (Figure 3).

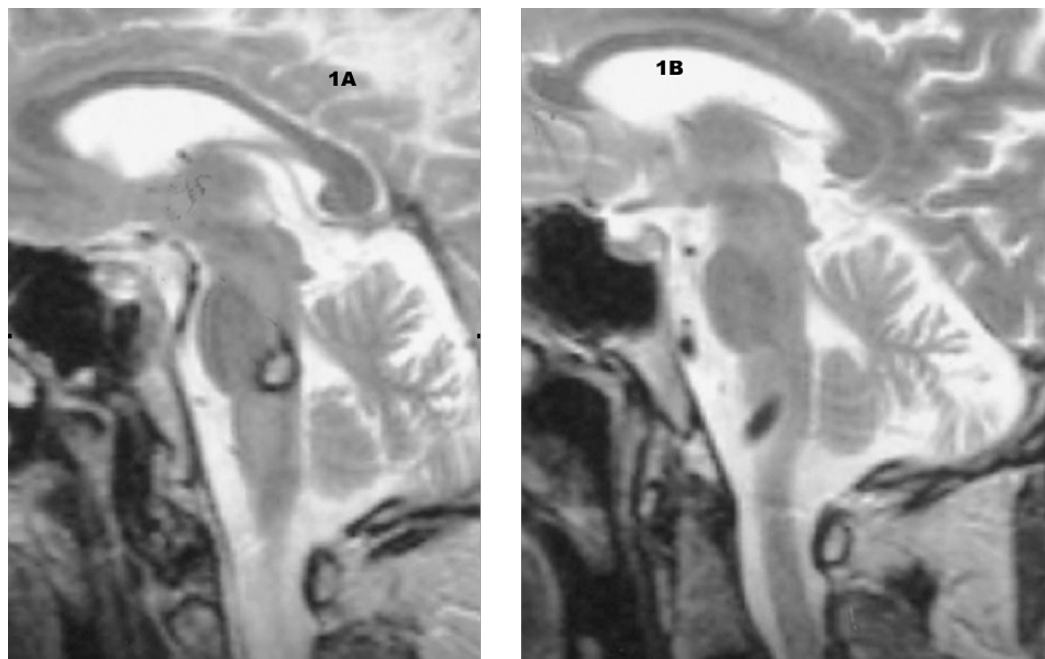


FIGURE 1: Patient 1.

1A. Sagittal T2-weighted MRI scan showing a cavernous angioma located on the floor of the 4th ventricle on the left side. 1B. Sagittal T2-weighted MRI scan showing the vertebral artery compressing the medulla oblongata on the right side.



FIGURE 3: Patient 1. The Angio-TC showing a long tortuous hypertrophic right vertebral artery. The contralateral vertebral artery is virtually absent.

Case 2

Case 2 was a 47-year-old male who had previously (in 1980 and 1994) undergone a cervical laminectomy and suboccipital craniectomy and subarachnoid space shunting due to Chiari II malformation (Figure 2a). After substantial surgical morphological resolution of the hydromyelia and tonsil descent, clinical signs with dysarthria, difficulty in deambulating and hyposthenia of the left hemisoma were observed. The patient experienced hypertension during drug therapy.

After the surgical procedure of cervical laminectomy, suboccipital craniectomy, and subarachnoid space shunting due to Chiari II malformation, the patient reported persistent uncertain deambulation, hyposthenia of the lower limbs, especially on the left side, and grasping defects.

Because of the persistent symptomatology, the patient was referred to our department.

Objectively, the following were noted: ataxia, dysarthria and, with regard to the Mingazzini test, a slight hyposthenia of the left hemisoma. The somatosensory evoked potentials and the brainstem activating evoked potentials were without any significant alterations. The encephalic MRI and Angio-MRI detected the presence of the megadolichovertebral artery inside the pontobulbar sulcus pressing on the left antero-lateral surface of the medulla oblongata. The contralateral vertebral artery was filliform (Figure 2b).

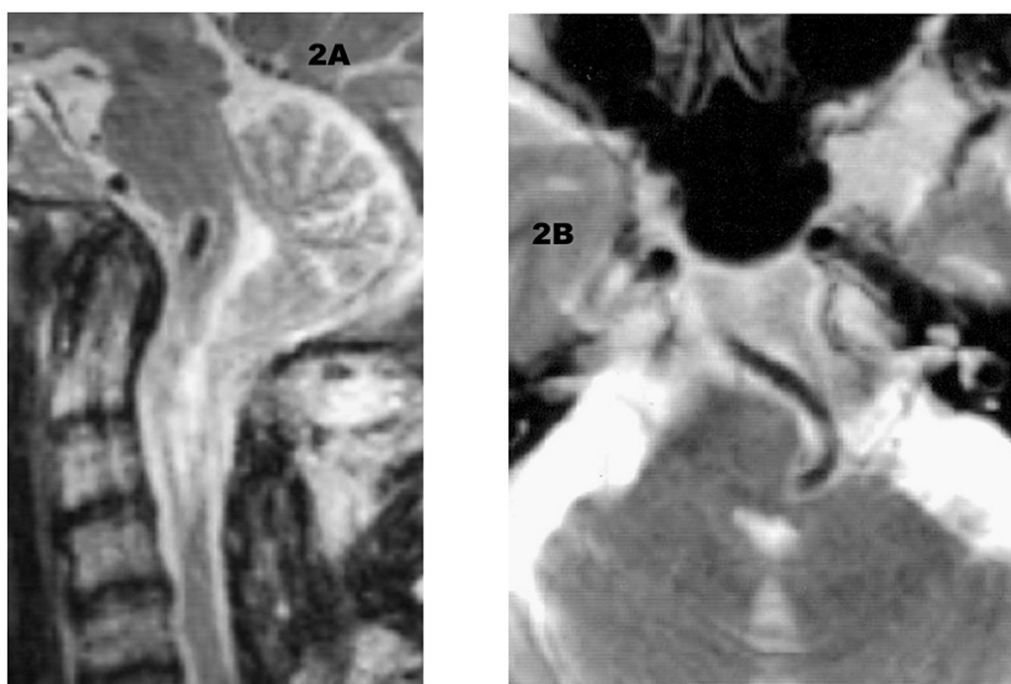


FIGURE 2: Patient 2.

2A. Sagittal T2-weighted MRI scan showing the presence of a Chiari II malformation 2B. Axial T2-weighted MRI scan showing the megadolichovertebral artery inside the pontobulbar sulcus impressing the left antero-lateral surface of the medulla oblongata.

Case 3

Case 3 was a 77-year-old male. The symptomatology began with progressive asthenia and ataxia in the lower limbs. He was first examined on a cervical level and a severe case of spondylodiscoarthrosis on different levels was discovered. However, surgery was not considered because, in the meantime, other neurological symptoms appeared: frenal-dysmetria of the left upper limb and rapid phase nystagmus to the right. The patient experienced hypertension during drug therapy.

Slight hypertonia of the lower limbs with a paraparetic-spastic walk on an enlarged basis was observed. A tripod or double cane was necessary for walking. The neuroradiological examination of the posterior cranial fossa showed a megadolico-basilar picture more clearly impressed on the right trunk. A microvascular decompression was therefore indicated.

Case 4

This 60-year-old man came to our attention because he had an ataxic walk ingravescens in the course of the last six months without cognitive deficits and sphincter disturbances. A cerebral CT scan was made which showed dilation of the ventricular system.

The neurological objectivity was characterized by an ataxic walk, marked positive Romberg with greater oscillation towards the right, the pharyngeal reflex was absent bilaterally, and cerebellar tests showed marked dysmetria on the right.

A cerebral MRI with a contrast medium was performed; besides confirming the hydrocephalus, it showed that the right vertebral artery was strongly ectatic at the ponto-bulbar level. The SSEP were also done which showed a serious block to the primary cortical activation when stimulated on the left and a serious block to medullary segmentary activation on a CV7 level as well as great attenuation of N20 breadth when stimulated on the left. The patient had a history of arterial hypertension which was being treated medically and of cardiopathy from hypertension.

Surgical technique

We have used a retrosigmoid approach which has been slightly enlarged, particularly towards the medial and low direction, which has given us the space we need to follow the dislocated vertebral artery and insert the Gore-Tex. The procedure was performed on the right in three cases and on the left in one case. Once the dura mater was open, the extremely tortuous vertebral artery appeared to have created a "niche" and indentation in the medulla oblongata. Several arachnoid adhesions were detached from the vertebral artery which has been then moved thanks to the absence of perforating arteries in this segment.

Surgical decompression was obtained by retracting the vertebral artery with a Gore-Tex pad (0.1 mm). The patch was wrapped around the vessel and anchored to the dura matter with 0.8 mm stitches, thus retracting the artery away from the brainstem. (Figure 4a) (Figure 4b).

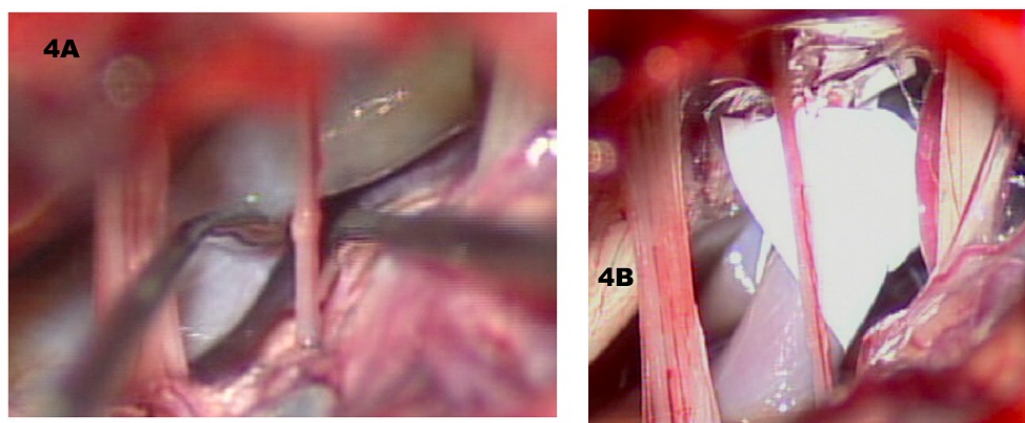


FIGURE 4: Intraoperative picture showing the vertebral artery

4A. Intraoperative picture showing the vertebral artery mobilised with medulla oblongata exposition. 4B. Intraoperative picture showing the vertebral artery retracted with a Gore-Tex pad around the vessel. The patch was encircled around the vessel and anchored to the dura.

Results

In the first case, during the first few weeks after the operation, a clear regression of the symptoms and signs of deficiency was noted. About 12 months later, after a long phase of clinical stabilization, the gait and static disturbances partially reappeared. The arterial hypertension was unvaried. Encephalic MRI controls showed that the introcession of the vertebral artery in the posterolateral medullar sulcus on the right remained unchanged. There were no significant variations in the SSEP and BAEP. The patient refused to undergo a second surgical procedure.

In the second case, immediately after the operation, a progressive regression of the symptomatology was noted and within 12 months the deficiency had completely disappeared. Two years after the operation, the patient's sound condition of health persists; he has resumed all his work and sports activities without any limitation whatsoever. Lower arterial hypertension values were obtained without drug therapy. Finally, encephalic MRI controls show a good stabilisation of the postoperative morphological result. (Figure 5).

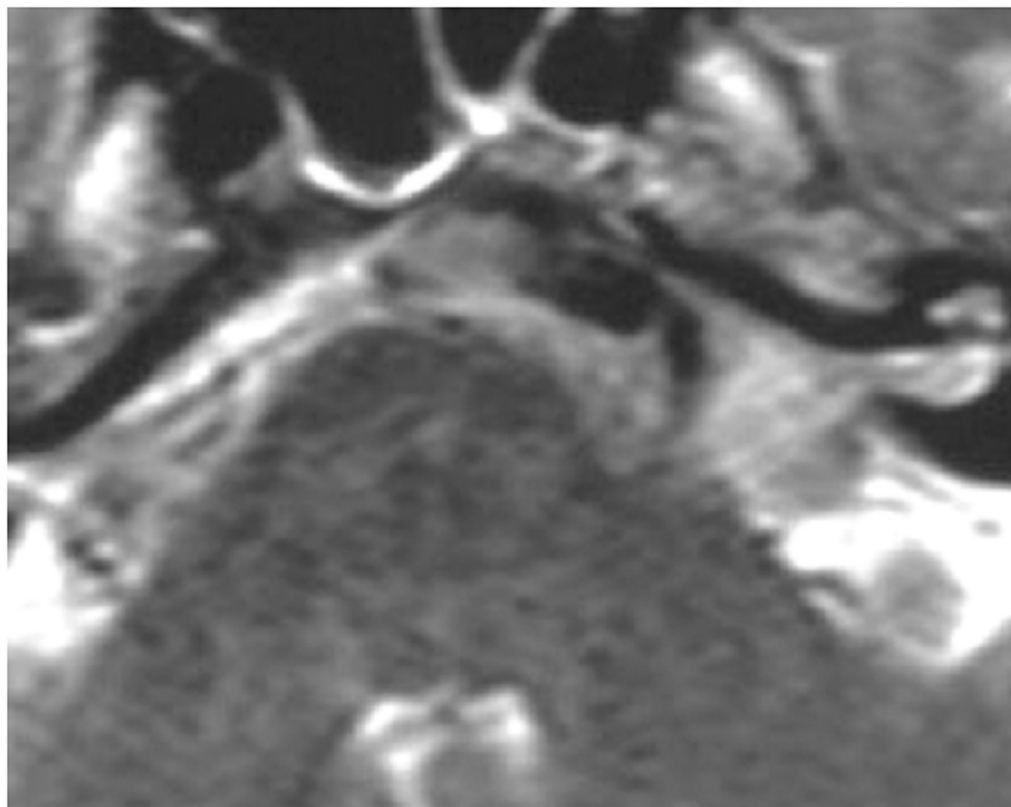


FIGURE 5: Patient 2. Postoperative Axial T1-weighted MRI scan showing good decompression of medulla oblongata

In the last two cases (patients affected also by spondylogenous cervical myelopathy and hydrocephalus), the first postoperative period was characterized by an initial worsening of the symptoms which lasted several weeks. Then there was a slow but constant improvement in all the atactic-dysmetric problems. Twelve months after the operation, examination showed that the first patient still has a modest hypertonic paraparetic-spastic walk, but he no longer needs to use a tripod; in the second case, after six months he has recovered well from ataxia and can walk without help. Four months after the operation, in both cases, the MRI examination showed a dislocation of the megadolico-basilar with less impression on the right trunk. The hydrocephalus in the fourth patient remained unchanged.

Discussion

Compression on the nerve structure by an arterial vessel can produce neurological and vegetative alterations [11-14]. Many, such as trigeminal neuralgia (characterised by paroxysmic pain) and Bell's spasm, are well-known and have been documented for many years [2, 15-18]. However, in contrast, neuralgia of the glossopharyngeal nerve is much rarer and may therefore cause diagnostic difficulties [19-20]. Additionally, in the cases of tinnitus and vertigo, indication for MVD (microvascular decompression) treatment is difficult due to the uncertain differential diagnosis with other pathologies of the inner ear [21-23]. Similarly, difficulties are encountered for nervous or essential hypertension caused by vascular compression on the lateral fossa of the medulla oblongata [24-26]. Other rare cases of neurovascular conflict can give rise to spasmodic torticollis (involving the glossopharyngeal nerve) and cyclic oculomotor spasm with paresis (involving the oculomotor nerve) [27-29]. The common denominator in all these cases is the vessel-nerve contact.

However, since this mechanism for cranial nerves may be well -defined and therefore relatively straightforward to identify, the mechanism of compression at the brainstem is far less common. Indeed, literature pertaining to this condition is very scarce [30-31]. In our opinion, there could be various reasons for this; however, first and foremost, the clinical picture has most likely been unnoticed or undervalued and probably attributed to general vasculopathy, especially when considering that we are generally dealing with vasculopathic patients. Moreover, these patients remain in neurological environments and never undergo surgical observation. Even if a patient does come under the surgeon's observation, not many neurosurgeons are used to performing this type of surgery, and hence the condition may be misdiagnosed. The occurrence of compression could be more frequent due to the tortuosity of the basilar artery rather than that of the vertebral artery. Surgeons who perform MVD are well aware that moving the basilar artery, in the case of nervous conflict, is practically impossible due to the size and rigidity of the vessel.

Finally, the four cases described herein reinforce our theories as outlined above. The patients were sent to a neurosurgeon due to the surgical pathology present, i.e. a cavernoma of the roof of the fourth ventricle, a Chiari malformation, a cervical spondylomyelopathy, and hydrocephalus, but not for compression by a megadolichovertebral artery on the medulla oblongata.

Conclusions

The concept of microvascular decompression is currently well-defined and appears as a method applicable for curing numerous manifestations arising from the hyperfunctioning of cranial nerves in the posterior cranial fossa mentioned previously.

We believe that, although the associated literature is scarce and consists only of case reports, neurovascular conflict on the medulla oblongata is much more frequent than it may seem. This is a diagnostic-therapeutic picture that should be more frequently considered, especially by those active in the clinical neurological environment. However, attention should be paid to distinguishing the cases involving the basilar artery, for which MVD is practically almost unobtainable, from the less frequent cases involving the vertebral artery as a cause of the compression, for which, on the contrary, it is possible to consider surgical treatment to shift and anchor the vessel with decompression of the medulla oblongata.

Additional Information

Disclosures

Human subjects: Consent was obtained by all participants in this study. Waived issued approval Waived. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Fukushima T: Microvascular decompression for hemifacial spasm: Results of 2890 cases . Neurovas Surg. Carter LP, Spetzler RF, Hamilton MG (ed): McGraw-Hill, New York; 1995. 1133-1147. [10.1590/S0004-282X2008000300026](https://doi.org/10.1590/S0004-282X2008000300026)
2. Jannetta PJ, Kassam A: Hemifacial spasm. J Neurol Neurosurg Psychiatry. 1999, 66:255-6. [10.1136/jnnp.66.2.255a](https://doi.org/10.1136/jnnp.66.2.255a)

3. McLaughlin MR, Jannetta PJ, Clyde BL: Microvascular decompression of cranial nerves: lessons learned after 4400 operations. *J Neurosurg.* 1999, 90:1-8. [10.3171/jns.1999.90.1.0001](https://doi.org/10.3171/jns.1999.90.1.0001)
4. Ryu H, Yamamoto S, Sugiyama K: Neurovascular compression syndrome of the eighth cranial nerve. Can the site of compression explain the symptoms?. *Acta Neurochir.* 1999, 141:495-501. [10.1007/s007010050330](https://doi.org/10.1007/s007010050330)
5. Bergsneider M, Becker DP: Vascular compression syndrome of the vestibular nerve: a critical analysis. *Otolaryngol Head Neck Surg.* 1995, 112:118-24. [10.1001/archotol.133.11.1165](https://doi.org/10.1001/archotol.133.11.1165)
6. Moller MB, Moller AR, Jannetta PJ: Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: selection criteria and operative results in 207 patients. *Acta Neurochir.* 1993, 125:75-82. [10.1007/BF01401831](https://doi.org/10.1007/BF01401831)
7. Hongo K, Nakagawa H, Morota N: Vascular compression of the medulla oblongata by the vertebral artery: Report of two cases. *Neurosurg.* 1999, 45:907-10. [10.1097/00006123-199910000-00039](https://doi.org/10.1097/00006123-199910000-00039)
8. Ubogu EE, Chase CM, Verrees MA: Cervicomedullary junction compression caused by vertebral artery dolichoectasia and requiring surgical treatment: Case Report. *J Neurosurg.* 2002, 96:140-143. [10.3171/jns.2002.96.1.0140](https://doi.org/10.3171/jns.2002.96.1.0140)
9. Tomasello F, Alafaci C, Salpietro FM: Bulbar compression by an ectatic vertebral artery: a novel neurovascular construct relieved by microsurgical decompression. *Neurosurg.* 2005, 56:117-24. [10.1227/01.NEU.0000146684.23593.B4](https://doi.org/10.1227/01.NEU.0000146684.23593.B4)
10. Bejjani GK, Sekhar LN: Repositioning of the vertebral artery as treatment for neurovascular compression syndromes. *J Neurosurg.* 1997, 86:728-32. [10.3171/jns.1997.86.4.0728](https://doi.org/10.3171/jns.1997.86.4.0728)
11. Krayenbühl H, Yasargil MG: The vascular diseases in the region of the basilar and vertebral arteries: An anatomopathological, clinical and neuroradiological study. *Fortschr Geb Rontgenstr Nuklearmed.* 1957, 87:1-170.
12. Jannetta PJ, Segal R, Wolfson SK: Neurogenic hypertension: Etiology and surgical treatment—Part II: Observations in an experimental nonhuman primate model. *Ann Surg.* 1985, 201:391-6. [10.1097/0000658-198503000-00023](https://doi.org/10.1097/0000658-198503000-00023)
13. Moss MS, West RJ: Ectasia of the vertebral artery as a cause of isolated pyramidal tract signs: Case report and a review of the literature. *Br J Neurosurg.* 1996, 10:497-499.
14. Salvi F, Mascali M, Bortolotti C: Hypertension, hyperreflexia, and pyramidal paresis due to vascular compression of the medulla. *Neurology.* 2000, 55:1381-1384.
15. Girard N, Poncet M, Caces F: Three-dimensional MRI of hemifacial spasm with surgical correlation. *Neuroradiology.* 1997, 39:46-51. [10.1007/s002340050366](https://doi.org/10.1007/s002340050366)
16. Ogawa A, Suzuki M, Shirane R: Repositioning of the tortuous vertebrobasilar artery for trigeminal neuralgia: A technical note. *Surg Neurol.* 1992, 38:232-235. [10.1016/0090-3019\(92\)90174-L](https://doi.org/10.1016/0090-3019(92)90174-L)
17. Jannetta PJ: Outcome after microvascular decompression for typical trigeminal neuralgia, hemifacial spasm, tinnitus, disabling positional vertigo, and glossopharyngeal neuralgia (honored guest lecture). *Clin Neurosurg.* 1997, 44:331-83.
18. Jannetta PJ: Neurovascular compression in cranial nerve and systemic disease. *Ann Surg.* 1980, 192:518-525. [10.1097/0000658-198010000-00010](https://doi.org/10.1097/0000658-198010000-00010)
19. Sampson JH, Grossi PM, Asaoka K: Microvascular decompression for glossopharyngeal neuralgia: long-term effectiveness and complication avoidance. *Neurosurg.* 2004, 54:884-9, 889-90. [10.1227/01.NEU.0000114142.98655.CC](https://doi.org/10.1227/01.NEU.0000114142.98655.CC)
20. Fischbach F, Lehmann TN, Ricke J: Vascular compression in glossopharyngeal neuralgia: demonstration by high-resolution MRI at 3 tesla. *Neuroradiology.* 2003, 45:810-811. [10.1007/s00234-003-1078-x](https://doi.org/10.1007/s00234-003-1078-x)
21. Moller AR: The cranial nerve vascular compression syndrome: A review of treatment. *Acta Neurochir.* 1991, 113:18-23. [10.1007/BF01402109](https://doi.org/10.1007/BF01402109)
22. De Ridder D, Ryu H, De Mulder G: Frequency specific hearing improvement in microvascular decompression of the cochlear nerve. *Acta Neurochir.* 2005, 147:495-501. [10.1007/s00701-005-0497-3](https://doi.org/10.1007/s00701-005-0497-3)
23. De Ridder D, Ryu H, Moller AR: Functional anatomy of the human cochlear nerve and its role in microvascular decompressions for tinnitus. *Neurosurg.* 2004, 54:381-388. [10.1227/01.NEU.0000103420.53487.79](https://doi.org/10.1227/01.NEU.0000103420.53487.79)
24. Frank H, Schobel HP, Heusser K: Long-term results after microvascular decompression in essential hypertension. *Stroke.* 2001, 32:2950-2955.
25. Levy EI, Scarrow AM, Jannetta PJ: Microvascular decompression in the treatment of

- hypertension: review and update. *Surg Neurol*. 2001, 55:2-10. [10.1016/s0090-3019\(00\)00352-9](https://doi.org/10.1016/s0090-3019(00)00352-9)
26. Levy EI, Clide B, McLaughlin MR: Microvascular decompression of the left lateral medulla oblongata for severe refractory neurogenic hypertension. *Neurosurg*. 1998, 43:1-9. [10.1097/00006123-199807000-00001](https://doi.org/10.1097/00006123-199807000-00001)
27. Shtange LA, Ogleznev K, Grigorian I: The results of microvascular decompression of the accessory nerve in patients with spastic torticollis. *Zh Vopr Neurokhir Im N N Burdenko*. 1998, 1:17-9.
28. Brunori A, Greco R, Bruni P: Surgical treatment of spasmodic torticollis: is there a role for microvascular decompression? With an illustrative case report. *J Neurosurg Sci*. 1996, 40:43-51.
29. Odkvist LM, Thell J, Von Essen C: Vestibulo-oculomotor disturbances in trigeminal neuralgia and hemifacial spasm. *Acta Otolaryngol*. 1998, 105:570-5. [10.3109/00016488809119522](https://doi.org/10.3109/00016488809119522)
30. Milandre L, Bonnefoi B, Pestre P: Vertebrobasilar arterial dolichoectasia: Complications and prognosis. *Rev Neurol*. 1991, 147:714-722.
31. Grasso G, Alafaci C, Passalacqua M: Landmarks for vertebral artery repositioning in bulbar compression syndrome: anatomic and microsurgical nuances. *Neurosurg*. 2005, 56:160-4. [10.1227/01.NEU.0000146685.17628.05](https://doi.org/10.1227/01.NEU.0000146685.17628.05)