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Chapter 57

Surgical treatment of cranial neuralgias

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TRIGEMINAL NEURALGIA

Introduction

Trigeminal neuralgia (TN) is a relatively uncommon disease, whose incidence is estimated to be about 5/100 000 individuals per year. It is characterized by attacks of recurring, paroxysmal, shock-like pain within the distribution of one or more branches of the trigeminal nerve. Light tactile stimuli, as well as sudden temperature changes perceived at the hemiface, vocalization, chewing, or teeth-brushing may trigger such attacks. Another feature helping to make the diagnosis of TN is the absence of a significant loss of facial sensation in the cutaneous regions where pain is referred. Even if new drugs have recently been introduced in the treatment of TN (Tew and Keller, 1977; Farago, 1987; Fromm and Terrence, 1987; Lindstrom and Lindblom, 1987; Lechin et al., 1988), about half of all patients eventually require surgery for pain relief. The most common drugs used in such cases are carbamazepine, phenytoin, oxcarbazepine, clonazepam, and gabapentin. Drug resistance or drug intolerance can be commonly observed in patients with a long history of disease.

Since the first description by Fothergill in 1773, from which the clinical features of TN are nowadays well known, many different pharmacological and surgical treatment modalities have been applied. Most of the invasive ones, such as gasseriectomy (Rose, 1890), retrogasserian neurotomy (Spiller and Frazier, 1901), juxtaprotuberantial neurotomy (Dandy, 1929), trigeminal tractotomy (Sjoqvist, 1937), temporal intradural decompression (Taarnhoj, 1952, 1982), gasserian ganglion alchoholization (Taptas, 1911: lateral approach; Hartel, 1911: anterior approach), gasserian ganglion electrocoagulation (Kirschner, 1942), injection of hot water (Jaeger, 1957) or phenol (Jefferson, 1963) or glycerol (Hakanson, 1981) in the trigeminal cistern and gasserian ganglion cryolysis (Fasano, 1976), have only historic value. Nowadays the neurosurgical armamentarium includes more traditional treatment options, either percutaneous (such as radiofrequency thermorhizotomy and balloon microcompression) or open techniques, such as microvascular decompression, along with novel radiosurgical techniques. All of these treatment options seem to have a good success rate with low risks, so that the ideal algorithm of treatment is still far from being established. In this chapter the authors report on their experience in the treatment of this painful condition and discuss the hypothesized etiopathogenesis of the disease.

Microvascular decompression

Surely, a milestone in the management of medically intractable TN is microvascular compression of the trigeminal nerve, whose concept was first described by Dandy in 1934, rediscovered by Gardner and Miklos (1959), and fully recognized and popularized by Jannetta (1967). In the past 30 years thousands of patients have undergone successful microvascular decompression and today it represents one of the most widely used surgical options for TN. Several studies agree on the high rate of long-term success (Table 57.1) and even authors who are against the concept of microvascular compression perform it for its effectiveness (Adams, 1989).

Nevetherless, controversies still exist about the exact role of neurovascular conflict in the pathogenesis of the disorder, about the possible involvement of the same mechanism also in patients affected by multiple sclerosis, and about the existence of reliable prognostic factors.

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Table 57.1

Completely painfree patients (CPFPs) after microvascular decompression for trigeminal neuralgia

References	Number of patients	Number (%) of CPFPs	Significant recurrence (%)	Follow-up (mean)
Taarnhoj (1982)	350	225 (64.3)	113 (32.3)	Up to 11.5 years
Szapiro et al. (1985)	68	56 (82)	2 (3)	1–5 years
Burchiel et al. (1988)	36	19 (53)	11 (30)	7.5–11.5 years (8.5 years)
Bederson and Wilson (1989)	252	189 (75)	44 (17)	0.5–16 years (5 years)
Dahle et al. (1989)	54	43 (79)	11(21)	3–7 years (3.1 years)
Sindou et al. (1990)	60	50 (83)	2 (3)	16 months
Klun (1992)	178	167 (94)	5 (3)	0.5–12 years (5.2 years)
Yamaki et al. (1992)	60	38 (63)	9 (15)	0.5–5.5 years
Sindou and Mertens (1993)	420	/–(91)	/–(6)	?
Sun et al. (1994)	61	46 (75)	10 (16)	1–10 years (80 months)
Mendoza and Illingworth (1995)	133	95 (71)	18 (13)	0.5–15 years (5.3 years)
Barker et al. (1996)	1185	903 (76)	282 (24)	1–20 years (6.2 years)
Kondo (1997)	281	244 (87)	23 (8)	>5 years
Liao et al. (1997)	80	?	5	0.75–4 years
Coakham and Moss (1998)	>150	?	/–(10)	Up to 17 years
Present report (2004)	563	428 (76)	84 (15%)	5–13 years (4.5 years)

Our experience with microvascular decompression started in 1990 and so far 563 patients, including 38 patients affected by multiple sclerosis, have been operated upon. All patients who were drug-resistant or intolerant, who did not want to experience any sensory disturbance, and who were eligible for general anesthesia underwent this kind of surgery as first option. Advanced age was not considered as an absolute contraindication at our institution. All patients had brain magnetic resonance with gadolinium before operation in order to exclude intracranial lesions (such as neoplasms) that could be responsible for the symptomatology. Absence of clear neurovascular conflicts in posterior cranial fossa on neuroradiological studies did not contraindicate surgery, and in fact almost all of the patients who were successfully operated on at our institution did not present such a magnetic resonance imaging (MRI) picture.

Surgical technique and side-effects

Exposure of the cerebellopontine cistern, where trigeminal roots reside, was performed through a small (less than 20 mm in diameter) retromastoid craniectomy, in the supine position with the head rotated to the opposite side of neuralgia and the ipsilateral shoulder slightly elevated. The skin incision was done a few millimeters medial to the mastoid notch (mediolaterally), perpendicular to the inion–zygomatic line, and extending for two-thirds above and for one-third below the mastoid notch. The craniectomy is centered on the astherion, which is the pont of convergence of occipitomastoid, lambdoid, and parietomastoid sutures. The margins of the transverse and sigmoid sinuses were exposed; the dura was then opened along the line bisecting their angle. The fifth cranial nerve was exposed through a supracerebellar approach, thus avoiding lateral retraction of cerebellar hemisphere and traction of VII–VIII cranial nerve complex. So as to avoid anatomical modification before dural opening, lumbar cerebrospinal fluid (CSF) draining was not performed, nor was mannitol used. Conversely, CSF outflow following dural opening was useful in reducing the need for retracting the cerebellar hemisphere. In approaching trigeminal nerve, care was taken to spare at least two petrous veins, which drain into the superior petrous sinus.

The trigeminal nerve was then examined microsurgically for vascular (arterial, venous, or both) compression at the root entry zone and along the whole cisternal course (Figure 57.1) A neurovascular contact was graded as a "severe conflict" when there was a clear groove on the trigeminal root. Neurovascular

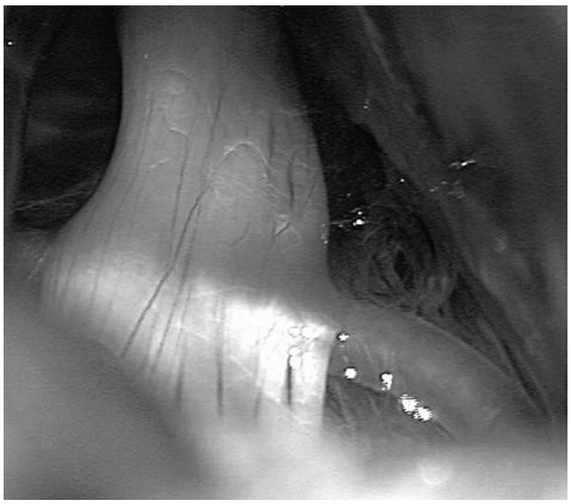

Fig. 57.1. Vascular relationship between trigeminal nerve and the superior cerebellar artery in the cerebellopontine cistern. The distortion of the trigeminal rootlets is evident from this intraoperative microscopic photograph.

contacts without root distortion were graded as "mild conflicts." Sometimes many small venous vessels literally "going through" and distorting the trigeminal sensitive root were seen. In our series the superior cerebellar artery was the vessel most commonly found to be responsible for trigeminal compression, followed by anterior inferior cerebellar artery and basilar artery. The nerve was then cautiously dissected free without unnecessary manipulation. Any compressive arterial vessels were kept away from the nerve as well as from its root entry zone into the brainstem by the use of little pieces of Teflon or fibrillar oxidized cellulose (fibrillar Surgicel). Since in our experience an inflammatory tissue reaction to the Teflon felt was found to be related in some cases to the recurrence of pain, fibrillar absorbable oxidized cellulose has been used since 2002. However, care is taken to avoid, when possible, any contact between the implant and the nerve. Compressive veins were electrocoagulated and divided. Perioperative steroids (dexamethasone) were routinely used.

In our series there was no mortality and no permanent morbidity. Ataxia, disequilibrium, and gait disturbances sometimes found in the early postoperative period generally decreased at hospital discharge (3 days after operation) and fully and spontaneously recovered within 2 weeks without the need for rehabilitation. Collecting data from the literature series on more than 3000 published cases (Table 57.2), the mortality rate is 0.3% (Broggi, 2000) (12 of 3033). Cranial nerve morbidity is reported, but generally diplopia, dysphagia, facial weakness, vertigo, and trigeminal hypoesthesia are all transient. Injury to the acoustic branch of the eighth cranial nerve is the only relevant long-term cranial nerve dysfunction reported in several series, ranging from 0.1% to 3% (Table 57.2). Probably this is the only complication that cannot be prevented in all cases because of the extreme vulnerability of the internal auditory artery and its cochlear branches, as well as its unpredictable intracisternal course. In our hands switching the approach from laterocerebellar to supracerebellar reduced the manipulation of the VII–VIII cranial nerve complex and the incidence of this complication to less than 1%.

Other reported complications, such as CSF leakage, hemotympanum, sigmoid sinus thrombosis, cerebellar infarct, and hematoma can be reduced in incidence with a careful surgical technique and perfect hemostasis.

We did not find any age-related statistically significant difference in incidence of surgical complications and so we perform microvascular decompression without an absolute age limit. Furthermore, in elderly patients surgical exposure of the cerebellopontine angle was found to be easier because of atrophy and subsequent enlargement of cisternal spaces, and the postoperative course was generally uneventful with early mobilization. Multiple sclerosis (MS) patients tolerate this kind of surgery as well as non-MS patients and a worsening of MS symptoms related to surgery was never observed, maybe because of the use of perioperative steroids.

Results and prognostic factors

At long-term follow-up (10–17 years) 76% of patients were found to be completely painfree without medication, 5% were found to be painfree with a dosage of drugs that was lower than in the preoperative period, and 15% required repeated surgery or high dosage of drugs. We were unable to follow up 4% of patients. The outcome in the MS group was worse. Only 39% of patients were completely painfree without medication at long-term follow-up; an additional 5% reported no pain with low-dosage, sporadic assumption of drugs.

Despite the high recurrence rate these results show that a generally considered contraindicated surgery can achieve excellent results in some MS patients. Unfortunately, however, as has already been reported (Broggi et al., 1999), we were not able to find out any prognostic factor that might allow for a better selection of surgical candidates; this obviously reflects the compartmentalization of knowledge about the pathogenesis of TN in those patients. We utilized a statistical analysis of essential TN group in order to relate likelihood of postoperative recurrence of tic

Table 57.2

Microvascular decompression: mortality and long-term side-effects

References	Patients (*n*)	Mortality	Cerebellar infarct	Def VIII°	Def VII°	Dipl	Def V°	PD
Taarnhoj (1982)	350	2 (1.1%)	0.3 %	1.4 %	0.6 %	0.3 %	0	0
Barba and Alksne (1984)	37	0	0	0	0	0	5%	0
Zorman and Wilson (1984)	125	0	0	3%	0	0	0	0
Szapiro et al. (1985)	70	1 (1.43 %)	1.4 %	0	0	0	0	0
Bederson and Wilson (1989)	252	2 (0.07 %)	0	3%	0	0	0	0
Dahle et al. (1989)	57	1 (1.7%)	0	0	0	0	1.7%	1.7%
Sindou et al. (1990)	60	0	0	0	0	0	0	0
Klun (1992)	220	3 (1.3 %)	0	0.4 %	0	0	0	0
Sun et al. (1994)	61	0	0	1.5 %	0	0	1.5 %	1.5 %
Meneses et al. (1995)	50	0	0	0	0	0	0	0
Pamir et al. (1995)	32	0	3 %	0	0	0	0	0
Mendoza and Illingworth (1995)	133	1 (0.7 %)	1.4%	0	0	0	0	0
Barker et al. (1996)	1336	2 (0.2 %)	0.1%	1 %	0	0	0	0
Present report (2004)	563	1 (0.2 %)	1 (0.2%)	0.6%	0	0	0.8%	0

Def VIII: deficit of VIIIth cranial nerve; def VII: deficit of VIIth cranial nerve; Dipl: diplopia; Def V: deficit of Vth cranial nerve; PD: painful dysesthesia.

to the following variables: patient's age and sex; involved side and branch; duration of symptoms; history of previous trigeminal ablative procedures; kind of neurovascular conflict (arterial, venous, or both); postoperative numbness; and arterial hypertension. A long duration of clinical history (> 84 months) was the only variable which was found to be statistically associated to a worse outcome ($P < 0.05$). No other statistically significant prognostic factor was identified.

Etiopathogenetic considerations

A peripheral hypothesis (Kerr, 1967; Rappaport and Devor, 1994), central hypothesis (Dubner et al., 1987), and, more recently, theories trying to reconcile central and peripheral hypotheses (Fromm et al., 1984; Pagni, 1993; Scaioli et al., 1996) about the etiopathogenesis of TN have been invoked. Nevertheless it seems to remain a mystery. It seems likely that both trigeminal nerve lesions and central lesions that affect trigeminal pathways (MS, ischemia) (Vilming et al., 1981; Balestrino and Leandri, 1997) appear to play an etiopathogenetic role in TN. Vascular cross-compression is now increasingly accepted as an important etiological factor. We found a vascular contact in most cases, even in patients with MS. Sometimes the involved vessels are subtle and the root does not seem to be grossly compressed. Our MR data definitively demonstrate that the involvement of trigeminal pathways within the brainstem is very common in TN MS patients. It is possible that demyelination of trigeminal fibers at the level of trigeminal root entry zone in the case of vascular cross-compression (Kerr, 1967; Waxman and Ritchie, 1981; Fromm et al., 1984; Jannetta, 1993; Hilton et al., 1994; Rappaport and Devor, 1994; Love et al., 1998) and demyelination of the trigeminal pathways within the brainstem in the case of MS (Olaston et al., 1966) may result in abnormal ephaptic transmission of impulses. We found that vascular conflict (and possible consequent demyelination) and MS demyelination can coexist and that they may cooperate in the genesis of painful attacks.

The classic distinction between the supposed "all-central" mechanism for MS-associated TN and the "all-peripheral" mechanism for the vascular compression-related TN should therefore come under reconsideration. In its place we offer a unique (TN MS patients are included), mixed central–peripheral mechanism in which abnormal impulses arise from demyelinated axons (MS, vascular compression, and any other possible cause of demyelination along the central and the peripheral course of trigeminal axons) and modulate the nuclear activity. Minimum myelin damage, without any nerve hypofunction, might be involved in the etiopathogenesis of idiopathic TN (Dubner et al., 1987). Major myelin damage may be responsible for MS-associated TN based on the finding of possible clinical signs of trigeminal nerve hypofunction (Vilming et al., 1986), MRI signs of demyelination, and, unfortunately, by the recurrence of

pain after MVD. The concept of a central neuromodulatory role of impulses coming from the area of cross-compression also explains the possibility that a long-lasting alteration of discharge modalities of the trigeminal root can cause lowering of the pain threshold, as suggested by reports on extracranial neurovascular conflicts (Rose, 1890; Franzini et al., 1995).

If this mixed peripheral–central hypothesis appears to be compatible with our (Broggi, 2000) and others' (Adams, 1989) apparently contradictory findings in TN, an alternative all-central hypothesis might also be considered. Supporters of this all-central mechanism deny any pathological role for vascular compression. According to this view, microvascular decompression elicits pain relief because it produces a sufficient trauma which then interferes with normal nerve functioning which then dampens the abnormal brainstem activity responsible for TN (Dandy, 1934). Such activity, according to us, could be initialized by nucleus caudalis and then spread to sensory nuclei of thamalus and then to somatosensory, limbic, and associative cortical circuits of cerebral cortex. In our series we were not able to identify any precise prognostic factor. In particular, no statistically significant difference in the outcome between patients with severe versus mild conflicts was found, which we believe adds further emphasis to the major role played by central mechanisms in patients with MS-related TN.

However, microvascular decompression certainly acts on pain modulation by peripheral pathological impulses and, even if it could not be considered as the *"*definitive etiological cure*"* (Taarnhoj, 1982) it certainly is the only therapeutic option able to interfere with the peripheral etiologic mechanisms of TN without causing any sensory disturbance. Of course, as stated above, the importance of the absence of intracranial (and, in particular, at the cerebellopontine angle) lesions must be kept in mind, such as neoplasms or arteriovenous malformations at preoperative MRI studies, in order to exclude other potential causes of TN.

PERCUTANEOUS METHODS

Hartel's landmarks for cannulation of foramen ovale (Hartel, 1911) are: (1) a point immediately inferior to the medial portion of the ipsilateral pupil in the anterior–posterior plane; (2) a point situated approximately 2.5 cm anterior to the external auditory meatus in a lateral plane; and (3) the cannula's entry point (about 3 cm lateral the ipsilateral side of the oral commissure).

After cannulation use of lateral fluoroscopic mages is important in order to exclude penetration into the foramina of the skull base, such as inferior orbital fissure (located anterior to the foramen ovale) and the jugular foramen (located posterior to it). Such misplaced cannulation could potentially lead to serious neurovascular injuries. Even punctures of the internal carotid artery, with subsequent catastrophic consequences, have been reported in the literature after percutaneous thermocoagulation (Rish, 1976). Since Hartel introduced this simple and direct percutaneous approach to the foramen ovale and gasserian ganglion in 1911, many different methods of creating therapeutic damage to the trigeminal root and ganglion have arisen.

In order to reduce trigeminal sensory input, chemical agents such as alcohol, phenol, and glycerol (with or without phenol) were used. The possibility of diffusion of more aggressive neurolytic agents (such as alcohol) to untargeted structures and different individual response to chemical neurolysis made the results of the injection of chemicals into the trigeminal cistern and ganglion quite unpredictable. Due to an unfavorable recurrence rate and a high incidence of side-effects these techniques were progressively abandoned in favor of controlled radiofrequency thermal rhizotomy and mechanical balloon microcompression.

Radiofrequency retrogasserian controlled thermorhizotomy

Radiofrequency retrogasserian controlled thermorhizotomy became the widely preferred treatment for TN after Sweet and Wepsic introduced this technique in 1974. The primary objective in such a procedure is generation of a thermal lesion within the trigeminal division whose sensory distribution corresponds to the location of patient's referred pain. The temperature of the electrode for lesioning generally ranges from 65°C to 90°C, and the duration of lesion usually lasts 60–100 seconds, with sequential increases of 5°C.

Again, with the aid of a fluoroscope it is possible to increase the probability of reaching such a goal, based on the clivus radiological profile in lateral projections. Electrode tips superimposed on the clivus profile will generate lesions within the second trigeminal branch; tips located beyond or beneath the clivus profile will lesion the first and the second branches, respectively. It is also possible to generate lesions in different gasserian locations (Figure 57.2).

Several experimental data supporting the effectiveness of thermorhizotomy for the differential destruction of small-diameter nerve fibers have been reported (Frigyesi et al., 1975; De la Porte and Siegfried, 1983; Broggi and Siegfried, 1997) and its efficacy has been confirmed by many authors in large series of patients (Sheldon et al., 1955; Siegfried and Vosmansky, 1975; Siegfried, 1981, 1984; Apfelbaum,

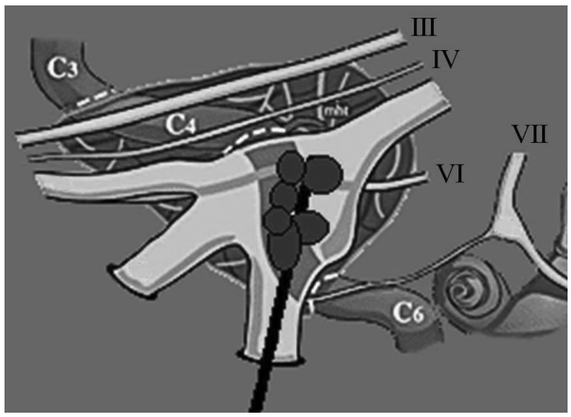

Fig. 57.2. Different possible gasserian ganglion thermoablative sites which can be generated using the lateral clivus profile as a point of reference in lateral fluoroscopic intraoperative views.

1984; Frank et al., 1985; Mittal and Thomas, 1986; Broggi et al., 1990; Taha and Tew, 1996). These experimental and clinical data show that thermorhizotomy allows for sparing of the majority of facial touch sensibility and hypalgesia or analgesia generally involves only the targeted trigeminal branches.

More than 1700 patients have been treated so far at Istituto Nazionale Neurologico Carlo Besta since 1974. We were able to follow up 97% of patients for a time ranging from 2 to 15 years (mean follow-up 72 months): 71% of patients were found to be completely painfree without medication, 11% were painfree with low dosage of antineuralgic drugs, while 15% were still experiencing severe pain requiring high dosage of drugs or surgery (Table 57.3). Regarding the amount of the inflicted sensory deficit, our data suggest that induced postoperative analgesia prevents the recurrence of pain in most patients. In other words, patients with postoperative hypalgesia have a pain recurrence probability of 41% versus 7.5% for patients with postoperative analgesia. In all patients the sensory deficit tends to diminish with time; nevertheless a high percentage of patients with the more severe sensory postoperative deficit (analgesic patients) complain of dysesthesia. The total percentage of patients who required drugs for severe dysesthesia was 5%, with 1.5% with painful anesthesia that we were never able to alleviate definitively by any of the more advanced surgical antalgic techniques (open or percutaneous trigeminal tractotomy, trigeminal stimulation, cortical stimulation, deep-brain stimulation, CSF direct drug infusion). These complications are clearly related to the technique itself and cannot be completely avoided even with meticulous surgical technique, above all in the cases requiring repeated thermorhizotomy.

Table 57.3

Thermorhizotomy for trigeminal neuralgia: long-term results and side-effects in 1700 cases

Completely painfree without medication	71%
Pain requiring high dosage of drugs or surgery	15%
Painfree with low dosage of drugs	11%
Masseter weakness	10%
Dysesthesia requiring medical treatment	5%
Painful anesthesia	1.5%
Ocular palsy and diplopia	0.5%
Corneal reflex impairment without keratitis	19.7%
Corneal reflex impairment with keratitis	0.5%
Cerebral hemorrhage	0%
Death	0%

By monitoring the corneal reflex during the procedure, however, major ocular deafferentation complications can be generally avoided and keratitis requiring tarsorrhaphy was observed in only 0.5% of patients even if the involvement of the first branch was not considered to be a contraindication to this kind of surgery. Masseter weakness with minor chewing impairment appeared in 10% of patients, ocular palsy and diplopia in 0.5%. Major neurological morbidity due to intracranial bleeding was never observed. Mortality was null. This method can be proposed to patients accepting the risk of sensory disturbances when previous less aggressive procedures failed.

Balloon microcompression of the gasserian ganglion

The observation by Sheldon et al. in 1955 that deliberate direct compression of the trigeminal ganglion was able to relieve trigeminal pain led Mullan and Lichtor in 1983 to develop a percutaneous technique for controlled compression of the trigeminal ganglion that could be carried out under short general anesthetic. The results that we were able to obtain using balloon microcompression of the gasserian ganglion in 235 patients operated upon since 1992 are reported in Table 57.4. The end point for compression was the achievement of a pear-shaped balloon in the Meckel's cave (Figure 57.3). The balloon is then maintained inflated for approximately 1 min. A longer compression resulted in a profound hypoesthesia that often led to the complaining of dysesthesias. Results derived from the literature are summarized in Table 57.5. This method appears to have the same limitations that characterize trigeminal surgery whatever the lesional procedure used, that is: the greater the sensorial deficit, the longer the painfree interval but the higher the rate of severe dysesthesia.

Table 57.4

Percutaneous microcompression for trigeminal neuralgia: long-term results in 235 cases

Completely painfree without medication	58%
Requiring low dosage of drugs	12%
Requiring high dosage of drugs or surgery	30%
Painful anesthesia	0%
Requiring drugs for dysesthesia	4%
Permanent diplopia	0.4%
Cheratitis	0%

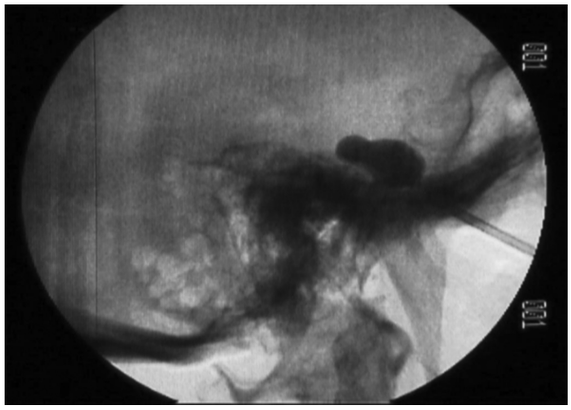

Fig. 57.3. Intraoperative fluoroscopic view of a correctly positioned "pear-shaped" balloon used for percutaneous microcompression of gasserian ganglion.

However, percutaneous microcompression is easy to perform, and the recurrence rate is acceptable with a low rate of complications even in the case of repeated surgery. Diplopia was sometimes observed but it is generally transient. Since in our opinion painful anesthesia and keratitis appear to be too high a price to pay for pain relief, this is at present the method we prefer when microvascular decompression fails or is refused by the patient.

RADIOSURGERY

Stereotactic gamma knife radiosurgery was first reported for the treatment of TN by Leksell in 1971. Its use, however, remained restricted to few centers until the mid-1990s when it started to become more and more popular. Radiosurgical treatment of TN has been well investigated with gamma knife devices involving fixed cobalt (^{60}Co) sources. Planning of the target and determination of isodose curves require 2-mm thick (or less) brain MR scans encompassing the course of trigeminal nerve at its exit from the anterolateral portion of the pons (Figure 57.4), often followed by gadolinium-enhanced images; severely claustrophobic patients can be opportunely sedated with oral or endovenous agents, or can undergo brain computed tomography (CT) with or without intracisternal contrast (Worthington et al., 2000).

Indications for gamma knife radiosurgery are substantially the same as for microvascular decompression, except, of course, for the elegibility for general anesthesia and the patient's willingness to undergo surgery. Actual maximal doses, centered on the cisternal trigeminal nerve, are between 75 and 90 Gy (100% isodose). Doses below 70 Gy are unlikely to be effective at ameliorating symptoms (Kondziolka et al., 1996). Few reports exist concerning TN treated using linear accelerator (LINAC)-based devices. In recent years these devices have reached the level of mechanical precision that is required for such functional treatments. The first study to report preliminary data on treatment of TN with Cyberknife was the one by Romanelli et al. (2003). In this study the authors reported a response rate of 70% (7 out of 10 patients). Lim et al. (2005) reported an initial pain relief rate of 92.7% after a median of 7 days of treatment, which decreased to 78% at 11-month follow-up. In our institution Cyberknife has been available since March 2004 but our data are too preliminary to be reported. Substantial advantages have been supposed in safety and comfort over other modalities but so far the evidence is based on

Table 57.5

Trigeminal neuralgia: reported results of percutaneous microcompression

	Recurrence rate	Follow-up	Patients (*n*)
Skirving and Dan (2001)	32%	10.5 years	496
Natarajan (2000)	8%	1 year	40
Abdennebi et al. (1997)	32.5%	51 months	200
Brown and Gouda (1997)	26%		141
Peragut et al. (1991)	20.6%	16.5 months	70
Lobato et al. (1990)	9.7%	10–35 months	144
Mullan and Lichtor (1983)	12%	0.5–4.5 years	50

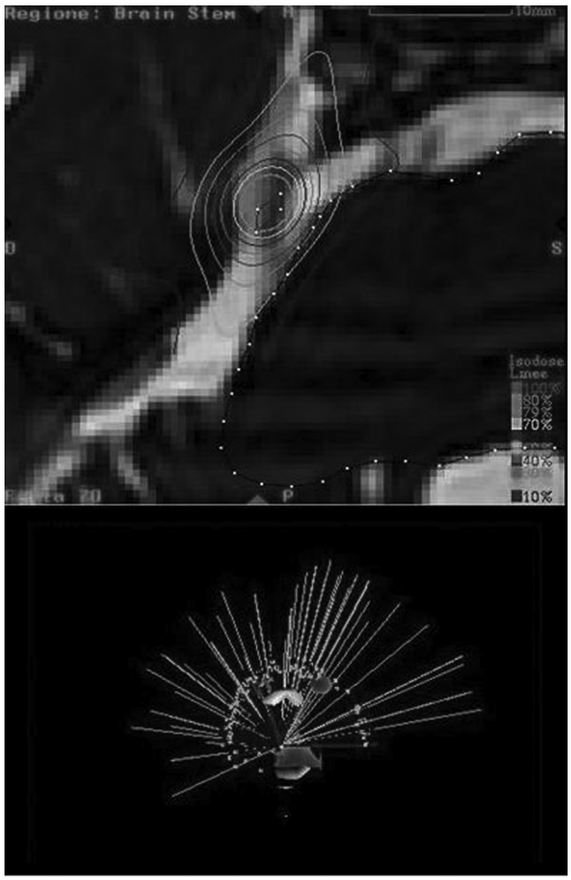

Fig. 57.4. Isodose distribution comprising the entire intracisternal course of trigeminal nerve. Concentrical isodose lines are also depicted in this T_2-weighted magnetic resonance imaging scan.

case series with a single randomized study comparing two methods of delivery of radiotherapy (Pollock et al., 2001). The results obtained in some of the more significant series are reported in Table 57.6. From an analysis of the literature the following conclusions can be drawn:

1. Radiosurgery should be considered as a lesional procedure
2. A strong correlation between the development of new facial sensory loss and achievement and maintenance of pain relief after this procedure has been described (Rish, 1976).
3. Quality of data is generally poor: case series have different patient populations, varying doses of radiation and targets, a variety of assessment methods, and differing follow-up.
4. In all, 70–80% of patients are painfree in the short term, although up to 50% may relapse.
5. Side-effects include facial dysesthesia (up to 12%), corneal irritation (Matsuda et al., 2002), vascular damage, hearing loss, and facial weakness, varying with the dose plan and target area.
6. Follow-up is short and uncertainty persists about possible late complications of radiation therapy.

SUMMARY

Microvascular decompression is the only surgical option that allows for long-term pain relief while avoiding any sensory disturbance. In our opinion it remains the treatment of choice for all patients with drug-resistant typical TN. Old age and central demyelination *per se* do not constitute absolute contraindications to this kind of surgery. No age-related statistically significant difference in incidence of surgical complications has been demonstrated. In addition,

Table 57.6

Results of radiosurgery for idiopathic trigeminal neuralgia

References	Excellent pain relief	Good pain relief	Failures	Follow-up
Shaya et al. (2004)	40%	30%	30%	14 months
Herman et al. (2004)	50%	28%	22%	37.5 months
Goss et al. (2003)	76%	24%	32%	4–13 months
Kanner et al. (2004)	Excellent + good	71.4%	23.2%	
Zheng et al. (2001)	52%	31%	17%	23.7 months
Kondziolka et al. (2002)	55.8% of patients had complete or partial pain relief at 5 years		44.2%	60 months
Matsuda et al. (2002	52%	29%	19%	13 months
Nicol et al. (2000	73.8%	21.4%	4.8%	14 months
Han et al. (1999	42%	35%	23%	9 months
Kondziolka et al. (1996)	58%	36%	6%	18 months

although the results of microvascular decompression in patients affected by MS (as well as the results of percutaneous methods: Broggi et al., 1990) are less satisfactory, about 40% of MS TN patients were found to be painfree at long-term follow-up. Since sensorial deficits can be far from negligible and are not well tolerated in some patients treated with lesion procedures, our policy is to delay destructive surgery as much as possible. When these procedures cannot be avoided percutaneous microcompression appears to be the easiest to perform with a low complication rate and good long-term results. In cases requiring more aggressive treatment because of recurrent pain, thermorhizotomy can be performed. The use of radiosurgery is still under investigation and further studies are required to clarify its role in the treatment of TN and the long-term follow-up rate of responders. Unfortunately, in MS patients both microvascular decompression and lesioning procedures cannot prevent pain recurrence due to MS-related evolving demyelination. Thus, new treatments aiming to modulate the activity of central trigeminal pathways should be investigated to improve the quality of life for these unfortunate patients.

Glossopharyngeal neuralgia

Microvascular decompression for glossopharyngeal neuralgia

Idiopathic glossopharyngeal neuralgia (GN) is characterized by severe, paroxysmal episodes of lancinating pain localized to the external ear canal, the base of the tongue, the tonsil, or the area beneath the angle of the jaw. The pain may originate in the external ear canal and then irradiate to the throat, or vice versa, and is similar as regards pattern of recurrence and clinical characteristics to that experienced with TN, except for triggering factors (yawning, swallowing, and coughing in the case of GN). Painful attacks can be associated with hemodynamic instability that can lead to life-threatening syncopal episodes (Ferrante et al., 1995), hypotension (Weinstein et al., 1986), or bradycardia. Females appear to be more affected than males, with a ratio of approximately 2:1 (Patel et al., 2002).

Several surgical approaches to medically intractable GN have been described, but most rely on the destruction of the glossopharyngeal and vagus nerves. In 1936, Lillie and Craig described an anomalous arterial loop in a patient affected by GN. Again, today microvascular decompression represents one of the most widely used surgical options for GN, though controversies still exist on its role in this kind of neurovascular conflict. Surgery on the lower cranial nerves is in fact generally considered dangerous, and only a few authors reported on the long-term results of microvascular decompression for GN, due to the rarity of this disease. To bring new insight to this topic we critically reviewed 17 consecutive patients who received microvascular decompression between 1990 and 2007 at our institution. Patients received the diagnosis of typical idiopathic GN if their symptoms met the guidelines of the International Headache Society. Individual symptoms, clinical history, operative findings, and complications were recorded. The most common pain territory distributions were epipharynx, followed by epipharynx and external ear, epipharynx and posterior hemitongue, external ear, and posterior hemitongue. In 3 cases GN was associated with TN and in 1 case with hemifacial spasm.

Duration of preoperative symptoms ranged between 45 days and 12 years (mean 4.6 years). Thirteen patients had GN for more than 4 years. One patient had received section of stylomastoid ligament.

Operative results were assessed by clinical follow-up and periodic phone surveys. All of these patients had been given previous medical therapy (carbamazepine, diphenilhydantoin, and, more recently, lamotrigine, gabapentin, and pregabalin also) to which they had become refractory or intolerant. Contrast-enhanced CT and MRI were routinely performed to exclude cerebellopontine angle mass lesions and to find signs of demyelinating disease or neurovascular compression. As most cases had had MRI in other hospitals before admission to our institute, sequences varied and specific studies such as magnetic resonance tomographic angiography were used only in some cases.

Surgical technique

Exploration of the cerebellopontine and lateral cerebellomedullary cisterns was performed as for TN, except for the slightly lower skin incision (located half above and half below the mastoid notch). The margin of sigmoid sinuses was exposed from its beginning to the region behind the mastoid tip. A 15–18 mm dural incision parallel to the course of sigmoid sinus was performed. Microsurgical sharp opening of the arachnoid of the cerebell-medullary cistern allowed the course of IX–X cranial nerves to be exposed by using gentle gravity-aided tension on the cerebellum rather than a fixed spatula. Sharp dissection of the arachnoidal adherences around the nerves allowed for the full exposition of glossopharyngeal and vagal nerves, including the root entry zone in the retro-olivar sulcus. The most commonly involved vessel was the posterior inferior cerebellar artery, followed by vertebral artery. Such compressive arteries were relocated away from the nerves and fixed in the final position far from the nerves themselves using muscle or fibrillar Surgicel.

Compressive veins were electrocoagulated and cut. Great care was taken to identify and respect all the small perforating arteries that could limit artery relocation. Brainstem auditory evoked potentials and endoscopic assistance were sometimes utilized in order to visualize "dark" microscopic areas better (Broggi et al., 1995).

Results

At the first operation a microvascular compression at the vagoglossopharyngeal root entry zone was found in all patients. The stabbing, paroxysmal pain typical of vasoglossopharyngeal neuralgia disappeared immediately after surgery in 15 out of 17 patients (88%) and faded away within 2 weeks in another 2 (12 %). Two patients required repeated surgery after 2 and 5 years for a drug-resistant recurrence of pain. In both incomplete decompression was found at repeated surgery.

A total of 15 out of 17 patients (88%) are painfree without medication at long-term follow-up (1–17 years, mean 7.5 years). The remaining 2 patients are under medication (low-dose carbamazepine) for pain paroxysms that reappeared after a few weeks and 1 year respectively and that are, however, less frequent and severe than in the preoperative period.

There was neither mortality nor long-term surgical morbidity in this series. No patient had permanent deficits of cranial nerves. Transient function impairment of cranial nerves was observed in almost one-third of cases. The postoperative course was commonly characterized by cephalalgia and nausea due to deliquoration that is always required to expose the cerebellomedullary cistern. Cephalalgia lasted longer (5–7 days) in 4 patients. In 1 of these patients there was a CSF rhinorrhea that ceased after 3 days of external lumbar CSF drainage.

DISCUSSION

Despite the fact that new drugs, such as gabapentin, lamotrigine, levetiracetam, and pregabalin, have been introduced in the treatment of GN (as well as for TN), many patients still require surgery because of refractoriness or intolerance. The diagnosis of GN is clinical and the role of neuroimaging is key only to detect possible causes, including vascular compression, tumors, or demyelinating plaques, as for TN. Again, the absence of an MRI-identified neurovascular compression should not exclude patients with intractable pain from open surgery.

The recognition of an involvement of the vagus nerve in symptoms led to the term "vagoglossopharyngeal neuralgia." In fact, the coexistence of symptoms mediated by ninth and 10th cranial nerves is well explained from an anatomical point of view, since both share the retro-olivar sulcus (the superior portion of the posterior lateral sulcus of medulla oblungata) as the common place of emergence from the brainstem, and where they can be distorted or compressed by a vascular loop or a tumor. The descending trigeminal tract and the nucleus caudalis are also shared as the first station of pain fiber relay coming from both nerves.

Most surgical treatments have historically focused on the lesion of glossopharyngeal and vagal nerve fibers. These lesional surgical procedures include peripheral procedures (extracranial, such as direct surgical neurotomies or percutaneous radiofrequency thermal rhizotomy, or intracranial, such as direct section of glossopharyngeal and vagal nerves in the cerebellopontine angle) and central procedures (percutaneous or open trigeminal tractotomy-nucleotomy, or nucleus caudalis DREZtomy).

As for TN, lesional procedures for pain in general should be avoided when alternative and safer treatments are available. Concerns about safety of microvascular decompression in GN are related to old reports of mortality and morbidity and should be taken into account, since this kind of surgery, along with refinement of microsurgical and anesthesiological techniques, can now be performed with a very low complication rate. In our series there was no mortality and no permanent morbidity. We agree that manipulation of lower cranial nerves can often be associated with morbidity, but our data show that this morbidity can be transient if absolute respect of brainstem vascularization is employed. Cases where the vertebral artery is responsible for root entry zone compression constitute a major challenge and require meticulous attention to avoid any damage to perforating vessels when it is manipulated. When a complete mobilization of the artery away from the retro-olivar sulcus is hampered by short perforators, the simple interposition of some pieces of Surgicel between the artery and the root entry zone should be considered.

Radiosurgery has also been employed with highly variable results that, anyway, are never comparable with the 90% of long-term completely painfree patients without any medication that microvascular decompression warrants.

Microvascular decompression should therefore be considered as the first choice treatment also in all cases of GN. Old patients should be included since in our experience they do not present a statistically significant increased risk of complications. Peripheral lesional procedures are invariably associated with a deficit of ninth and 10th cranial nerves that is only

Table 57.7

Clinical results of microvascular decompression for glossopharyngeal neuralgia

References	Number of patients	Immediate result: number (%) of CPFPs	Long-term result: number (%) of CPFPs	Follow-up
Wakiya et al.(1989)	16	15 (93.75%)	15 (93.75)	1–48 months
Resnick et al. (1995)	40	32 (79%)	30 (76)	0.5–13 years
Kondo (1997)	17	16 (94%)	16 of 17 (1 death)	5–16 years
Patel et al. (2002)	217	145 (67%)	121 of 208(58)	4 years (mean)
Sampson et al. (2004)	47	46 (98%)	28 of 29 (96.5) with long-term follow-up	10.5–17.5 years

CPFPs: completely painfree patients.

Table 57.8

Complication rate in recent surgical series of microvascular decompression for glossopharyngeal neuralgia with more than 10 cases

References	Number of patients and years	Mortality	Transient cranial nerve palsy	Permanent cranial nerve palsy	Cerebrospinal fluid leak
Wakiya et. al. (1989)	16	0	1 (6.25%)		0
Resnick et al. (1995)	40	2 (5%)	4 (10%)	3 (8%)	0
Kondo (1997)	20 1980–1995	1 (5%)	6 (30%)	2 of 17 (11.8%)	0
Patel et al. (2002)	217 1973–2000	13 (5.8%)*	/	62 (28.5%)†	25 (11.4%)‡
Sampson et al. (2004)	47 1984–1991	0	16 (34%)	5 (17.2%) of 29 patients	0

*0% mortality after 1987.
†3% after 1995.
‡2% after 1987.

acceptable in desperate cases where all other treatments have failed.

Coming finally to the etiopathogenesis of GN, it is generally considered, because of clinical and pathological similarities, to be the same as that of typical TN.

CONCLUSIONS

Table 57.7 and 57.8 show the clinical results and complication rates in microvascular decompression for GN in several series, respectively. Even if only few studies report long-term follow-up or patients with GN treated with microvascular decompression in the lateral cerebellomedullary cistern (Resnick et al., 1995; Taha and Tew, 1995; Kondo, 1998; Patel et al., 2002), we think that microvascular decompression is a safe and effective treatment for GN in patients of all ages. Considering that most of patients can withdraw from medication after surgery, it should also be proposed as first choice treatment for patients who do not tolerate the idea of chronic antiepileptic drug administration.

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