Clinical Assessment of Surgical Treatment of Neurovascular Compression Syndromes

Mohamed Eltoukhy[1], Mohamed El shazly Ghaneim[1], Amr AAM Elkatatny[1]

[1]Tutor Department of neurosurgery, Kasr Alainy Medical School Teaching Hospitals, Faculty of Medicine and University Hospitals, Cairo University, Cairo, Egypt

Abstract

Background: Neurovascular compression syndrome is a wide variety of clinical syndromes occurring due to the dysfunction of cranial nerves secondary to compression by a vascular loop. Trigeminal neuralgia is the most common example of this condition. Medical treatment, as carbamazepine, has been long prescribed to control such painful condition. Microvascular decompression operation represents a suitable option for many patients that failed to respond to conservative treatment or unable to be compliant to treatment.

In this article, we investigate 20 different neurovascular compression presentations, surgical nuances and outcomes of microvascular decompression as well as providing the surgical, clinical and radiological methods dealing with two most challenging cases to evaluate the efficacy and safety of this procedure.

Abbreviations*:* *CN: cranial nerves; GN: glossopharyngeal neuralgia; HFS: hemifacial spasm; NVCS: Neurovascular compression syndrome; MVD: Microvascular decompression; TN: trigeminal neuralgia.*

Keywords**:** *neurovascular compression syndromes; trigeminal neuralgia; glossopharyngeal neuralgia; microvascular decompression.*

Introduction

Neurovascular compression syndrome (NVCS) is a wide variety of clinical syndromes occurring due to the dysfunction of cranial nerves secondary to compression. Evidence suggests that the likely etiology is vascular compression at the root entry zone leading to focal demyelination and aberrant neural discharge. Secondary causes such as multiple sclerosis or brain tumors can also produce symptomatic similar diseases.[1]

Treatment must be individualized to each patient. Medical treatment as with carbamazepines has been long prescribed to control such painful condition. Many other interventional techniques are described for the control of these conditions. Microvascular decompression represents a suitable option for many patients that failed to respond to conservative treatment or unable to be compliant to treatment due to side effects.[10]

Methods

§ Patients

This study involved twenty patients, in the period between June 2015 and June 2017, presented with variable signs and symptoms of cranial nerve neuropathies. Causes of the condition were described to be due to vascular compression at the brain stem, trigeminal neuralgia (TN), hemifacial spasm (HFS), intractable vertigo, glossopharyngeal neuralgia (GN) and geniculate neuralgia. Patients were admitted and managed at the Neurosurgery department of Cairo University Hospitals, where data collected prospectively while exclusion criteria included tumors, cysts and vascular malformation.

§ Data Collection

Ø History Taking :

Corresponding author:
Mohamed Eltoukhy
Email drtoukhy.ns@gmail.com
pg@kasralainy.edu.eg

Age, gender, occupation, time of initial complaint till the time of presentation, history of any type of tumors the patient suffered from, history of comorbidities e.g. D.M and smoking.

Ø Clinical Examination :

All patients were clinically evaluated and examined with special care to the ***cranial nerve examination*** especially Trigeminal nerve (**CN V**) and Facial nerve (**CN VII**), motor deficit, or sensory deficit.

Ø Investigations :

a) Routine laboratory investigations.

b) Radiological investigations:

v Computerized tomography (**CT**) of brain and skull.

v Magnetic resonance imaging (**MRI**) of brain and cerebellopontine angle.

v Further imaging modalities as fast imaging employing steady-state acquisition **(FIESTA** sequence) or magnetic resonance angiography (**MRA**) might also be done when needed.

Ø Management

a) Counseling and Consent.

b) Pre-operative Management :

prophylactic antibiotics were given.

c) Operative Management : All 20 patients underwent microvascular decompression (MVD) surgery. Exploration of the cerebellopontine angle (CPA) is performed through retrosigmoid approach in ¾ prone or lateral decubitus position. The margins of transverse and sigmoid sinuses are minimally exposed and the angle between them is well identified. The dura is opened along the line bisecting its angles. Mastoid air cells, whenever encountered, are immediately sealed with muscle and bone wax in order to avoid postoperative cerebrospinal fluid (CSF) leakage. The root entry zone (REZ) at the brainstem is properly explored and the offending vessel is identified. This is helped by observing the characteristic indentation on the corresponding nerve at the REZ and the unique subtle change of color secondary to possible demyelination of the affected portion of the nerve. After freeing the offending vessel off the REZ of the affected nerve, a potential space is created between them using sharp arachnoid dissection, a proper insulator e.g. Polytetrafluoroethylene (PTFE) felt or Dacron is placed. Further inspection is carried out either with the microscope or with the aid of endoscope to ensure that adequate decompression is done and that no unnecessary vascular loops are still in contact with the nerve

d) Follow up and Outcome :

Outcomes were assessed postoperatively, immediately after recovery, few days postoperatively, on discharge and six months postoperatively.

Facial pain is graded according to the Barrow Neurological Institute Pain Scale;

I. No pain, no medication.

II. Occasional pain, no medications required

III. Some pain, adequately controlled with medications.

IV. Pain, partially but inadequately controlled with medications.

V. Severe pain or no pain relief

Other non-painful conditions were evaluated on a similar 5-tier scale to unify the measuring values:

I. complete improvement, no medication

II. Occasional symptoms, no medications required

III. Some symptoms, adequately controlled with medications.

IV. Severe symptoms, partially but inadequately controlled with medications.

V. Severe debilitating or socially disabling disease.

Complications were carefully followed up, regardless of modality of its management, and evaluated according to a 3-tier scale:

0. No deficits or only slight subjective complaints.

1. Slight cranial nerve or cerebellar dysfunction, not

bothersome for daily life.

2. Both subjective and objective cranial nerve or cerebellar dysfunction, problematic for daily life.

Results

v Patients Data:

60% of included patients (12 patients) were males while 40% (8 patients) were females.

Table (1): Patients Age and Sex

	Age Groups (yrs.)					Total
	1	2	3	4	5	
	< 35	35- 45	45 - 55	55-65	> 65	
Male	2(10%)	1 (5%)	6(30%)	2(10%)	1(5%)	12(60%)
Female	0	4(20%)	3(15%)	1 (5%)	0	8 (40%)
Total	2	5	9	3	1	20
Percentage	10%	25%	45%	15%	5%	100%

v Clinical Presentation:

a) Duration of symptoms: 14 patients (70% of cases) had the clinical symptoms of NVCS for 5 years or less while 6 patients (30%) indicated that the symptoms lasted for more than 5 years, with an overall arithmetic mean of 45.65 months and a median of 42 months prior to surgery.

b) Laterality of the disease: About 65% of patients (13 cases) were affected with the right-sided NVCS, and the rest percentage represents 35% of patients (7 cases) had left sided complaint.

c) Affected nerve root, typical and atypical presentations:

Trigeminal nerve was the mostly affected cranial nerve reported in 17 cases (85%), followed by facial nerve in 2 cases (10%) with hemifacial spasm and 1 case (5%) with glossopharyngeal neuralgia. However, Trigeminal nerve roots were affected variably among TN patients. 8 patients experienced symptoms related to mandibular division **V3** (7 patients with typical TN symptoms and 1 patient with atypical symptoms of burning sensation and pain that persisted between the attacks), 7 patients had symptoms related to both mandibular **V3** and maxillary **V2** divisions and only 2 patients experienced affection of the whole 3 divisions of trigeminal nerve as shown in figure (1).

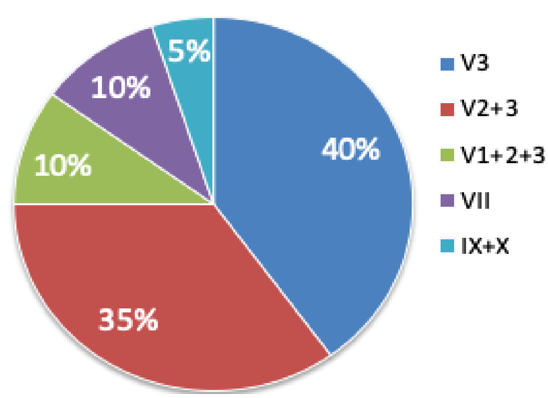

Figure (1): *Distribution of NVCS patients in regard to affected cranial nerves.*

V: Trigeminal nerve; VII: Facial nerve; IX+X: lower cranial nerves

(IX: Glossopharyngeal nerve; X: Vagus nerve)

Overall, the majority of patients in the study, 17 cases (85%), had typical presentations of NVCS and only 15 % of the cases had atypical form of presentation either objectively in symptoms or subjectively during the neurological assessment done after admission (table 2).

Table (2) Distribution of NVCS patients in regard to affected nerve root, typical and atypical clinical presentations.

Affected Nerve		Clinical Presentation		
		Typical (%)	Atypical (%)	Total (%)
Trigeminal (V) Nerve	V3	7 (35%)	1(5%)	8 (40%)
	V2+3	7 (35%)	0	7 (35%)
	V1+2+3	1 (5%)	1 (5%)	2 (10%)
Facial Nerve		1 (5%)	1 (5%)	2 (10%)
Lower Cranial Nerves		1 (5%)	0	1 (5%)
Total		17 (85%)	3 (15%)	20 (100%)

d) Severity of illness: All patients had severe intractable disease graded by a 5-tier scale, as discussed earlier, upon their admission. Majority of patients (60%) were graded four out of five with symptoms that decreased by medical treatment but yet inadequately as the disease was still incapacitating. However, only 8 patients (40%) had unimproved symptoms with any conservative treatments.

v Intraoperative Findings:

The offended vessels were identified in figure (2) as follows:

1) Superior cerebellar artery (SCA) in 11 cases (55%) with TN, about 64.7% of them were presented with the same manifestation of the disease.

2) Anterior inferior cerebellar artery (AICA) in 5 cases (25%). About four cases of them represent (23.6%) of total number of TN patients.

3) Posterior inferior cerebellar artery (PICA) in 2 cases (10%) with hemifacial spasm for one patient (5%), and glossopharyngeal neuralgia for the other one (5%).

4) One case of TN (5.8% of TN patients and 5% of the total number of cases) had a venous loop as the compressing vessel.

5) Last patient showed a compression by ectatic vertebrobasilar artery (VBA) (5.8% of TN patients and 5% of the total number of cases).

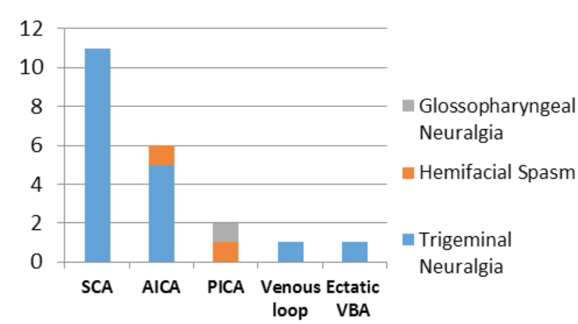

Figure (2): Offending vessels as seen microscopically intraoperatively.

SCA: Superior cerebellar artery; AICA: Anterior inferior cerebellar artery; PICA: Posterior inferior cerebellar artery; VBA: Vertebrobasilar artery v Postoperative Outcomes:

Upon discharge; complete recovery was reported in 70% of patients with an excellent disease control at discharge with no need of further medications, a lesser control of the disease was reported in 25% of the patients who showed sporadic symptoms that didn't require the use of medications while the remaining 5 % of patients needed a small dose of medications to control recurrence of symptoms.

Six months follow up reported decreased percentage of patients experienced symptoms of recurrence (from 35% to 5%) with no drug dependency, indicating that percentage of symptom-free patients raised to 90% and only 5% of cases still needed control with medications.

Non-specific complications, as postoperative minor headache and nausea, were insignificant to our study and self-limiting. Significant complications were encountered in 5 patients with no reported mortalities:

· 1 patient experienced postoperative disequilibrium and posterior fossa venous insufficiency symptoms.

· 2 patients had partial facial palsy (H&B Grade II and III respectively) that completely resolved in one patient and persisted in the other one.

· 1 case had postoperative tinnitus and sensorineural hearing loss.

· 1 case had CSF leak that was managed initially with conservative measures and failed, then was treated by temporary lumbar subarachnoid drain insertion for two days.

Case Presentation

Case (1)

A 59 years old male presented with an eight year recurring left sided facial pain, fitting the clinical picture of typical TN affecting both mandibular **V3** and maxillary **V2** distributions. Carbamazepine was taken to the maximum dose with only partial relief. The patient tried percutaneous injection that provided only temporary relief of the symptoms.

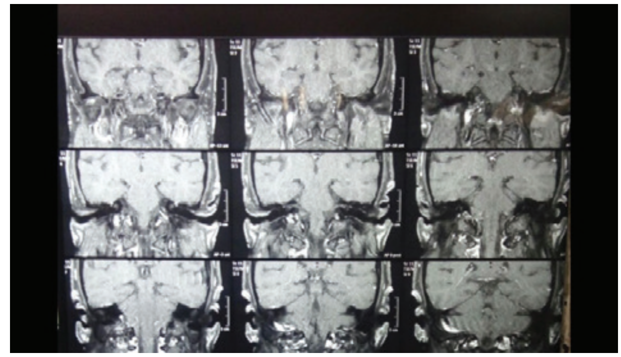

Figure (3): *Patient (1) MRI/ T1WI coronal cuts shows no structural lesions affecting trigeminal nerve*

Patient was operated upon by microvascular decompression of the left trigeminal nerve. The offending vessel was found to be the **superior cerebellar artery (SCA)**. It compressed the nerve from the medial and anterior aspects.

Figure (4): *Intraoperative findings in patient (1)*

*A: Shows the **superior cerebellar artery** (black arrow) compressing the **left trigeminal nerve** (white arrow) at the root entry zone; B: Sharp arachnoid dissection is carried on; C: Insulator was put in the plane created between the nerve and the vessel; D: Final exploration of the root entry zone.*

The patient went through a smooth postoperative period with an improvement of pain to VAS score 2. No postoperative complications were recorded apart from mild headache. Patient complete recovery was recorded with one week and six months postoperative follow-up with no neurological deficits.

Case (2)

A 37 years old male patient presented with gradual onset and progressive course of involuntary, irregular, clonic movements of the right sided facial muscles. It initially involved the orbicularis oculi muscle, followed by gradual spread to the other parts of face. The patient was experiencing more than twenty attacks per day and each one lasts for about one minute the thing that disabled him socially. The complaint lasted for three and a half years during which he sought medical advice and was prescribed medical treatment and a trial of botulinum toxin injection was done; however, the improvement was unremarkable. He had no relevant medical or surgical history and no special habits of medical importance.

MRI neuromaging showed no tumors, cysts or malformations that could cause the condition (figure 5).

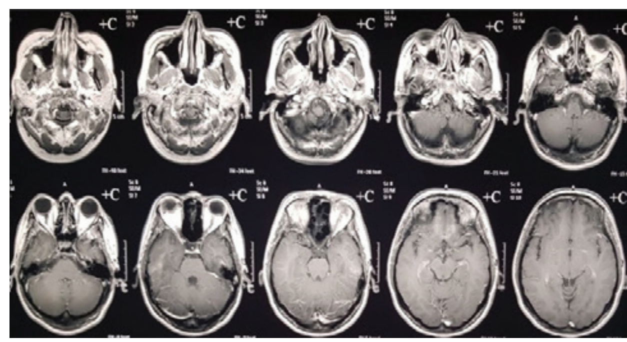

Figure (5): *Patient (2) MRI Brain T1WI for exclusion of any tumors that could cause the hemifacial spasm*

The nerve was found to be compressed medially by the **anterior inferior cerebellar artery (AICA).** Dissection of the artery off the nerve and an insulator was put to ensure the new position of the artery. Endoscopy was used to explore the root entry zone to ensure adequate decompression. **Another loop** of the same artery was found using the 30 degree-angled lens and additional maneuver using insulator was performed. (figure 6).

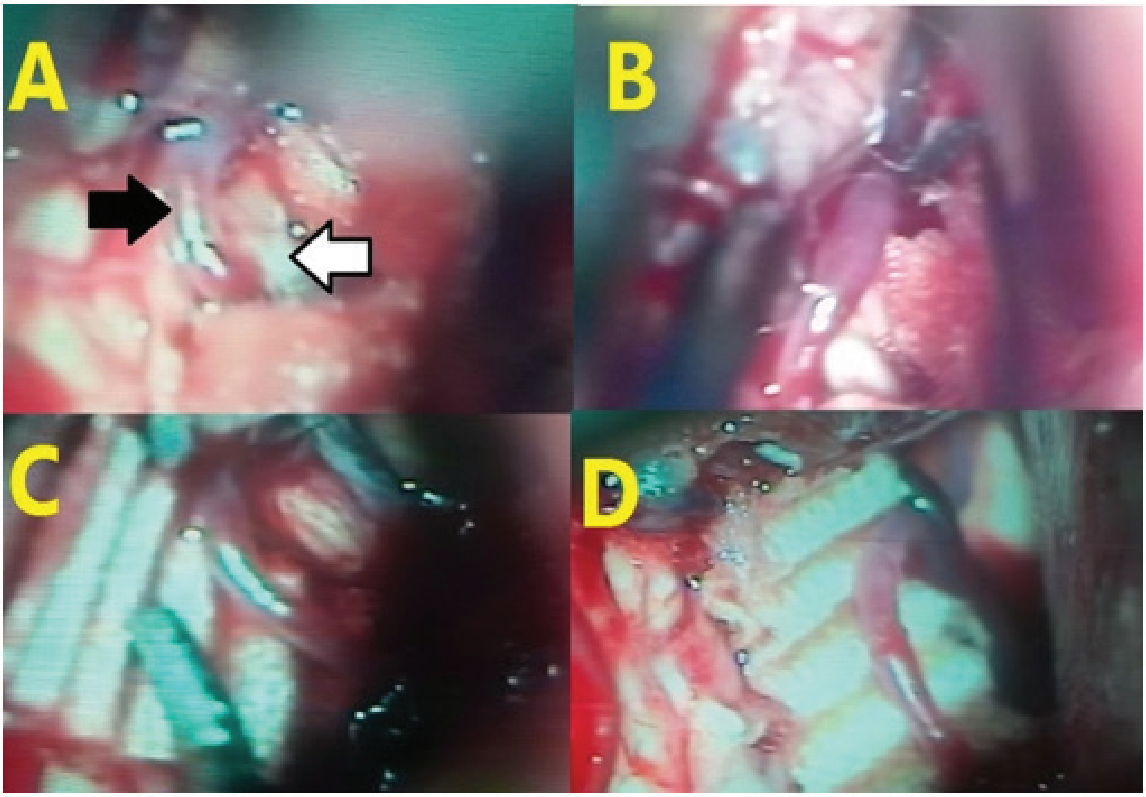

Figure (6): Patient (2) facial nerve decompression. The facial nerve (white arrow) is being compressed at the root entry zone by anterior inferior cerebellar artery (white stars).

Immediate postoperative period was smooth with no postoperative complications. The patient had partial improvement of the condition immediate postoperatively with preserved full facial functions.

One week, one month and six months postoperative patient follow-up showed good wound healing, more improvement of the condition and complete resolution of the abnormal movements respectively.

Discussion

This study included 20 patients presented at Cairo University Hospitals with the clinical syndromes of neurovascular compression of cranial nerves at the brain stem. Male to female ratio was 1.5:1, indicating male predominance. However female patients were presented at younger ages than males. This finding is a quite similar to that indicated by ***Jagannath et al*** who conducted a prospective study, on 182 patients underwent MVD operations and included 84 males (61.3%) and 53 females (38%) with a ratio of 1.5:1. Nevertheless, this finding is contradicted by other studies who indicated predominance of females and a higher means of ages at the time of presentation.[11,12]

35 % of the total cases and 41.1% of total number of patients of trigeminal neuralgia had symptoms related to both the mandibular **V3** and maxillary **V2** nerves distribution, while only 2 patients experienced a disease that affected the whole distribution of the trigeminal nerve with a percentage of 10% of the total cases and a percentage of 11.7% of the total cases of trigeminal neuralgia. This study also included 2 cases of hemifacial spasm with a total percentage of 10% and a case of glossopharyngeal neuralgia with a percentage of 5%.

Jagannath et al, in their study in 2012, found that the mandibular **V3** and maxillary **V2** divisions of the trigeminal nerve predominated with about 68% of the cases.[11] Another study, ***Sandel and Eide*** in 2013 published a retrospective review article of patients with typical and atypical presentations; 65 % of trigeminal neuralgia patients were without constant pain and 35 % were with constant pain while for hemifacial spasm patients the percentage of division between the two groups was 95 % for typical presentation and 5 % for atypical disease.[13] Whereas, the duration of symptoms prior to NVD surgery according to ***Slettebø and Eide,*** ranged from 5 to 10 years with median of 7 years and all of the study's patients had typical presentation of the disease.[12]

During our surgical procedures, the offending element had been identified as follows; 55% was the superior cerebellar artery (SCA) all of which were cases of trigeminal neuralgia, the anterior inferior cerebellar artery (AICA) with a percentage of 25% of the cases. 10% cases were found to have the posterior inferior cerebellar artery (PICA) as the offending vessel. A venous loop as the compressing vessel represented only 5% of the total number of cases and 5.8% of total number of trigeminal neuralgia cases, 5% of cases also showed compression by an ectatic vertebral-basilar artery (VBA). As for other authors' experiences with offending vessels, ***Jagannath et al*** found that the superior cerebellar artery was the commonest cause of compression in 71.5% and more than one artery were found in relation to the nerve in 15.3%.[11] While according to ***Zhong et al.***, the offending vessels were observed to be arteries, artery combined with vein or veins only. Comparatively to our study, more than one offending vessel were found (74%), which included (SCA, 41%), (AICA, 29%), petrosal vein(s) (35%), (PICA, 9%) and vertebral artery (VA, 6%).[14]

While most patients in our study had excellent disease control at discharge as 70% were being completely symptom-free with no need of further medications (GI), and 25% of the patients showed a lesser control of the disease having sporadic symptoms that didn't require the use of medications (GII), while the remaining 5 % of the patients showed less favorable outcome with the need for medications, nevertheless, in a small dose to control recurring those symptoms (GIII). Six month follow-up, the percentage of patients that experienced recurrence of symptoms with no drug dependency dropped from 25% to 5% and the percentage of symptom-free patients raised to 90% of the patients, while 5% of the cases still were dependent on a low dose of medications. Similarities were found between our results and previous ones. ***Zhong et al.*** recognized that pain free or spasms cease occurred in 88.3%, improved at some degree in 7.2% and the symptoms were unimproved at all in 4.5%.[14] Another study was for ***Sandel and Eide*** in 2013, where 54.4 % of the hemifacial patients reported immediate spasm relief after MVD. Within 3 months, 75.4 % had improved and 7% of the patients reported that their symptoms relieved more 6 months after surgery.[13] Recently, ***Wang et al.*** concluded that out of the 55 patients included in their study, 39 patients (76.5 %) had achieved control of their pain, 34 (67.7 %) being totally pain-free (BNI I) with an excellent result of the procedure and five having a good result while still experiencing some forms of mild pain but not requiring any medication (BNI II). For these 39 patients, relief was immediate in 29 (74.3 %) and delayed in 10 (25.6 %). Outcome was not satisfactory in the 16 other patients (29 %). four patients had a recurrence of pain with a 3.9 years average lapse (range 1.7-5.7 years). Of these 16, four patients underwent a second procedure: thermo-rhizotomy in three and balloon compression in one.[15]

On the basis of our experience and a review of literature, we conclude the following:

1- The diagnosis of neurovascular compression syndromes is made on a clinical basis; however, modern imaging techniques can be beneficial in visualizing the vessel-nerve conflict

2- Microvascular decompression is relatively safe and cost-effective procedure that provide both satisfactory and long standing results.

3- Proper anatomy knowledge is crucial to surgeons performing this operation.

4- Although the operation carries morbidities, most of them are not serious and usually transient and well controllable.

Auxiliary tools such as endoscopies and intraoperative neurophysiologic monitoring are gaining more and more importance in the management and reducing the complications of these operations

Funding: Self-funding

Ethical Clearance: taken from

Ethical committee of Cairo university.

Conflict of Interest: Nil

References

1. Haller S, Etienne L, Kövari E, et al. Imaging of neurovascular compression syndromes: trigeminal neuralgia, hemifacial spasm, vestibular paroxysmia, and glossopharyngeal neuralgia. AJNR Am J Neuroradiol 2016;37:1384–92.
2. Pearce JM. Migraine: a cerebral disorder. The Lancet. 1984 Jul 14;324(8394):86-9.
3. Lewy FH, GRANT FC. Physiopathologic and pathoanatomic aspects of major trigeminal neuralgia. Archives of Neurology & Psychiatry. 1938 Dec 1;40(6):1126-34.
4. Cole CD, Liu JK, Apfelbaum RI. Historical perspectives on the diagnosis and treatment of trigeminal neuralgia. Neurosurgical focus. 2005 May;18(5):1-0.
5. Dandy WE. Trigeminal neuralgia and trigeminal tic douloureux. In: Lewis D, ed. Practice of Surgery. Hagerstown, MD: WF Prior CO, 1932: 177-200
6. Dandy WE. Concerning the cause of trigeminal neuralgia. The American Journal of Surgery. 1934 May 1;24(2):447-55.
7. Stone J, Goodrich JT, Cybulski GR . John Hunter's contribution to neuroscience, in Whitaker H , Smith CUM, Finger S (eds): Brain, Mind and Medicine : Essays in Eighteenth Century Neurosceince. New York, Springer, 2007, pp. 67-84
8. Frazier CH. Operation for the radical cure of trigeminal neuralgia: analysis of five hundred cases. Annals of surgery. 1928 Sep;88(3):534.
9. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. Journal of Neurosurgery 1967: 26: 159-162.
10. Luzzi S, Del Maestro M, Trovarelli D, et al. Endoscope-Assisted Microneurosurgery for Neurovascular Compression Syndromes: Basic Principles, Methodology, and Technical Notes. Asian J Neurosurg. 2019;14(1):193–200.
11. Jagannath PM, Venkataramana NK, Bansal A, Ravichandra M. Outcome of microvascular decompression for trigeminal neuralgia using autologous muscle graft: A five-year prospective study. Asian journal of neurosurgery. 2012 Jul;7(3):125.
12. Slettebø H, Eide PK. A prospective study of microvascular decompression for trigeminal neuralgia. Acta neurochirurgica. 1997 May 1;139(5):421-5.
13. Sandel T, Eide PK. Long-term results of microvascular decompression for trigeminal neuralgia and hemifacial spasms according to preoperative symptomatology. Acta neurochirurgica. 2013 Sep 1;155(9):1681-92.
14. Zhong J, Li ST, Zhu J, Guan HX, Zhou QM, Jiao W, Ying TT, Yang XS, Zhan WC, Hua XM. A clinical analysis on microvascular decompression surgery in a series of 3000 cases. Clinical neurology and neurosurgery. 2012 Sep 1;114(7):846-51.
15. Wang DD, Raygor KP, Cage TA, Ward MM, Westcott S, Barbaro NM, Chang EF. Prospective comparison of long-term pain relief rates after first-time microvascular decompression and stereotactic radiosurgery for trigeminal neuralgia. Journal of neurosurgery. 2017 Feb:1-0