The treatment of trigeminal neuralgia with microvascular decompression. The role of magnetic resonance angiography in the indication of surgical treatment in trigeminal neuralgia

Summary of Ph.D. Thesis

Ádám Kuncz

Department of Neurosurgery
Albert Szent-Györgyi Medical and Pharmaceutical Center
Faculty of General Medicine
University of Szeged

Szeged, 2006

List of abbreviations

CSF	-	cerebrospinal fluid
CT	-	computer tomography
MRA	-	magnetic resonance angiography
MRI	-	magnetic resonance imaging
MVD	-	microvascular decompression
NC	-	neurovascular compression
PIFP	-	persistent idiopathic facial pain
TN	-	trigeminal neuralgia
TNWIP	-	trigeminal neuralgia with interparoxismal pain
3 D TOF	-	three dimensional time of flight

I. Introduction

The first exact description of trigeminal neuralgia (TN) is originated from John Locke in 1677. The term *tic douloureux* was given by Nicholas André in 1756 unequivocally referring to the spasms of the face caused by the intolerable pain. In 1773, John Fothergill described 16 cases of periodic, shock-like facial pain. Since then, the periodic painful attacks of the face have become known as the syndrome of TN.

I. 1. The clinical features of TN

TN is a condition characterized by electric shock-like, excruciating paroxysms of pain in the distribution of the trigeminal nerve, most often in the maxillary and mandibular nerves. The pain during any single episode is always unilateral and seldom lasts for more than a few seconds or a minute. The paroxysms can recur frequently, sometimes several times a day. The interval between the pains becomes shorter and shorter, indicating the progressive nature of the disease. Another characteristic feature is the initiation of pain by obvious tactile stimuli applied to certain areas on the face, lips or tongue, the "trigger zones", or by movements of these parts. Flushing of the affected side of the face and watering of the eye are usual. The attack usually stops abruptly, but may linger with diminishing severity and is followed by a refractory period of up to 2 or 3 minutes. Usually, the patient chooses at all costs to avoid any form of physical stimulation of the face, but a few patients obtain amelioration by applying very firm pressure or rubbing the affected part.

I. 2. Epidemiology of TN

TN is an uncommon cause of facial pain, occurring with a mean annual incidence of 4 cases per 100 000 population. The incidence of TN increases with age, predominantly affects the older age group. There is a slight predominance of females to males, the right side of the face is slightly more frequently affected than the left, and rarely bilateral TN can occur.

I. 3. Pathomechanism of TN

I. 3. 1. Nomenclature

In many cases of TN, well-circumscribed pathological conditions can be revealed in the background by imaging (CT and MRI). In these cases, the condition is called **symptomatic TN**. The pathological process may affect the sensory center and pathways of the trigeminal nerve in the brainstem. In these cases, the origin is **central**. The effects of pressure, tension, distortion, some kind of lesion by a tumor, vascular malformation, aneurysm, inflammation, developmental disorder and trauma of the skullbase, etc. on the peripheral structures of the trigeminal nerve (Gasserian ganglion and nerve roots) cause TN of **peripheral** origin. If no other cause than vascular compression of the trigeminal nerve can be identified, the TN is called **classical** (idiopathic or genuine).

I. 3. 2. Peripheral causes

The arguments for a peripheral extrinsic origin include pathological features often seen at the level of the posterior root and occasionally at the level of the Gasserian ganglion. Compression, distortion or stretching of the trigeminal nerve by a normal or aberrant vessel, aneurysm, arteriovenous malformation, a tumor, dural thickening, etc., most often in the posterior and less frequently in the middle cranial fossa, can cause TN. Irritation of trigeminal terminals by chronic oral and dental disease may also be the cause of tic douloureux.

I. 3. 2. 1. Histological examinations aiming at the pathomechanism of TN

In the 1960-s, when excision of the Gasserian ganglion was an accepted surgical method for the treatment of TN, samples were taken from the Gasserian ganglion and the near sensory nerve roots. Electron microscopic studies of the specimens have shown a pathology characterized by vacuolated ganglion cells, degenerative hypermyelination, and segmental demyelination with denudation of the axons.

During 12 microvascular decompressions (MVD) biopsies were taken from the trigeminal nerves at the site of neurovascular compression (NC) and the samples were investigated electron microscopically. A strong correlation could be found between the NC and the histological changes. In the cases of the most severe NC, there were areas where only a few axons remained and all of them were demyelinated. In this demyelinated areas the surviving nude axons were in bundles in close membrane-to-membrane contact. Adjacent areas contained more surviving axons, including a significant proportion having a residual myelin sheath, albeit disrupted. In all cases, the degenerative changes were most severe at the site of NC, and distant from this sitethey became less.

I. 3. 2. 2. The pathophysiology of pain paroxysm in TN

Sensory neurons frequently become electrically hyperexcitable when injured and a source of an abnormal spike discharge. In cases of TN, such ectopic pacemaker activity could arise from the following:

1) dys- or demyelinated root axons;

2) swollen endbulbs and sprouts at the end of severed axons; and
3) axotomized cell somata within the trigeminal ganglion.

When ectopic firing occurs spontaneously, it gives rise to background paresthesias and burning sensations. Other injured sensory neurons are silent, but have a hair-trigger threshold such that momentary stimulation induces a burst of spontaneous firing lasting for seconds or even minutes. This triggered activity or neuronal afterdischarge, is considered to be the background of the ignition hypothesis i.e. the cause of the pain paroxysms associated with TN. Although the afterdischarge is set off by an external stimulus, the spiking itself is self-sustaining, a reflection of the intrinsic repetitive firing tendency of injured afferent neurons. Afterdischarge bursts may also occur without any obvious trigger stimulus.

The intense paroxysms of pain and their sudden onset indicate that something synchronizes the afterdischarge burst in large numbers of trigeminal ganglion neurons. The ignition hypothesis proposes that synchronization of the afterdischarge is a result of neuron-to-neuron crossexcitation at the site of root compression or in the trigeminal ganglion.

I. 3. 3. Central mechanism

Pathological functions of the sensory nuclei in the brainstem and spinal cord of the trigeminal nerve can be suspected in those cases where no anatomical lesion can be identified with sensitive imaging methods. The several ideas of central mechanisms are based on complicated neurophysiological and neurohormonal processes which are theoretical at present. A central mechanism can be considered in cases of multiple sclerosis where demyelinated foci can be proved in the brainstem via MRI.

I. 4. The clinical management of TN

I. 4. 1. Medical treatment

Carbamazepine, phenytoin, Baclofen, clonazepam, gabapentin or a combination of these agents are the first line of treatment. Carbamazepine, which has proved to be the most beneficial remedy in TN, is effective in more than 80% of the patients initially, but 25% later become refractory to the treatment. If medical treatment fails to relieve the pain or if side-effects force discontinuation of the treatment, surgery deserves consideration.

I. 4. 2. Surgical treatment

I. 4. 2. 1. For many years, TN was treated with various kinds of **ablative methods**; many of them had severe side-effects and impaired the nerve functions and they are no longer recommended. There are still ablative methods, such as the controlled thermocoagulation of the Gasserian ganglion and rootlets, the glycerol rhizotomy and balloon compression of the Gasserian ganglion which are widely used with moderate side effects. Recently the stereotactic irradiation of the entry zone of the trigeminal nerve has been also used. In contrast with these ablative methods, which provide treatment of the symptoms, MVD is a nonablative surgical method in which the normal anatomy is restored.

I. 4. 2. 2. History of MVD

In 1934, Walter Dandy published his observation during lateral suboccipital exposures for TN that vascular compression of the trigeminal root is a major cause of TN. In 1959, Gardner & Miklos first reported the treatment of TN by a neurovascular decompression procedure. The first MVD was performed in 1966. Over the subsequent 35 years, the work of Jannetta and other neurosurgeons led to this procedure becoming an accepted mode of treatment for TN. In Hungary, the first MVD was accomplished by Mérei with the active assistance of Jannetta in 1973. From that time on up to 1989, only a few MVDs were performed in the country, until our team started to use this method regularly to treat TN.

I. 4. 2. 3. Surgical anatomy

NC of the trigeminal nerve may be caused by arteries and veins. Most frequently, the superior cerebellar artery is the compressing vessel as it normally courses near the entry zone of the trigeminal nerve; however, any elongated, tortuous artery may create a loop affecting the nerve. The veins also display great variations in this region and may compress the nerve.

The most frequent site of NC is at the entry zone of the trigeminal nerve to the pons. The significance of this fact is that the nerve root near the brainstem is a central nervous system segment containing an oligodendroglial myelin sheet, while the distal part of the nerve contains a Schwann cell-produced myelin sheet. The former is very sensitive, while the peripheral part is more resistant to compression.

II. Aims of the study

1. To learn the surgical method of the "keyhole" approach to the cerebellopontine angle and to find the optimal positioning of the patient.
2. To evaluate the relation between three facial pain groups (typical TN, TN with non-neuralgic interparoxysmal pain (TNWIP) and persistent idiopathic facial pain (PIFP)) and the magnetic resonance angiography (MRA) findings.

3. To assess the value of MRA as indicated by the clinical symptoms and surgical findings.
4. To judge the correlation between the clinical symptoms, the type of vascular compression and the outcome of MVD.
5. To evaluate the surgical and neurological complications following MVD, and the measures to prevent them.

III. Patients and methods

III. 1. Clinical symptoms of the patients

Three dimensional time of flight (3D TOF) MRA was performed in 310 patients with typical TN (214, 69%), TNWIP (65, 21%) or PIFP (31, 10%). Symptomatic TN cases were excluded. The three clinical groups were created in accordance with the International Classification of Headache Disorders. The "classical TN" group was divided into two subgroups. The third group involved PIFP, previously known as atypical facial pain. The characteristics of the three clinical groups are as follows:

1. Typical TN: Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve. The pain is intense, sharp, superficial or stabbing and/or precipitated from trigger areas or by trigger factors. Attacks are stereotyped in the individual patients. There is no clinically evident neurology deficit. TN is not attributed to another disorder. The pain never crosses to the opposite side, but it may rarely occur bilaterally. Following a painful paroxysm, there is usually a refractory period during which pain cannot be triggered. The TN is usually responsive, at least initially, to pharmacotherapy.
2. TNWIP (atypical TN): the same as above, but there is a dull background pain.
3. PIFP: pain in the face, present daily and persisting for all or most of the day. The pain is confined at onset to a limited area on one side of the face, and is deep and poorly localized. The pain is not associated with sensory loss or other physical signs.

III. 2. Imaging techniques

The 3 D TOF MRA investigations were made on two MR units: a 0.5-Tesla (T) Elscint Gyrex VD1x and a 1-T Signa Horizon LX.) Maximum-intensity projections in 3 standard planes (coronal, sagittal and axial) were focused on the nerve root entry zone of both trigeminal nerves. The thin-slice reconstructed images were used to clarify the fine anatomical relationship of the nerve and the vessels. All measurements were repeated after the intravenous administration of gadolinium to visualize the veins. The 3 D TOF MRA performed without contrast material visualizes the arteries, while with contrast material the veins can also be seen. Neurovascular compression (NC) was predicted if distortion of a nerve, caused by a vessel, was observed in the 3 D TOF MRA images, or there was no visible gap between the trigeminal nerve and the vessel (close contact).

III. 3. Surgical considerations

III. 3. 1. Indications for operation

MVD was considered only in those cases where NC was detected in the MRA images in accordance with the symptoms, and the medical treatment failed. MVD was offered if the patient was judged to be suitable for general anesthesia and accepted MVD.

All patients who met the criteria were enrolled in the surgical study between January 1994 and December 2004. MVD was performed in 116 cases in 114 patients (2 patients had bilateral TN and MVD). From the MRA-negative group, 6 patients were operated on by selective trigeminal rhizotomy.

III. 3. 2. Operative technique

The MVD is performed according to Jannetta. Most of the patients are operated on in a supine position. A retromastoid craniectomy is performed in the angle of the lateral and sigmoid sinus. The cerebellum is retracted and the cerebellopontine cistern is opened. NC has to be looked for according to the result of MRA and, additionally, every other compression has to be treated. The compressing vessel has to be mobilized as much as possible. A piece of gelatin foam, shredded Teflon felt, or artificial vessel implant is placed between the vessel and the nerve. In some venous compression cases, the veins are coagulated and dissected. A watertight dural closure is important to prevent cerebrospinal fluid (CSF) leakage. The bone edges are carefully waxed and mastoid cells are plugged with muscle pieces. The dura is covered with oxidized cellulose and/or gelfoam, and the wound is then closed in layers.

III. 4. Data collection and evaluation

III. 4. 1. Patients` follow up and outcome

The clinical symptoms, MRA findings and surgical observations were recorded. The surgical outcome was assessed 1 week, 6 months, and 1, 2, 4, 6, 8 and 10 years postoperatively. The outcome measures were classified according to the *Table 1.*

The recurrence was classified as moderate, which corresponds to the criteria of a good outcome, or as severe, which corresponds to the criteria of a poor outcome.

Table 1
Outcome measures

Outcome	Postoperative pain	Additional treatment
Excellent	Without Almost without	None
Good	Without Almost without	Intermittent low-dose medication
	Significantly less Moderately less	None/Intermittent low-dose medication
Poor	Without Almost without Significantly less Moderately less	High-dose medication
	No change Worse	High-dose medication/Additional surgical treatment

IV. Results

IV. 1. Clinical symptoms and 3D TOF MRA images

From among the 310 patients with facial pain who underwent 3 D TOF MRA, the result was positive in 179 cases (58%) and negative in 131 cases (42%). In the MRA-positive group, there were 146 typical TN cases, 32 TNWIP cases and 1 PIFP case. In the TN group (both typical TN and TNWIP), there were positive MRA results on the asymptomatic side in 30 cases (10.7%). The distribution of the MRA results with respect to the clinical symptoms revealed a significantly higher positive rate in the typical TN group than in the TNWIP group (68.2% vs. 49.2%, $p<0.01$). In the PIFP group, there was only 1 (3.2%) MRA-positive case.

There was a great difference between the proportions of the MRA-positive and negative cases, depending on which MR unit was used in the diagnostic procedure.

In the typical TN group, the rate of arterial compression was much higher (90.4%) than that in the TNWIP group (50%), whereas venous compression was seen more frequently in the TNWIP group (25%) than in the typical TN group (0.7%) ($p<0.001$).

IV. 2. Comparison of 3D TOF MRA images and intraoperative observations

Between January 1994 and December 2004, 112 patients underwent unilateral MVD and 2 patients underwent bilateral MVD.

In the 114 patients and 116 cases, we could always identify NC on MRA (which was mandatory for the MVD in this series). During surgery, NC was observed in 110 cases (95%), while in 6 cases (5%) we could find a loose contact, when an artery was running on the nerve, but there was no distortion of the nerve, or there was a visible gap between the nerve and the artery and the vessel touched the nerve periodically, only in the systolic phase. The MRA findings corresponded to the surgical observations. In 109 (94%) cases, the compression was arterial (99 pure arterial, and 10 mixed arterial + venous); only in 7 cases (6%) was pure venous compression found.

In the 6 patients operated on from the MRA-negative group for selective rhizotomy, no NC of the trigeminal nerves was observed during surgery.

As concerns the surgical findings, the rate of arterial NC was significantly higher in the typical TN group (91.8%) than in the TNWIP group (53%). Pure venous compression was found frequently (29.4%) in the TNWIP group, whereas it was rare (1%) in the typical TN group ($p<0.0001$).

IV. 3. Outcome after microvascular decompression

The median length of follow-up for the 114 patients (116 cases) was 40.3 months. One patient died from cardiac failure 3 weeks postoperatively; 7 other patients (6.1%) were lost to follow-up, 1 after 1 year, 4 after 2 years, and 2 after 4 years.

The immediate postoperative relief from tic and facial pain was complete in 91%, partial in 4% and absent in 4%. Six months after MVD, 89% gave excellent, 5% good and 5% poor results. One year after MVD, 82% gave excellent, 11% good and 7% poor results. Two years postoperatively, 74% gave an excellent, 18% a good and 8% a poor result. Four years after the procedure, 69% of the patients gave excellent, 23% good and 7% poor results.

Most of the recurrences, either severe or moderate, appeared within 2 years following MVD. In only 2 cases did the recurrent tic appear 4 and 6 years following MVD. Altogether 30 patients had some kind of recurrence. From among the 9 severe recurrent cases, 1 patient was treated with selective rhizotomy, 1 with stereotactic thalamotomy, and 3 with glycerol rhizotomy; the remaining recurrent cases resumed taking medication.

The distribution of the compressing vessels in the cases in which recurrence appeared is to be seen in *Table 2.*

Table 2

Compressing vessels in recurrent cases

Vessel (no.)	Severe recurrence (no./%)	Moderate recurrence (no./%)
Superior cerebellar artery (84)	4 (4.8)	16 (19)
Basilar artery (3)	1 (33)	2 (66)
Anterior inferior cerebellar artery (10)	1 (10)	-
Superior cerebellar artery+vein (10)	1 (10)	-
Vein (7)	2 (28.7)	2 (28.7)

While the rate of some kind of recurrence among the typical TN cases was only 21.4%, among the TNWIP cases it was 41.1% (approaching significance, p=0.054).

Four years after at the MVD, the rate of some kind of recurrence was 23.2% in typical TN group, while it was 60% in the atypical TN group.

IV. 4. Postoperative complications

Complications were analyzed as cranial nerve deficits or other surgical consequences. There was no mortality, posterior fossa hematoma or anesthesia dolorosa. Altogether 30 patients (26%) suffered some kind of complications, 3 of them 2 complications. The transient neurological deficits disappeared with time and the surgical complications could be treated; therefore for a long time only the permanent cranial nerve deficits (12%) caused problems for the patients. The most serious permanent complication was the keratitis due to the sensory loss of the first branch of the trigeminal nerve.

CSF leakage could be treated with lumbar CSF drainage in 5 cases, while in 1 case the wound had to be opened and duraplasty was performed. There were 2 wound infections, which needed a complete wound revision. Due to the retraction of the cerebellum, cerebellar edema and hydrocephalus developed in 1 case. This required the ventricular drainage of CSF.

V. Discussion

V. 1. The keyhole approach and positioning of patients for surgery

At the beginning of the learning process of the surgical technique, we used to perform a 10-12-cm long skin incision and a lateral suboccipital craniotomy 3-4-cm in diameter. When we had acquired practice in performing the MVD, the skin incision became 6-7-cm long and the craniotomy approximately 2 cm in diameter. The exact positioning of the craniotomy in the angle of the lateral and sigmoid sinus is crucial to perform the keyhole approach.

In this series, all 114 patients were operated on in either the prone, the park bench or (most of them) in supine position (introduced by P. Barzó). The main advantage of the supine position is the low risk of hypotensive circulatory problems and air embolism. A further advantage is that no retraction of the cerebellar lobe is necessary to visualize the intracisternal part of the fifth nerve, for it drops as a result of gravity. In patients positioned in the supine position, we had no complications related to the cerebellar retraction (cerebellar infarct, loss of hearing or facial palsy). It should also be mentioned that the compressed surface of the patient is large on the back, and the ventilation is not influenced by compression of the chest. This is important for elderly and corpulent patients.

V. 2. The relation between three facial pain groups (typical TN, TNWIP and PIFP) and the MRA findings. The value of MRA

In most classical TN cases, vascular compression of the trigeminal nerve is involved in the etiology, and MVD is a safe surgical method widely used throughout the world for the treatment of TN. However, there have been reports that NC can not always be found during surgical exploration. These negative findings varied from 0% to 89% with an average 7.5%; the rate in our initial series was 20%. Before considering a posterior fossa exploration for MVD, to prove the presence of NC of the trigeminal nerve is advisable. MRA investigations have demonstrated the usefulness of the preoperative evaluation of NC in TN cases.

In PIFP cases, the origin of the pain is unclear and the treatment is often not fruitful. We performed 3D TOF MRA in 31 patients with PIFP, and only 1 of them yielded a positive result. This was most probably only an accidentally positive NC because this patient showed no improvement after MVD. On the basis of these results, we can rule out NC in the background of PIFP.

In the TN groups, the MRA findings were positive in 68.2% in the typical TN group, and in 49.2% in the TNWIP group. Patients were selected for MVD only from among the MRA-positive cases (98 typical TN, and 17 TNWIP), and in all these cases NC was found during surgery. With regard to the good postoperative results after MVD in the literature and in the present series, NC appears to be an obvious etiological explanation of typical TN and TNWIP. However, it is noteworthy that, whereas typical TN was found to be caused by pure venous compression in only 0.7% on MRA and in 1% during surgery, TNWIP was caused by pure venous compression in 25% on MRA and in 29.4% during surgery. This might seem significant ($p<0.01$), but the small number of our cases permits only the suspicion that venous compression may cause TNWIP. On this basis, the different clinical symptoms (typical TN and TNWIP) seem to be dependent on the type of the compressing vessel.

It should be stressed that only patients who gave positive imaging results for NC were included in the MVD-treated group. It may be supposed that more cases from the MRA-negative group had NC, but the vessels were too small to be detected. The quality of the MR unit seems to be important in this process. In our series of patients with TN, there were 72 patients with negative MRA results on use of the 0.5-T machine. The relative data suggest that, of these cases, 31 would have been positive if the 1-T MR unit had been used. It means that approximately 31 patients were denied MVD due to “poor” MRA.

When the MRA investigations were performed with the 0.5-T machine, the positive ratio in the typical TN group was 60%; the corresponding value with the 1-T machine was 88%. This latter positive ratio approaches that for the surgically positive cases of Jannetta (39), and some cause other than NC (probably of central origin) in the background of typical TN can be suspected in only 12%. At this point, the question arises of whether it is worth performing MRA in patients with typical TN when the expected negative ratio is only 12%. The answer may be ″no″ if we consider selective partial rhizotomy as an alternative to MVD, while it may be ″yes″ if we consider other surgical treatment.

In the TNWIP group, the positive MRA ratio was 25% with the 0.5-T machine, and 57% with the 1-T machine. In this clinical group, preoperative MRA investigation seems to be indicated unequivocally. The previous published MRA studies were performed on 1.5-T MR machines. Even, with such technically superior equipments, there were false-negative cases, which indicates that small vessels and thickened arachnoids can probably go undetected on MRA. Unfortunately, there are no data regarding the type of TN in these cases.

The 10.4% NC on the asymptomatic side suggests that the clinical symptoms and MRA results must be evaluated together. If we consider the same proportion of asymptomatic NC on the symptomatic side, this may explain the unsuccessful MVDs, where causes other than NC were to be found in the background of TN.

There were 6 operative cases where only loose contact could be observed during surgery; however, an NC was obvious on MRA. The position of the head and the discharge of CSF during surgery may modify the relationship of the nerve and vessel. All 6 patients had typical TN, and 5 of them exhibited an excellent and 1 of them a good outcome.

V. 3. Correlation between clinical symptoms, type of vascular compression and outcome of MVD

Many factors which may contribute to the success of MVD have been discussed in the literature, in connection with large series of TN patients. This study focused on the clinical symptoms and type of vascular compression. Szapiro et al. consider the mode of pain manifestation of TN to be the most significant prognostic factor in the outcome of MVD. They found that 95% of the typical TN group and 58% of the atypical TN group could be cured. Tyler-Karaba et al.found these numbers to be 73% and 35% respectively. In this study, 78.6% of the typical TN group were considered cured, while 16.3% had a moderate and 5.1% a severe recurrence; in the TNWIP group, 58.9% were cured, while 23.5% had a moderate and 17.6% a severe recurrence. Four years after the MVD 76.8% of the typical TN patients seemed to be cured, while 19.6% had a moderate and 3.6% severe recurrence, while in the TNWIP only 40% was cured, 30% had moderate and severe recurrence. With respect to these results, we may say that the clinical appearance of the TN is an important prognostic factor in the outcome of MVD.

With regard to the special vessels and the number of all recurrences, it could be seen that, in the cases where the basilar artery and veins were involved in the NC, the probability of recurrence was higher (Table 3). The reason for the recurrences in the basilar artery-caused NC cases might be the stiffness of the artery, which could therefore be mobilized away from the nerve only with difficulty. Since we used gelatin foam between the nerve and vessel, the stiffness of the vessel might compress the gelatin foam and the NC could be newly formed.

The reason for the recurrences in cases involving the veins might be different. In most cases, the veins were divided and no chance of NC remained following MVD. In spite of this, venous compression predicts a higher rate of recurrence of TN. Since veins can be distinguished from arteries on MRA, the positive MRA result may play a predicting role in the prognosis following MVD.

V. 4. Complications following MVD

A literature review reveals a mortality rate of 0.5%, the risk of posterior fossa hematoma or infarction being 0.3% and 0.1% respectively. In this series, there were no such complications. The occurrence of other types of complications is significantly higher than in the literature. One of the reasons for this might be that the series in the literature contains hundreds of patient operated on by one surgeon. It is not surprising that, with increasing experience, the complication rate falls. In this series, patients were operated on by 6 surgeons, only 3 with appropriate experience. The deficits of cranial nerves are the consequences of either the traumatization of the nerves during the separation from the vessel or the excessive retraction of the cerebellum. With a careful surgical technique, almost all the complications can be avoided. The possibility of intraoperative monitoring of brainstem-evoked response was not available, but, since the keyhole approach has been used with the supine positioning of the patient, there have been no 7th or 8th nerve deficits.

V. 5. Questions to be answered in the future

We may merely hypothesize the potential different effects of arterial versus venous compression and the possible influence on the outcome of MVD. Arterial compression has two components: the constant compression due to the anatomical position of the vessel and the strong rhythmic pulsation force on the nerve. Venous compression has only a constant "positional" compressing force, but no strong pulsation. Both types of compression can cause focal demyelination, but there might be some difference between them. This pathological difference might be subtle, because both arterial and venous compression can cause typical TN and TNWIP, whereas arterial compression causes mainly typical TN, while in TNWIP venous compression can frequently be found. An animal model or intraoperative biopsy taken from the affected nerve or sample from the trigeminal nerve of cadavers who had previously had TN may settle this hypothesis.

VI. Conclusions (according to the aims of this study)

1. The surgical technique described by Jannetta could be learned and performed. The keyhole approach together with the supine positioning of the patient provides a safe approach to the trigeminal nerve in the pontocerebellar cistern.
2. MRA findings were different in the three facial pain groups:
    - PIFP is not related to NC of the trigeminal nerve.
    - NC can be identified on MRA on a 1-T MR device in 88% of the typical TN cases and 57% of the TNWIP cases.
    - TNWIP is caused by venous compression at a significantly higher rate than that for typical TN.
3. MRA can be involved in the decision-making when considering MVD.

- The type of MR unit significantly determines the accuracy of the MRA result and the percentage of MRA-positive cases. At least a 1-T MR unit is needed for the investigations.
- Arterial and venous compression can be differentiated.
- MRA-negative cases, and especially TNWIP cases, can be considered for treatment other than MVD.

4. The clinical symptoms, the type of NC and the outcome of MVD are related:
    - Typical TN cases can be cured by MVD at a higher rate (~80%) than can TNWIP cases (~60%). This may be the most important prognostic factor in MVD.
    - The type of NC may play a role in the recurrence of TN following MVD.
    - The presence of venous compression is a sign of a poor prognosis.
    - In TNWIP, venous compression is more frequent than in typical TN.
5. The keyhole technique and supine position of the patient for MVD may prevent edema cerebellar edema and cranial nerve deficit complications; the experience of the surgeon plays a significant role in the prevention of complications.

VII. New results in this study

1. The author has reported the first series of TN cases treated with MVD in Hungary.
2. The largest number of patients with TN and PIFP were investigated with three dimensional MRA. The findings were evaluated according to the clinical symptoms.
3. The prognostic value of the MRA regarding to the post MVD result was determined by comparison of MRA and the surgical findings.
4. PIFP is ruled out as a NC of the trigeminal nerve.
5. In TNWIP, the venous compression is more frequent than in typical TN.
6. At least a 1-T MR unit is necessary for the investigations to reveal NC properly.

Acknowledgments

I wish to express my sincere gratitude and appreciation to:

Professor Mihály Bodosi, former director of the Department of Neurosurgery, for his generous support and encouragement during the entire preparation of this thesis.

Professor Pál Barzó, director of the Department of Neurosurgery, for his cooperation and critical correction of most of the manuscripts.

Associate Professor Erika Vörös, for her help and cooperation in this work.

Professor Tamás Dóczi, director of the Department of Neurosurgery in Pécs, for his stimulating support and critical remarks.

Dr. Mátyás Bobest, former chief of the Department of Neurosurgery in Szombathely, for drawing my attention to MVD in TN.

My colleagues in the Department of Neurosurgery who gave their cases to the study.

My colleagues in the Department of Neurology and International Medical Center who referred and investigated the patients and took part in the study.

Dr. Miklós Árgyelán for assistance with statistical analysis.

The doctors and nurses of Anesthesiology and Intensive Care Unit, Department of Neurosurgery, and the staff in the operating theater for their untiring help and their care of the patients.

My family for their support and patience.

Original papers related to this thesis:

1. Vörös E, Szentgyörgyi R, **Kuncz Á**, Mencser Z, Kardos L, Milassin P:
A háromdimenziós "time of flight" mágneses rezonanciás angiographia trigeminus neuralgiában
Clin Neurosci/Ideggy Szle 50: 154-160, 1997

2. **Kuncz Á,** Mencser Z, Vörös E, Bodosi M:
Trigeminus neuralgia kezelése mikrovaszkuláris dekompresszióval
Orv Hetil 138: 34, 2051-2055, 1997

3. Vörös E, Palkó A, Horváth K, Barzó P, Kardos L, **Kuncz Á**:
Three-dimensional time-of-flight MR angiography in trigeminal neuralgia on a 0.5-T system
Eur Radiol 11: 642-647, 2001 (I.f.: 1, 321)

4. **Kuncz Á,** Vörös E, Barzó P, Tajti J, Milassin P, Bodosi M
Az MR-angiographia szerepe a mikrovaszkuláris dekompresszióval kezelt trigeminus neuralgias betegek műtéti eredményének prognosztizálásában.
Orv Hetil 146:2595-2602, 2005

5. **Kuncz A,** Vörös E, Barzó P, Tajti J, Milassin P, Mucsi Z, Elek P, Benedek K, Tarjányi J, Bodosi M
Comparison of clinical symptoms, MR-angiographic results in patients with trigeminal neuralgia and persistent idiopathic facial pain. Medium-term outcome after microvascular decompression of cases with positive MR-angiographic findings
Cephalalgia 26: 266-276, 2005 (I.f.: 3,133)

Publications related to this thesis:

1. **Kuncz Á,** Bodosi M**:**
A trigeminus neuralgia sebészi kezelése
Clin Neurosci/ Ideggy Szle 50: 358-359, 1997

2. **Kuncz Á**, Vörös E, Bodosi M:
Trigeminus neuralgia kezelése mikrovaszkuláris dekompresszioval
MOTESZ Magazin 8-9: 6-13, 1999

3. **Kuncz Á,** Vörös E, Bodosi M, Barzó P, Tajti J:
The role of MR-angiography in patient selection for microvascular decompression in the treatment of trigeminal neuralgia
In: Proceedings 12th World Congress of Neurosurgery
Editors: Glenn McCullogh and Peter Reilly
Sydney, Australia 754-756, 2001

4. Mencser Z, **Kuncz Á,** Vörös E:
Microvascularis dekompresszió a trigeminus neuralgiában /
Clin Neurosci/Ideggyógy Szle 48, 320-343, 1995.(abstract)

5. Vörös E., Kardos L., Milassin P., **Kuncz Á**., Mencser Z.:
Three-dimensional time-of-flight MR-angiography (3D TOF MRA) in patients in trigeminal neuralgia
Neuroradiology, 37, Suppl. 1. S.73., 1995. (abstract)

6. **Kuncz Á,** Szok D, Vörös E, Barzó P, Benedek K, Vécsei L, Bodosi M:
A klasszikus trigeminus neuralgia típusos és atípusos (interparoxizmális fájdalommal járó) változatának gyógyulási hajlama mikrovaszkuláris dekompressziót követően. A vénás kompresszió jelentősége
Cephalalgia Hungarica No.13, 12. 2005. (abstract)

Presentationts related to this thesis:

1. Mencser Z., **Kuncz Á.,** Vörös E.:
Microvascularis dekompresszió a trigeminus neuralgiában
Fiatal Neurológusok Fóruma, Szombathely, 1995

2. **Kuncz Á,** Mencser Z, Vörös E, Bodosi M:
Microvascular decompression in the treatment of trigeminal neuralgia
International Congress of Slovak Neurosurgical Society
Banská Bystrica-Tále, Slovakia, 1996

3. Vörös E, Kardos L, Milassin P, **Kuncz Á,** Mencser Z:
Three-dimensional time-of-flight MR-angiography (3 TOF MRA) in patients with trigeminal neuralgia
XXIst Congress and 5th Advanced Course of the European Society of Neuroradiology, Budapest, 1995

4. **Kuncz Á,** Bodosi M:
A trigeminus neuralgia sebészi kezelése
SZOTE Neurologiai Kerekasztal 1997

5. **Kuncz Á,** Mencser Z, Bodosi M:
Complications of the microvascular decompression
XIVth Congress of the Hungarian Neurosurgical Society, Debrecen, 1998

6. **Kuncz Á,** Vörös E, Bodosi M, Hakanson S:
Microvascular decompression in the treatment of trigeminal neuralgia. The role of MR –angiography in patients` selection
The Emirates Neuroscience Conference, Dubai, 2001

7. **Kuncz Á,** Vörös E, Bodosi M, Barzó P, Tajti J:
The role of MR –angiography in patients` selection for microvascular decompression in the treatment of trigeminal neuralgia
12th World Congress of Neurosurgery, Sydney, 2001

8. **Kuncz Á,** Vörös E, Bodosi M, Barzó P, Tajti J:
MR-angiography based selection of patients with trigeminal neuralgia and facial pain for microvascular decompression
12th European Congress of Neurosurgery, Lisboa, 2003

9. **Kuncz Á,** Vörös E, Bodosi M, Barzó P, Tajti J:
Trigeminus neuralgia és arcfájdalom esetén végzett MR-angiographia szerepe a mikrovaszkuláris dekompresszió indikációjában
Magyar Neuroradiologiai Társaság XII. Kongresszusa és nemzetközi továbbképző tanfolyama, Dobogókő, 2003

10. **Kuncz Á,** Szok D, Vörös E, Barzó P, Benedek K, Vécsei L, Bodosi M:
A klasszikus trigeminus neuralgia típusos és atípusos (interparoxizmális fájdalommal járó) változatának gyógyulási hajlama mikrovaszkuláris dekompressziót követően. A vénás kompresszió jelentősége
Magyar Fejfájás Társaság XII. Kongresszus, Siófok, 2005