

# FIVE-YEAR RESULT OF MICROVASCULAR DECOMPRESSION USING VIDEO ENDOSCOPY IN THE TREATMENT OF CLASSIC TRIGEMINAL NEURALGIA WITH PAROXYSMAL PAIN SYNDROME

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**Background.** The incidence of trigeminal neuralgia (TN) is 15 per 100,000 people per year. The effectiveness of the existing conservative methods of therapy does not exceed 50%. At the same time, the use of carbamazepine doubles the frequency of depressive conditions, and by 40% increases the incidence of suicidal thoughts. Microvascular decompression (MVD) of the trigeminal root is a "gold standard" treatment for patients with facial pain, however, due to the lack of awareness of the disease, not all the patients receive the adequate therapy timely. **Aims:** to evaluate the long-term results of video endoscopy-assisted microvascular decompression in the treatment of patients with classical trigeminal neuralgia (cNTN) with paroxysmal facial pain. **Methods.** In the period from 2014 to 2019, 62 patients were operated for classic NTN and paroxysmal facial pain. The average period from the onset of pain syndrome to surgery was 5 years (from 2 months to 15 years). All the patients in the preoperative period underwent conservative therapy (carbamazepine, gabapentin, pregabalin), which was not accompanied by significant pain reduction. Two (3%) patients had previously undergone a radiosurgical treatment using the Gamma Knife device, and 7 (11%) patients had an analgesic blockade without an effect at other hospitals. The maximum pain intensity upon the admission to the hospital, according to the visual analogue scale (VAS,) was 10 points, according to the BNI (Barrow Neurological Institute) pain syndrome scale — V. All the patients underwent MVD of the trigeminal nerve root using Teflon, and video endoscopic assistance during surgery was used in 9 patients. The average follow-up period after the surgery was  $3.4 \pm 1.7$  years (from 1 to 5 years). **Results.** In all (100%) the patients, the pain was completely relieved after the surgery (BNI I). Excellent and good results after MVD within 5 years were achieved in 97% of patients (BNI I–II). Facial hypesthesia, not causing discomfort and anxiety (BNI II), developed in 5 (8.1%) patients. The use of video endoscopy made it possible to identify the vessels compressing the trigeminal nerve root with a minimal traction of the cerebellum and cranial nerves. The development of cerebellar edema and ischemia occurred in one (1.6%) patient operated without the application of video endoscopy. **Conclusion.** The MVD method with video endoscopy is effective in the treatment of patients with cNTN with paroxysmal pain syndrome.

**Keywords:** trigeminal neuralgia, microvascular decompression, trigeminal nerve, anterior cerebellar artery, video endoscopy.

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## BACKGROUND

Trigeminal neuralgia (TN) is a disorder characterized by sudden and recurring unilateral short-term pains of the "electric shock" type, spreading along the innervation zones of the three branches of the trigeminal nerve. TN due to neurovascular conflict occurs in 3–15 people per 100,000 population per year, women suffer 3 times more often than men. TN can develop at any age but it is most typical for the age period from 50 to 70 years [1–3].

TN is divided into classic pain with paroxysmal and persistent facial pain (previously classified as atypical

and type 2 neuralgia), secondary and idiopathic. The reason for the development of classical TN is compression of the trigeminal nerve root by vessels (superior cerebellar, anterior inferior cerebellar arteries and superior petrosal vein) in the cerebellar cisterna. Secondary neuralgia occurs with pathology in the posterior cranial fossa (tumors, aneurysms, arteriovenous malformations, multiple sclerosis). The condition of 10% of patients in whom it is not possible to determine the cause of facial pain is referred to as idiopathic TN [4].

Pain associated with TN significantly reduces the quality of life. Sleep disorders develop, mental, depres-

sive disorders arise, up to 34% of patients with TN cannot actively engage in labor activity [5].

The drugs of choice for the treatment of TN are carbamazepine and oxcarbazepine. However, according to G. Di Stefano et al. [6], in more than 23% of cases, drugs are discontinued due to the development of adverse reactions in patients. The lack of effect on the introduction of carbamazepine is observed in 5% of patients. More than 50% of patients note a decrease in the effectiveness of drugs within 5 years of admission [6]. With prolonged pain syndrome, secondary changes develop in the thalamus and cerebral cortex, which reduces the effectiveness of conservative therapy [7, 8]. In 50% of patients depressive states are observed, in 40% — the presence of suicidal thoughts, the frequency of which significantly increases while taking anticonvulsants (carbamazepine, finlepsin), especially in patients under 25 years of age [5, 9, 10].

In the Joint Guidelines of the American Academy of Neurology and the European Federation of Neurological Societies, the surgery of microvascular decompression (MVD) is defined as the “gold standard” of surgical treatment of NTN [11]. However, difficulties in making the correct clinical diagnosis and insufficient awareness of patients and doctors in Russia about this method lead to the fact that many patients are treated for a long time and to no avail by dentists, otolaryngologists, and neurologists [12]. Meanwhile, timely and correct treatment is the key to the success of therapy for patients with classical TN.

**The aim** of the study was to evaluate the results of microvascular decompression using video endoscopy in patients with classic trigeminal neuralgia with paroxysmal facial pain.

## METHODS

### Study design

An open-label retrospective clinical study was performed.

### Eligibility criteria

#### Inclusion criteria:

- consent to participate in the research;
- age over 18;
- the presence of pharmacoresistant pain in the face according to VAS 10 points, according to the BNI scale — V;
- confirmed neurovascular conflict according to magnetic resonance imaging (MRI) in FIESTA or 3D mode — CISS or DRIVE;
- terms after surgery from 1 to 5 years.

#### Exclusion criteria:

- refusal to participate in the study;
- age under 18;
- terms after surgery less than 1 year.

### Conditions of conduction

The study was carried out in the period from 2014 to 2019 in the neurosurgical department of the Federal Scientific and Clinical Center of the FMBA of Russia.

### Description of medical intervention

Sixty-two patients with classic TN with paroxysmal facial pain were operated on.

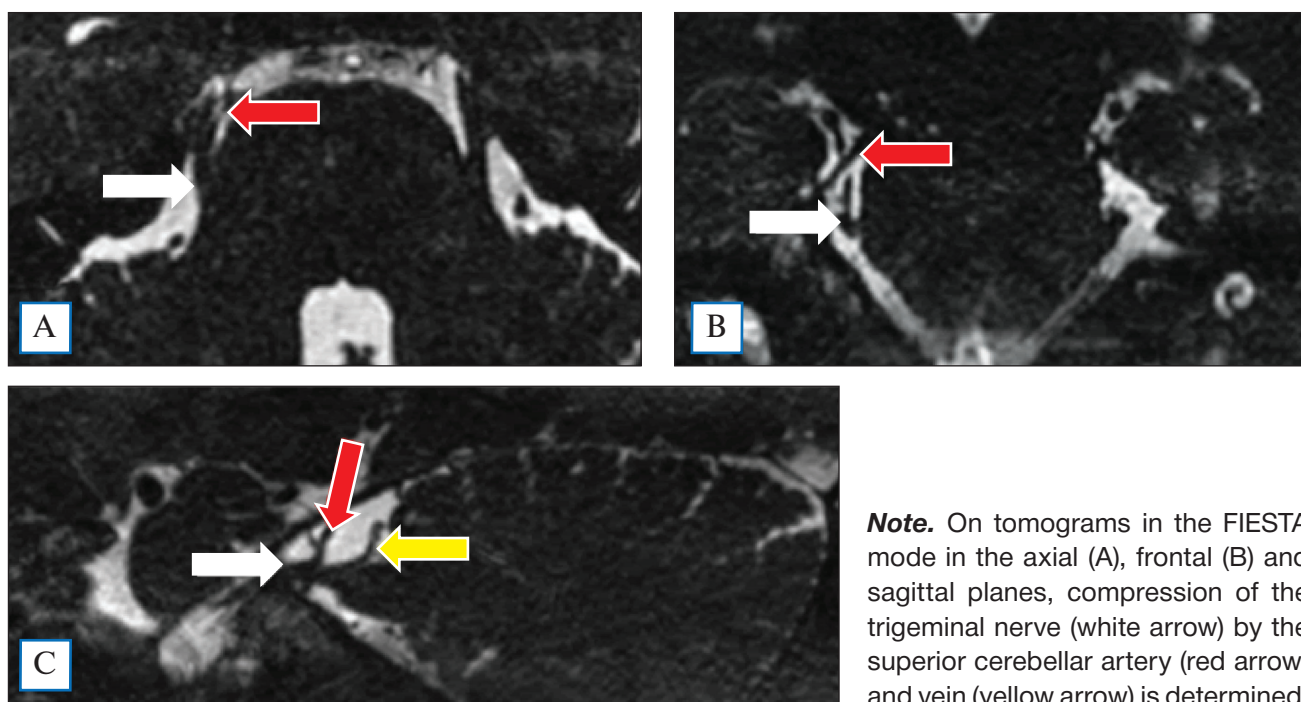
After clinical examination, the presence of neurovascular conflict was confirmed by MRI of the brain using a 1.5–3 Tesla apparatus in 3D-CISS or FIESTA or DRIVE mode. In all cases, a neurovascular conflict was detected between the superior cerebellar artery and/or vein and the trigeminal nerve root; in 35 (56.5%) patients, a decrease in the cross-sectional area of the nerve was noted (Fig. 1). Other neurosurgical pathology is excluded.

The surgery was performed with the patient in the prone position with the head turned towards the surgery by 15–20°. Retrosigmoid access was used; at the intracranial stage, 9 (14.5%) patients used a microscope and video endoscopy from Karl Storz with a diameter of 4 mm and a viewing angle of 30° and 70°. In 51 (82.3%) cases, osteoplastic craniotomy was performed, in the rest — resection. Video endoscopy made it possible to visualize the exit zone of the trigeminal nerve root and compressing vessels on the ventral surface of the trigeminal nerve root. After a neurovascular conflict was detected, the vessels were dissected from the trigeminal nerve root, followed by the installation of a Teflon pad between them (Fig. 2).

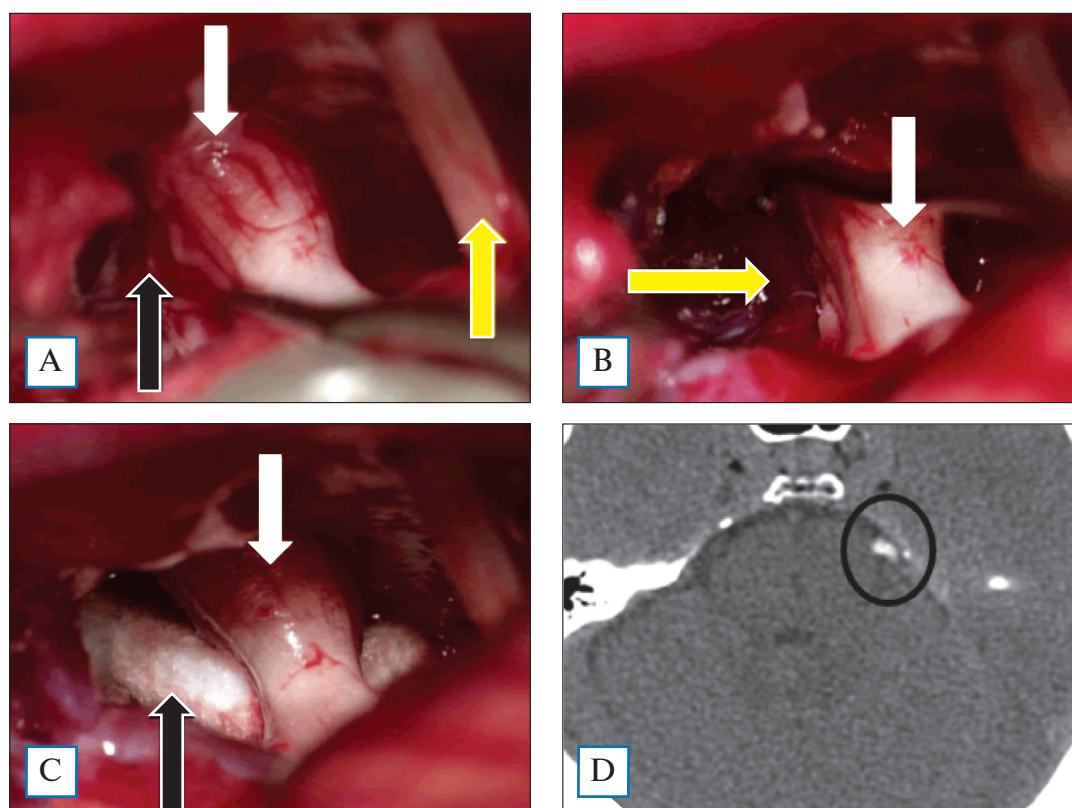
### Methods of outcome registration

The result of surgical treatment was assessed 1–5 years after the operation. To assess the pain syndrome, a 10-point visual analogue scale (VAS) was used, where 0 is no pain, and 10 is unbearable pain, to assess the severity of pain, the BNI scale (Barrow Neurological Institute), where I is no pain, and V — severe persistent pain. Sensory disturbances were noted on the BNI scale of facial numbness, where I is no numbness, and V is pronounced numbness, which is very disturbing to the patient. These scales are generally accepted and also fully reflect the clinical outcomes of treatment.

**Fig. 1.** Patient M. with trigeminal neuralgia: magnetic resonance imaging of the brain in FIESTA mode



**Fig. 2.** Patient M. with trigeminal neuralgia: stages of microvascular decompression and postoperative computed tomography images



**Note.** Intraoperative images: A — the root of the trigeminal nerve (white arrow), pronounced adhesive process (black arrow), facial nerve (yellow arrow); B — the root of the trigeminal nerve (white arrow), superior cerebellar artery (yellow arrow); C — the root of the trigeminal nerve (white arrow), Teflon gasket (black arrow); D — postoperative CT scans showing a Teflon pad in the projection of the trigeminal nerve root (circled).



### Ethical review

Treatment was performed according to the clinical guidelines for surgical correction of vascular compression syndromes of the cranial nerves, approved at the Plenum of the Board of the Association of Neurosurgeons of Russia (Kazan, 2014). All patients signed voluntary informed consent to participate in the clinical study.

### Statistical analysis

Statistical processing was performed using the Statistica 12.0 software (StatSoft Russia).

## RESULTS

### Subjects (participants) of the study

Sixty-two patients with classic NTN with paroxysmal facial pain were operated on, of which 18 were men and 44 were women. The age of the patients is from 31 to 73 (on average  $55 \pm 11.3$ ) years. The average period from the onset of pain syndrome to surgical treatment was  $5 \pm 3.2$  years (from 2 months to 15 years). Despite the conservative therapy (carbamazepine, gabapentin, pregabalin) carried out in the preoperative period, the maximum pain intensity upon admission to the hospital according to the VAS was 10 points, according to the BNI-V pain syndrome scale. In 2 (3.2%) patients, radiosurgical treatment using the "Gamma Knife" installation without significant effect. In 7 (11.3%) patients in other hospitals, blockades of the branches of the trigeminal nerve with analgesics were performed, against the background of which the intensity of pain decreased by more than 2 times, but the effect persisted for no more than 3 months, after which the intensity of the pain syndrome increased to the level that preceded blockade, and in 3 (42%) of 7 patients, hypesthesia developed additionally.

In all patients, pain was provoked by such actions as eating, talking, swallowing, washing, smoking, brushing teeth. When making a diagnosis, we were guided

by the recommendations of the International Headache Association<sup>1</sup>. In 41 (66.1%) patients, pain was felt on the right side. In 59 (95%) patients, pain localization was in the projection of the second and third branches of the trigeminal nerve, and in 3 (4.8%) patients only in the projection of the third branch. In 29 (46.8%) patients, the development of the disease was preceded by dental manipulations (tooth extraction, treatment; implant placement).

### Key study findings

The average follow-up period for patients after surgery was  $3.4 \pm 1.7$  years. Of 62 patients, 32 (52%) had a maximum follow-up period after surgery for 3 years, and in 20 (48%) — 5 years. After surgery, pain completely regressed in all patients: on the VAS scale — 0 points, on the BNI scale — I. 97% of patients (Table 1).

One year after the operation, all operated patients (62; 100%) had no pain. After 3 years, an excellent and good outcome of treatment (BNI I – II) was noted in 31 (96.9%) of 32 cases, in 1 (3.2%) patient after 2 years 6 months, pain reappeared (BNI IV). 5 years after the operation, an excellent result of treatment (BNI — I) after MVD was noted in 19 (95.2%) patients, in 1 (5%) — BNI — IV (after 4 years, facial pain appeared). 2 patients were sent for radiosurgical treatment (3 and 4 years after MVD) with the result of BNI IV treatment.

## DISCUSSION

The first mention of the pain syndrome characteristic of TN is described in the book of Arteus from Cappadokia "Cephalaea" [13]. A detailed clinical picture characteristic of TN was described by Avicenna in the 1000th year. The author noted the patient's complaints of "stitching", "tearing" and "stinging" pain in the face [14]. A similar observation was later presented by M. Fehr and E. Schmidt in 1688. In 1756, N. André described TN with tonic contractions of the facial muscles using the term "tic doloieux" [13].

<sup>1</sup> Access mode: <https://ihs-headache.org/ru/%D1%80%D0%B5%D1%81%D1%83%D1%80%D1%81%D1%8B/%D1%80%D0%B5%D0%BA%D0%BE%D0%BC%D0%B5%D0%BD%D0%B4%D0%B0%D1%86%D0%B8%D0%B8/>.

Table 1

### The result of microvascular decompression in patients with classical trigeminal neuralgia according to the BNI pain syndrome severity scale

Time after surgery, years	Treatment outcome according to BNI				
	I	II	III	IV	V
1	62 (100%)	0	0	0	0
3	30 (93,8%)	1 (3,1%)	0	1 (3,1%)	0
5	19 (95%)	0	0	1 (5%)	0

In 1748, Schlichtung first cut the infraorbital nerve in a patient with “facial neuralgia” [15]. Prior to the studies of F. Magendie and C. Bell in 1821, it was believed that the facial nerve is responsible for the movements of facial muscles and sensitivity on the face, and the trigeminal nerve is responsible for taste [16, 17].

In 1925 W. Dandy put forward the hypothesis of vascular compression as the main cause of TN [15], but this theory received support only after the 70s of the XX century. [eighteen]. For the treatment of patients with TN, W. Dandy developed a retrosigmoid approach and in 1932 presented a successful series of treatment for 250 patients with this pathology, 40% of whom had vascular compression of the nerve. During the operation, the surgeon dissected the sensitive portion of the trigeminal nerve while preserving the motor one [19].

The active use of P. Jannett’s microscope with the subsequent publication in 1967 of a series of successful MVDs contributed to the development of interest in the problem of surgical treatment of TN.

According to the recommendations of the International Headache Association [4], the diagnosis of classic trigeminal neuralgia is made in the presence of such criteria as:

- recurrent paroxysms of unilateral facial pain in one or more dermatomes of the trigeminal nerve without spreading beyond them and meeting the following criteria:
- a) pain has all of the following characteristics:
  - the duration of the pain from a split second to two minutes;
  - high intensity of pain;
  - the pain is sudden, sharp, shooting, stabbing like an «electric discharge»;
- b) the development of pain occurs against the background of trigger factors (washing, shaving, smoking, talking, brushing teeth) and various effects on trigger zones;
- pain is not associated with other diseases (tumors, arteriovenous malformations, aneurysms, multiple sclerosis);
- MRI or during surgery reveals the presence of a neurovascular conflict with morphological changes in the root of the trigeminal nerve (atrophy or displacement).

The development of TN occurs in several stages. At the first stage, as a result of constant vascular compression of the nerve, edema of the myelin sheath and thinning of the axial cylinders occur. The development of edema is accompanied by compression of intraneural vessels, which leads to ischemia of the root and its

demyelination. When the myelin sheath is destroyed, the conduction of the nerve impulse is disrupted, which is expressed by the spread of excitation from one axon to another, thereby causing the development of excitation in the “short circuit” type. Second, myelin-free areas of the nerve root are generators of ectopic excitation, which is clinically manifested by the development of a pain attack [20]. With prolonged pain syndrome, secondary changes develop in the thalamus and cerebral cortex, which reduces the effectiveness of treatment [7].

For patients with classical TN with paroxysmal facial pain, its absence during the interictal period is characteristic. This phenomenon is explained by hyperdepolarization of the sensitive portion of the trigeminal nerve root [21].

According to a number of studies, the 2nd and 3rd branches of the trigeminal nerve on the right are most often affected, and only in 5% of cases pain is localized in the area of the 1st branch of the trigeminal nerve. According to the authors, the age and duration of the disease do not affect the frequency of pain attacks, its intensity and morphological changes in the trigeminal nerve detected by MRI [22, 23].

In our study, all patients had unilateral facial pain, which in 41 (66%) cases was localized in the right half. In 59 (95%) observed pains spread along the 2nd and 3rd branches of the trigeminal nerve.

The technique of using Teflon as a spacer between the root of the trigeminal nerve and the vessels in the MVD of the trigeminal nerve was first proposed by P. Jannett, who in 1985 published a series of successful treatment of 51 patients with TN [24]. In our study, all patients with MVD underwent a phased dissection of the trigeminal nerve and the installation of a Teflon pad.

E. Pressman et al. [25] conducted a retrospective analysis of the results of the MVD of 4273 patients with NTI. The authors found that the incidence of complications such as meningitis, postoperative wound infection, liquorrhea, and cranial nerve damage reaches 12% [25]. According to H. Capelle et al. [26], the development of granulomas was noted in 1.5% of cases. The recurrence of pain syndrome varies from 1 to 5% per year [27].

In our series of observations of 62 patients, only 1 (1.6%) case developed edema and cerebellar ischemia on the first day after surgery, which required urgent surgical treatment in the form of resection of nonviable tissues and the installation of external ventricular drainage for 96 hours. the patient was discharged in satisfactory condition on the 10th day after the second

operation. There were no pains in the face and paresis in the limbs. Dizziness and sensory disturbances in the branches of the trigeminal nerve persisted, which completely regressed after 6 months. We associate the development of ischemia due to venous cerebellar infarction with the intersection of one of the veins flowing into the petrosal sinus, as well as with excessive traction of the cerebellum with retractors when visualizing neurovascular conflict due to the small size of the posterior cranial fossa and cerebellar rigidity.

The development of hypesthesia and a decrease in the corneal reflex on the side of the operation were noted by us in 5 (8%) patients on the BNI II scale of facial numbness (slight numbness that does not cause discomfort and anxiety).

During the MVD, it is necessary to stage-by-stage dissection of the trigeminal nerve, its thorough examination in order to detect compressing vessels, especially on its ventral (anterior) surface. According to the observations of J. Zhong et al. [28], incomplete decompression of the nerve root due to the “skipping” of the compressing vessel was the main cause of persistent pain in the postoperative period in 5% of patients.

In 9 patients during the operation, we used video endoscopy, with the help of which we were able to visualize the vascular relationship in the region of the anterior pons and on the anterior surface of the trigeminal nerve root with minimal traction of the cerebellum. The use of endoscopy with angles of 30° and 70° made it possible to detect neurovascular conflict in places inaccessible for viewing by a microscope: in 2 patients the cause of the conflict was a vein, in 5 — the superior cerebellar artery, in 2 more — a combination of a vein and an artery. J. Zhong et al. [28] with 1274 participants in the study, the cause of compression of the trigeminal nerve root in 74% of cases was several vessels, in 41% — the superior cerebellar artery, in 29% — the anterior inferior cerebellar artery, in 35% — the petrosal vein, in 9% — the posterior the lower cerebellar artery, in 9% — the vertebral artery. Compression of the nerve at the entrance to the bridge was observed in 55% of patients, in the middle part — in 36%, at the entrance to the Meckel cavity — in 9% [28].

In our study, the main causes of TN were a conflict between an artery and a nerve (in 34; 55%), a vein (in 7; 11%), a combination of venous and arterial compression (in 21; 34%). During arterial compression, NT was associated with the superior cerebellar and anterior inferior cerebellar artery in 32 (94%) 2 (6%) patients, respectively. In 5 (8%) patients, one of the superior petro-

sal veins (sometimes one or more) passed through the root of the trigeminal nerve, which required its coagulation and transection. In 2 (3%) patients, a branch of the superior cerebellar artery passed through the trigeminal nerve root, which was coagulated and transected, with no complications in the postoperative period. In 3 (5%) patients, in whom the superior cerebellar artery passed between the sensory and motor portions of the trigeminal nerve root, the sensitive portion of the root was dissected for decompression. In the postoperative period, all three developed hypesthesia on the side of the operation according to the BNI II numbness scale with a decrease in the corneal reflex.

According to our data, nerve penetration by a vein or artery was observed in 10 (13%) patients. In 35 (56%) cases, compression was in the projection of the exit of the trigeminal nerve root from the bridge, in 13 (21%) — in the middle part of the nerve, in 3 (5%) — in the place where the root entered the Meckel cavity. In 11 (18%) patients, there was a combination of the compression zone (in the projection of the root exit from the bridge and in its middle part). During the operation, in 49 (79%) cases, the presence of a pronounced adhesive process in the subarachnoid space drew attention.

## CONCLUSION

MVD is an effective method of treating patients with classical TN with paroxysmal facial pain. One year after surgery, excellent and good treatment results were achieved in 100% ( $n = 62$ ) patients, after 3 years in 97% (31 out of 32 patients), and after five years in 95% (19 out of 20 patients). The incidence of facial hypesthesia (according to the BNI II scale — not causing discomfort and anxiety) in the postoperative period was 8% ( $n = 5$ ). The use of video endoscopy made it possible to reveal a neurovascular conflict in the zone of the exit of the trigeminal nerve root from the pons with minimal traction of the cerebellum. Cerebellar ischemia developed in 1 (1.6%) patient and was associated with coagulation and transection of the vein compressing the root, as well as excessive traction of the cerebellum.

Thus, 5 years after the operation, facial pain may develop in 5% ( $n = 1$ ) of patients; to find out the cause of this phenomenon, it is necessary to continue research on a larger sample of patients.

## INFORMED CONSENT

All patients voluntarily signed an informed consent for the publication of personal medical information in an impersonal form in the journal “Clinical Practice”.

## ADDITIONAL INFORMATION

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**Competing interests.** The authors declare no conflict of interest which should be reported.

## AUTHOR CONTRIBUTIONS

All authors made an equal contribution to the design and preparation of the article manuscript, read and approved the final version before publication.

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