Chapter 37
One Size Does Not Fit All: Choosing a Treatment Strategy for Trigeminal Neuralgia

Giovanni Broggi, M.D., Paolo Ferroli, M.D., Angelo Franzini, M.D., and Luciano Galosi, M.D.

Trigeminal neuralgia (TN) is an uncommon disease (incidence 4.7 of 100,000) characterized by attacks of recurring, paroxysmal, shock-like pain within the distribution of one or more branches of the trigeminal nerve. Light tactile stimulation may trigger such an attack. Even if new drugs have been recently introduced in the treatment of TN (20, 26, 46, 49, 86), about half of all patients eventually require surgery for pain relief. Drug resistance or drug intolerance can, in fact, be frequently observed in patients with a long history of disease. Clinical features of TN have been well-known since the first description by Fothergill (22) in 1773, and many different surgical treatment modalities have been applied. Most of them such as gasserectomy (66), retrogasserian neurotomy (77), juxtaprotuberantial neurotomy (16), trigeminal tractotomy (76), temporal intradural decompression (81), gasserian ganglion alcoholization (Taptas-lateral approach) (84); (Hartel-anterior approach) (32), gasserian ganglion electrocoagulation (41), injection of hot water (35), phenol (38), or glycerol (30) in the trigeminal cistern and gasserian ganglion cryolysis (21), have only an historical value.

Nowadays, the neurosurgical armamentarium includes more traditional treatment options, either percutaneous, such as radiofrequency thermorhizotomy and balloon microcompression, or open, such as microvascular decompression (MVD), along with novel radiosurgical techniques. Because all these treatment options seem to have a good success rate with low risks, the ideal algorithm of treatment is still under debate. In this chapter, the authors report on their experience in the treatment of this painful condition and discuss the etiopathogenesis of the disease. Their own treatment algorithm is also presented.

MVD

The concept of microvascular compression of the trigeminal nerve described by Dandy (17) in 1934, rediscovered by Gardner and Miklos (28), and fully recognized and popularized by Jannetta (36) was a milestone in the management of medically intractable TN. In the past 30 years, thousands of patients have undergone successful MVD, and today it represents one of the most widely used surgical options for TN. Several studies agree on a high rate of long-term success (Table 37.1), and even authors against the concept of microvascular compression perform it for its effectiveness (2). There are still controversies about the role of vascular compression in the pathogenesis of the disorder, the possible involvement of the same mechanism also in patients affected by multiple sclerosis (MS), the existence of reliable prognostic factors, and the role of MVD in elderly patients. Our experience with MVD started in 1990, and to date, 563 patients, including 38 patients affected by MS, were operated on. All patients who did not want to experience any sensory disturbance underwent this kind of surgery as first option. Advanced age was not considered as a contraindication.

Results and Prognostic Factors

At long-term follow-up (0.5–13 yr; mean, 4.5 yr), 76% of patients were found completely pain free without medication, 5% were found pain free with a dose of drugs smaller than in the preoperative period, and 15% required repeated

surgery or a large dose of drugs. We were unable to follow up 4% of patients. The outcome in the MS group was worse with only 39% of patients completely pain free without medication at long-term follow-up and an additional 5% reporting no pain with a small dose, sporadic consumption of drugs. Cumulative proportion of completely pain-free patients in both non-MS and MS patients is reported in Figure 37.1. Despite frequent recurrence, these results show that a generally considered contraindicated surgery can achieve excellent results in some MS patients. Unfortunately, however, as has already been reported (8), we were not able to find out any prognostic factors that might allow for a better selection of surgical candidates, and the treatment of TN in MS patients still remains challenging. A statistical analysis of the essential TN group was used to relate likelihood of postoperative recurrence of tic to the following variables: patient's age and sex; involved side and branch; duration of symptoms; history of trigeminal ablative procedures; kind of neurovascular conflict (arterious, venous, or both); postoperative numbness; and hypertension. A long duration of clinical history (>84 mo) was found statistically associated with a worse outcome ($P < 0.05$). No other statistically significant prognostic factor could be identified (9).

Surgical Technique and Side Effects

Exploration of the cerebello-pontine angle is 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 (Figs. 37.2 and 37.3). The margins of the transverse and sigmoid sinuses are exposed; the dura is opened along the line bisecting their angle. The fifth cranial nerve is exposed (Fig. 37.4)through a supracerebellar approach, thus avoiding lateral retraction of cerebellar hemisphere and traction of VII-VIII cranial nerves complex. So as to avoid any anatomical modification before dural opening, lumbar cerebrospinal fluid (CSF) draining is not performed nor is mannitol used. In approaching trigeminal nerve, care is taken to spare at least two petrous veins. The trigeminal nerve is microsurgically examined for vascular compression at root entry zone and along the whole cisternal course. A neurovascular contact is graded as a severe conflict when there is a clear groove on the trigeminal root. Neurovascular contacts without root distortion are defined as mild conflicts. The nerve is cautiously dissected free without unnecessary manipulation. Any compressive arteries are kept away from the nerve and from its root entry zone into the brain stem by the use of little pieces of fibrillar oxidized cellulose (Fibrillar Surgicel, Johnson and Johnson, New Brunswick, NJ) after sharp dissection of arachnoid bands.

In our experience, an inflammatory tissutal reaction to the Teflon felt was found in some cases to be related to the recurrence of pain, and fibrillar absorbable oxidized cellulose has been used since 2002 to avoid “teflomas” distorting the nerve root and causing recurrent pain. Even if surgery is performed, care is taken to avoid, when possible, any contact between the implant and the nerve. Compressive veins are electrocoagulated with bipolar (Malis, Codman) and divided. Perioperative steroids (dexamethasone; 8–16 mg daily for 3–5 d depending on clinical conditions) were routinely used.

In our series, we observed one postoperative hematoma that could be evacuated without long-term sequelae and one hemorrhagic infarction of the cerebellar lobe in a 78-year-old woman who died from a pulmonary infection after 1 month in the intensive care unit. No major permanent morbidity was recorded. Ataxia, disequilibrium, and gait disturbances were sometimes found in the early postoperative period and at hospital discharge (3 d after surgery), and she fully recovered within 2 weeks without rehabilitation. Collecting data from the literature series on more than 3000 published cases, the mortality rate is 0.3% (9). 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

VIII cranial nerve is the only relevant long term cranial nerve dysfunction reported in several series, ranging from 0.1 to 3% (Table 37.2). 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. 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 from 1 to 0.4%.

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 performed MVD without an absolute age limit. Furthermore, in elderly patients, surgical exposure of cerebellopontine angle was found to be easier because of atrophy, and the postoperative course was generally uneventful with early mobilization. MS patients tolerate as well as the non-MS patients this kind of surgery, and a worsening of MS symptoms related to surgery was never observed, perhaps because of the use of perioperative steroids.

Etiopathogenetic Considerations

A peripheral hypothesis (40, 64), a central hypothesis (19), and, more recently, theories supporting central-peripheral hypotheses (27, 60) for TN etiopathogenesis have been proposed. Nevertheless, it remains a puzzling mystery. Both trigeminal nerve lesions and central lesions affecting trigeminal pathways (MS and ischemia) (4, 87) have been reported to play an etiopathogenetic role in TN. Vascular cross compression is now increasingly accepted as an important etiological factor. We found a vascular conflict in most cases, even in patients with MS. Sometimes the involved vessels are subtle, and the root does not seem 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 (27, 34, 37, 40, 51, 64) and demyelination of the trigeminal pathways within the brainstem in the case of MS (59) 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, thus, 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 (19). Major myelin damage may be responsible for MS-associated TN based on the finding of possible clinical signs of trigeminal nerve hypofunction (87), magnetic resonance imaging (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 a decrease in the pain threshold, as suggested by recent reports on extracranial neurovascular conflicts (24, 67).

If this mixed peripheral-central hypothesis seems to be compatible with our (9) and others (2) 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, MVD elicits pain relief because it produces a sufficient trauma that interferes with normal nerve functioning, which then dampens the abnormal brainstem activity responsible for TN (17). In our series, we were not able to identify any prognostic factor. In particular, no statistically significant difference in the outcome between patients with severe versus mild conflicts was found that we believe adds further emphasis to the major role played by central mechanisms in patients with MS-related TN.

However, MVD certainly interferes with the pathological impulses that arise from the region of demyelination induced by chronic vascular cross compression, and even if it could not be considered as the definitive etiological cure (83), it is the only therapeutic option able to obtain pain relief without causing any sensory disturbance.

Percutaneous Methods

Since Hartel introduced his simple and direct percutaneous approach to the foramen ovale and gasserian ganglion in 1911 (32), several different methods to create therapeutic damage to the trigeminal root and ganglion became available. To reduce trigeminal sensory input, chemical agents such as alcohol, phenol, and glycerol with or without phenol were used. Possible diffusion of more aggressive neurolytic agents, such as alcohol, to untargeted structures and different individual responses to chemical neurolysis made the results of the injection of chemicals into the trigeminal cistern and ganglion quite unpredictable. Because an unfavorable recurrence rate and a frequent 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 (TRZ) became the widely preferred treatment for TN after Sweet and Wepsic(79) introduced this technique in 1974. In the following years, experimental data supporting the effectiveness of TRZ for the differential destruction of small diameter nerve fibers have been reported (10, 18, 25), and its efficacy has been confirmed by many authors in large series of patients (3, 11, 23, 55, 70–72, 85). These experimental and clinical data showed that TRZ 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 at Instituto Nazionale Neurologico Carlo Besta since 1974. We were able to follow up with 97% of patients for a time ranging from 2 to15 years (mean follow-up, 72 mo); 71% of patients were found to be completely pain free without medication, 11% pain free with a small dose of antineuralgic drugs, and 15% still experiencing severe pain requiring a large dose of drugs or surgery (Table 37.3).

Regarding the amount of the inflicted sensory deficit, our data suggest that induced postoperative analgesia prevents the recurrence of pain in most of 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 dysesthesias. The total percentage of patients who required drugs for severe dysesthesia was 5%, with 1.5% of painful anesthesia that we were never able to definitively alleviate by any of the more advanced surgical antalgic techniques (open or percutaneous trigeminal tractotomy, trigeminal stimulation, cortical stimulation, deep brain stimulation, or CSF direct drug infusion). These complications are clearly related to

the technique itself and cannot be completed avoided, even with meticulous surgical techniques, especially in the cases requiring repeated TRZ (Fig. 37.5).

However, by monitoring the corneal reflex during the procedure, major ocular deafferentation complications can be generally avoided, and keratitis requiring tarsorrhaphy was observed in only 0.5% of patients, even when the involvement of first branch was not considered as a contraindication to this kind of surgery. Masseter weakness with minor chewing impairment appeared in 10% of patients, whereas ocular palsy and diplopia appeared in 0.5%. Major neurological morbidity caused by 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 have failed.

Balloon Microcompression of the Gasserian Ganglion

The observation that deliberate direct compression of the trigeminal ganglion was able to relief trigeminal pain by Sheldon in 1955 (69) led Mullan, in 1978, to develop a percutaneous relief controlled compression of the trigeminal ganglion that could be performed under short general anesthetic (56). The result that we were able to obtain by using balloon microcompression of the gasserian ganglion (percutaneous microcompression [PMC]) in 235 patients operated on since 1992 are reported in Table 37.4. The end point for compression was the achievement of a pear-shaped balloon in the cavum Meckel (Fig. 37.6). The balloon was then maintained inflated for approximately 1 minute. 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 37.5. This method seems to have the same limitations that characterize trigeminal surgery whatever the lesional procedure used, that is, the more the sensorial deficit, the longer the pain-free interval but the more frequent the severe dysesthesia.

However, PMC is easy to perform, and the recurrence rate is acceptable with a low rate of complication, even in the case of repeated surgery. Diplopia was sometimes observed, but it was generally transient. Because painful anesthesia and keratitis seem, in our opinion, too high of a price to be paid for pain relief, this is now the method we prefer when MVD fails or is refused by the patient.

Radiosurgery

Stereotactic gamma-knife radiosurgery (Electra, Sweden) was first reported for the treatment of trigeminal neuralgia by Leksell in 1971 (47). Its use, however, remained restricted to few centers until the mid-1990s when it started to become more widely used. Radiosurgical treatment of TN has been well investigated with gamma-knife devices involving fixed cobalt sources. 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. Only one study reporting on patients treated with CyberKnife (Accuray, Sunnyvale, CA) is available in the literature (65). In our Institution, CyberKnife has been available since March 2004 (Fig. 37.7), but our data are too preliminary to be reported. Substantial advantages have been supposed in safety and comfort over other modalities, but the evidence is based on case series with a single randomized study comparing two methods of delivery of radiotherapy (63). The results obtained in some of the more significant series of the literature are reported in Table 37.6. From the analysis of the literature, the following conclusions can be drawn:

• Radiosurgery should be considered as a lesional procedure.
• A strong correlation between the development of new facial sensory loss and achievement and maintenance of pain relief after this procedure has been described (63).
• 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.
• Seventy to 80% of patients are pain free in the short term, although up to 50% may relapse.
• Side effects include facial dysesthesia (up to 12%), corneal irritation, vascular damage, hearing loss, and facial weakness, varying with the dose plan and target area.
• Follow-up is short, and uncertainty persists about possible late complications of radiation therapy.

CONCLUSIONS

MVD is the only surgical option that allows for obtaining long-term pain relief while avoiding any sensory disturbance. In our opinion, it still remains the treatment of choice for all patients with drug-resistant typical TN. Old age and central demyelinations do not constitute absolute contraindications to this kind of surgery. Any age-related statistically significant difference in incidence of surgical complications has been demonstrated. In addition, although the results of MVD in patients affected by MS (as well as the results of percutaneous methods) (11) are less satisfactory, approximately 40% of MS-TN patients was found completely pain free at long-term follow-up. Because sensorial deficits can be far from negligible and well tolerated in some patients treated with lesive procedures, our policy is to delay as much as possible destructive surgery. When these procedures cannot be avoided, PMC should be first proposed because it is easy to perform with infrequent general morbidity, especially on the trigeminal sensitivity. In cases requiring more aggressive treatments because of recurrent pain, TRZ can be used. The use of radiosurgery is still under investigation, and further studies are required to clarify its role in the treatment of TN.

Radiosurgery experts propose their radiation therapy methodology as an etiological strategy because of modifications of axonal conductivity at the root entry zone level. However, radiosurgery should be considered as a lesional method until this hypothesis will be definitively confirmed. In MS patients, unfortunately, both MVD and lesioning procedures cannot prevent pain recurrence because of 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 of these patients, refractory to all available surgical and medical therapies. Chronic deep brain stimulation at the thalamic-hypothalamic level might be an interesting option that is at present under evaluation at our institution.

TABLE 37.1. Completely pain-free patients after MVD for trigeminal neuralgia

Author(s)	No. of patients	No. (%) of CPFPs	Significant recurrence (%)	follow-up (mean)
Taarnhoj (82)	350	225 (64.3)	113 (32.3)	up to 11.5 yr

Szapiro et al. (80)	68	56 (82)	2 (3)	1–5 yr
Burchiel et al. (13)	36	19 (53)	11 (30)	7.5–11.5 yr (8.5 yr)
Bederson and Wilson (7)	252	189 (75)	44 (17)	0.5–16 yr (5 yr)
Dahle et al. (15)	54	43 (79)	11(21)	3–7 yr (3.1 yr)
Sindau et al. (73)	60	50 (83)	2 (3)	/–(16 mo)
Klun (42)	178	167 (94)	5 (3)	0.5–12 yr (5.2 yr)
Yamaki et al. (88)	60	38 (63)	9 (15)	0.5–5.5 y
Sindau et al. (74)	420	/-(91)	/-(6)	?
Sun et al. (78)	61	46 (75)	10 (16)	1–10 y (80months)
Mendoza and Illingworth (53)	133	95 (71)	18 (13)	0.5–15 y (5.3 y)
Barker et al. (6)	1185	903 (76)	282 (24)	1–20 y (6.2 y)
Kondo (43)	281	244 (87)	23 (8)	>5 y
Liao et al. (48)	80	?	5	0.75–4 y
Coakham and Moss (14)	>150	?	/-(10)	Up to 17 y
Present report (2004)	563	428 (76)	84 (15%)	0.5–13 years (4.5 y)

CPFPs=completely pain free patients.

TABLE 37.2. MVD: mortality and long term side effects

Author(s)	N° of patients	Mortality	Cereb Inf	Def VIII°	Def VII°	Dipl	Def V°	PD
Taarnhoj (82)	350	2 (1.1%)	0.3 %	1.4 %	0.6 %	0.3 %	0	0
Barba and Alksne (5)	37	0	0	0	0	0	5%	0
Zorman and Wilson (90)	125	0	0	3%	0	0	0	0
Szapiro et al. (80)	70	1 (1.43 %)	1.4 %	0	0	0	0	0
Bederson and Wilson (7)	252	2 (0.07 %)	0	3%	0	0	0	0
Dahle et al. (15)	57	1 (1.7%)	0	0	0	0	1.7%	1.7%
Sindau et al. (73)	60	0	0	0	0	0	0	0
Klun (42)	220	3 (1.3 %)	0	0.4 %	0	0	0	0
Sun et al. (78)	61	0	0	1.5 %	0	0	1.5 %	1.5 %
Meneses et al. (54)	50	0	0	0	0	0	0	0
Pamir et al. (61)	32	0	3 %	0	0	0	0	0
Mendoza and Illingworth (53)	133	1 (0.7 %)	1.4%	0	0	0	0	0
Barker et al. (6)	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

Cereb inf=cerebellar infarct; Def=deficit; Dipl=diplopia; PD=painful dysaestesia

TABLE 37.3. TRZ for trigeminal neuralgia:

Long term results and side effects in 1700 cases

Completely pain free without medication	71%
Pain requiring high dosage of drugs or surgery	15%
Pain free 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 haemorrhage	0%
Death	0%

TABLE 37.4. PMC for trigeminal neuralgia: Long term results in 235 cases

Completely pain free 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%
Keratitis	0%

TABLE 37.5. Trigeminal neuralgia: reported results of PMC

	Recurrence rate	Follow up	Number of patients
Skirving et al. (75)	32%	10.5 years	496
Natarajan (57)	8%	1 year	40
Abdennebi et al. (1)	32.5%	51 months	200
Brown et al. (12)	26%	/	141
Peragut et al. (62)	20.6%	16.5 months	70
Lobato et al. (50)	9.7%	10-35 months	144
Mullan et al. (56)	12%	0.5-4.5 years	50

TABLE 37.6. Results of radiosurgery for idiopathic trigeminal neuralgia

Author, year	Excellent pain relief	Good pain relief	Failures	Follow-up
Shaya et al. (68)	40%	30%	30%	14 months
Herman et al. (33)	50%	28%	22%	37.5 months
Goss et al. (29)	76 %	24%	32%	4-13 months
Kanner et al. (39)	Excellent + good 71.4%		23.2%	/
Zheng et al. (89)	52%	31%	17%	23.7 months
Kondziolka et al. (45)	55.8% of patients had complete or partial pain relief at 5 years		44.2%	60 months
Matsuda et al. (52)	52%	29%	19%	13 months
Nicol et al. (58)	73.8%	21.4%	4.8%	14 months
Han et al. (31)	42%	35%	23%	9 months
Kondziolka et al. (44)	58%	36%	6%	18 months

REFERENCES

1. Abdennebi B, Mahfouf L, Nedjahi T: Long-term results of percutaneous compression of the gasserian ganglion in trigeminal neuralgia (series of 200 patients). **Stereotact Funct Neurosurg** 68:190–195, 1997.

2. Adams CBT: Microvascular decompression: An alternative view and hypothesis. **J Neurosurg** 57:1–12, 1989.

3. Apfelbaum RI: Surgery for tic doloreaux. **Clin Neurosurg** 31:351–368, 1984.

4. Balestrino M, Leandri M: Trigeminal neuralgia in pontine ischaemia. **J Neurol Neurosurg Psychiatry** 62:297–298, 1997.

5. Barba D, Alksne JF: Success of microvascular decompression with and without prior surgical therapy for trigeminal neuralgia. **J Neurosurg** 60:104–107, 1984.

6. Barker FG, Jannetta JJ, Bissonette DJ, et al.: The long term outcome of microvascular decompression for trigeminal neuralgia. **N Engl J Med** 334:1077–1083, 1996.

7. Bederson JB, Wilson CB: Evaluation of microvascular decompression and partial sensory rhizotomy in 252 cases of trigeminal neuralgia. **J Neurosurg** 71:359–367, 1989.

8. Broggi G, Ferroli P, Franzini A, Pluderi M, La Mantia L, Milanese C: Role of microvascular decompression in trigeminal neuralgia and multiple sclerosis. **Lancet** 354:1878–1879, 1999.

9. Broggi G: Microvascular decompression for trigeminal neuralgia: comments on a series of 250 cases, including 10 patients with multiple sclerosis. **J Neurol Neurosurg Psychiatry** 68:59–64, 2000.

10. Broggi G, Siegfried J: The effect of graded thermocoagulation on trigeminal evoked potentials in the cat. **Acta Neurochir Suppl (Wien)** 24:175–178, 1997.

11. Broggi G, Franzini A, Lasio G, Giorgi C, Servello D: Long-term results of percutaneous retrogasserian thermorhizotomy for “Essential” trigeminal neuralgia: considerations in 1000 consecutive patients. **Neurosurgery** 26:783–787, 1990.

12. Brown JA, Gouda JJ: Percutaneous balloon compression of the trigeminal nerve. **Neurosurg Clin North Am** 8:53–62, 1997.

13. Burchiel KJ, Clarke H, Haglund M, et al: Long term efficacy of microvascular decompression in trigeminal neuralgia. **J Neurosurg** 69:35–38, 1988.

14. Coakham HB, Moss T: Microvascular decompression [letter]. **J Neurosurg** 88:617–618, 1998.

15. Dahle L, Von Essen C, Kourtopoulos H, et al: Microvascular decompression for trigeminal neuralgia. **Acta Neurochir (Wien)** 99:109–112, 1989.

16. Dandy WE: An operation for the cure of tic douloureux: partial section of the sensory root at the pons. **Arch Surg** 18:687–734, 1929.

17. Dandy WE: Concerning the cause of trigeminal neuralgia. **Am J Surg** 24:447–55, 1934.

18. De la Porte C, Siegfried J: Étude des paramètres de thérmocoagulation à haute fréquence du ganglion de Gasser dans le traitement de la nevralgie du trijumeaux pour l'obtention d'une analgesie sans anésthesie. **Neurochirurgie** 29:191–195, 1983.

19. Dubner R, Sharav Y, Gracely RH, et al: Idiopathic trigeminal neuralgia: Sensory features and pain mechanisms. **Pain** 31:23–33, 1987.

20. Farago F: Trigeminal neuralgia: its treatment with two new carbamazepine analogues. **Eur Neurol** 26:73–83, 1987.

21. Fasano VA: Diagnostic and therapeutic studies using the cryoprobe in various forms of facial pain, not of the migraine or trigeminal type. **Minerva Med** 67:1845–1849, 1976.

22. Fothergill J: Observations on the use of herlock: Medical observation and inquires by a society of physicians. London, 1773.

23. Frank F, Gaist T, Fabrizi A, Cohadon F, Laporte A, Sedan R, Peragut JC, Sethian M, Sturiale C: Rèsultats de la thérmocoagulation percutanée sélective du ganglion de Gasser dans la nevralgie faciale essentielle. Synthèse des resultats obtenus chez 939 patients traités. **Neurochirurgie** 31:179–182, 1985.

24. Franzini A, Scaioli V, Leocata F, et al.: Pain syndrome and focal myokymia due to anterior interosseous neurovascular relationships: Report of a case and neurophysiological considerations.

J Neurosurg 82:578–580, 1995.

25. Frigyesi T, Siegfried J, Broggi G: The selective vulnerability of evoked potentials in the trigeminal sensory root to graded thermocoagulation. **Exp Neurolol** 49:11–21, 1975.

26. Fromm GH, Terrence CF. Comparison of L-baclofen and racemic baclofen in trigeminal neuralgia. **Neurology** 37:1725–1728, 1987.

27. Fromm GH, Terrence CF, Maroon JC: Trigeminal neuralgia: current concepts regarding etiology and pathogenesis. **Arch Neurol** 41:1204–1207, 1984.

28. Gardner WJ, Miklos MV: Response of trigeminal neuralgia to decompression of sensor y root. Discussion of cause of trigeminal neuralgia. **JAMA** 170:1773–1776, 1959.

29. Goss BW, Frighetto L, DeSalles AA, Smith Z, Solberg T, Selch M: Linear accelerator radiosurgery using 90 gray for essential trigeminal neuralgia: results and dose volume histogram analysis. **Neurosurgery** 53:823–828; discussion 828–830, 2003.

30. Hakanson S: Trigeminal neuralgia treated by the injection of glycerol into the trigeminal cistern. **Neurosurgery** 9:638–646, 1981.

31. Han PP, Shetter AG, Smith KA, et al.: Gamma knife radiosurgery for trigeminal neuralgia: Experience at the Barrow Neurological Institute. **Stereotact Funct Neurosurg** 73:131–133, 1999.

32. Hartel F: Die Leitungsans thesie und injektions behandlung des Ganglion Gasseri und der Trigem inusstmme langenbecks. **Langenbecks Arch Klin Chir Ver Dtsch Z Chir** 100:193, 1911.

33. Herman JM, Petit JH, Amin P, Kwok Y, Dutta PR, Chin LS: Repeat gamma knife radiosurgery for refractory or recurrent trigeminal neuralgia: Treatment outcomes and quality-of-life assessment. **Int J Radiat Oncol Biol Phys** 59:112–116, 2004.

34. Hilton DA, Love S, Gradidge T, Coakham HB: Pathological findings associated with trigeminal neuralgia caused by vascular compression. **Neurosurgery** 35:299–303, 1994.

35. Jaeger R: Permanent relief of tic douloureux by Gasserian injection of hot water. **Arch Neurol Psychiatry** 77:1–7, 1957.

36. Jannetta PJ: Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. **J Neurosurg** 26:159–162, 1967.

37. Jannetta PJ: Vascular compression is the cause of trigeminal neuralgia. **AAPS J** 2:217–227, 1993.

38. Jefferson A: Trigeminal root and ganglion injection sussing phenol in glycerine for the relief of trigeminal neuralgia. **J Neurol Neurosurg Psychiatry** 26:345–352, 1963.

39. Kanner AA, Neyman G, Suh JH, Weinhous MS, Lee SY, Barnett GH: Gamma knife radiosurgery for trigeminal neuralgia: comparing the use of a 4-mm versus concentric 4- and 8-mm collimators. **Stereotact Funct Neurosurg** 82:49–57, 2004.

40. Kerr FWL: Evidence for a peripheral etiology of trigeminal neuralgia. **J Neurosurg** 26:168–174, 1967.

41. Kirschner M: Die Behandlung der trigeminusneuralgia. **Munchen Med Wochenschr** 89:235–239, 1942.

42. Klun B: Microvascular decompression and partial sensory rhizotomy in the treatment of trigeminal neuralgia: personal experience with 220 patients. **Neurosurgery** 30:49–52, 1992.

43. Kondo A: Follow-up results of microvascular decompression in trigeminal neuralgia and hemifacial spasm. **Neurosurgery** 40:46–52, 1997.

44. Kondziolka D, Lunsford LD, Flickinger JC, Young RF, Vermeulen S, Duma CM, Jacques DB, Rand RW, Regis J, Peragut JC, Manera L, Epstein MH, Lindquist C: Stereotactic radiosurgery for trigeminal neuralgia: A multi-institutional study using the gamma unit. **J Neurosurg** 84:940–945, 1996.

45. Kondziolka D, Lunsford LD, Flickinger JC: Stereotactic radiosurgery for the treatment of trigeminal neuralgia. **Clin J Pain** 18:42–47, 2002.

46. Lechin F, Van Der Dijs B, Amat J: Definite and sustained improvement with pimozide of two patients with severe trigeminal neuralgia. **J Med** 19:243–256, 1988.

47. Leksell L: Stereotaxic radiosurgery in trigeminal neuralgia. **Acta Chir Scand** 137:311–314, 1971.

48. Liao JJ, Cheng WC, Chang CN, et al: Reoperation for recurrent trigeminal neuralgia after microvascular decompression. **Surg Neurol** 47:562–570, 1997.

49. Lindstrom P, Lindblom U: The analgesic effect of tocainide in trigeminal neuralgia. **Pain** 28:45–50, 1987.

50. Lobato RD, Rivas JJ, Sarabia R, Lamas E: Percutaneous microcompression of the gasserian ganglion for trigeminal neuralgia. **J Neurosurg** 72:546–553, 1990.

51. Love S, Hilton DA, Coakham HB: Central demyelination of the Vth nerve root in trigeminal neuralgia associated with vascular compression. **Brain Pathol** 8:1–11, 1998.

52. Matsuda S, Serizawa T, Sato M, Ono J: Gamma knife radiosurgery for trigeminal neuralgia: the dry-eye complication. **J Neurosurg** 97:525–528, 2002.

53. Mendoza N, Illingworth RD: Trigeminal neuralgia treated by microvascular decompression: a long term follow-up study. **Br J Neurosurg** 9:13–19, 1995.

54. Meneses MS, Clemente R, Russ HHA, et al: Traitment microchirurgical de la névralgie du trijumeau: Etude sur 50 cases. **Neurochirurgie** 41:349–352, 1995.

55. Mittal B, Thomas DG: Controlled thermocoagulation in trigeminal neuralgia. **J Neurol Neurosurg Psychiatry** 49:932–936, 1986.

56. Mullan S, Lichtor T: Percutaneous microcompression of the trigeminal ganglion for trigeminal neuralgia. **J Neurosurg** 59:1007–1012, 1983.

57. Natarajan M: Percutaneous trigeminal ganglion balloon compression: Experience in 40 patients. **Neurol India** 48:330–332, 2000.

58. Nicol B, Regine WF, Courtney C, et al: Gamma knife radiosurgery using 90 Gy for trigeminal neuralgia. **J Neurosurg** 93:152–154, 2000.

59. Olafson RA, Rushton JG, Sayre GP: Trigeminal neuralgia in a patient with multiple sclerosis. An autopsy report. **J Neurosurg** 24:755–759, 1966.

60. Pagni CA. The origin of tic douloureux: A unified view. **J Neurosurg Sci** 37:185–194, 1993.

61. Pamir MN, Zirh TA, Ozer AF, et al: Microvascular decompression in the surgical management of trigeminal neuralgia. **Neurosurg Rev** 18:163–167, 1995.

62. Peragut JC, Gondin-Oliveira J, Fabrizi A, Sethian M: Microcompression of Gasser's ganglion: A treatment of essential facial neuralgia—Apropos of 70 cases. **Neurochirurgie** 37:111–114, 1991.

63. Pollock BE, Phuong LK, Foote RL, Stafford SL, Gorman DA: High-dose trigeminal neuralgia radiosurgery associated with increased risk of trigeminal nerve dysfunction. **Neurosurgery** 49:58–62; discussion 62–64, 2001.

64. Rappaport ZH, Devor M: Trigeminal neuralgia: the role of self sustaining discharge in the trigeminal ganglion. **Pain** 56:127–138, 1994.

65. Romanelli P, Heit G, Chang SD, Martin D, Pham C, Adler J: CyberKnife radiosurgery for trigeminal neuralgia. **Stereotact Funct Neurosurg** 81:105–109, 2003.

66. Rose WM: Removal of the gasserian ganglion for severe neuralgia. **Lancet** II:914, 1890.

67. Scaioli V, Franzini A, Leocata F, et al: Hand dystonia and neuralgic pain due to neurovascular contact to cervical spinal root [letter]. **Mov Disord** 11:102–104, 1996.

68. Shaya M, Jawahar A, Caldito G, Sin A, Willis BK, Nanda A: Gamma knife radiosurgery for trigeminal neuralgia: A study of predictors of success, efficacy, safety, and outcome at LSUHSC. **Surg Neurol** 61:529–534; discussion 534–535, 2004.

69. Sheldon CH, Pudenz RH, Freshwater DB, Crue BL: Compression rather than decompression for trigeminal neuralgia. **J Neurosurg** 12:123, 1955.

70. Siegfried J: Percutaneous controlled thermocoagulation of Gasserian ganglion in trigeminal neuralgia: Experiences with 1,000 cases, in Samii M, Jannetta PJ (eds): *The Cranial Nerves*. Berlin and Heidelberg, Germany, Springer-Verlag, 1981, pp 322–330.

71. Siegfried J: La nevralgie typique du trijumeau : Considerations cliniques, étiopatogeniques et therapeutiques. **Med Hyg** 41:1753–1757, 1984.

72. Siegfried J, Vosmansky M: Technique of the controlled thermocoagulation of trigeminal ganglion and spinal roots, in Krayenbuhl H (ed): *Advances and Technical Standards*. Berlin, Germany, and New York, Springer-Verlag, 1975, pp 199–209.

73. Sindou M, Amrani F, Mertens: Decompression vasculaire microchirurgicale pour nèvralgie du trijumeau: Comparaison de deux modalitès techniques et dèduction physiopathologiques—Etude sur 120 cas. **Neurochirurgie** 36:16–26, 1990.

74. Sindou M, Mertens P: Microsurgical vascular decompression in trigeminal and glosso-vago- pharyngeal neuralgias: A 20 year experience. **Acta Neurochir Suppl (Wien)** 58:168–170, 1993.

75. Skirving DJ, Dan NG: A 20-year review of percutaneous balloon compression of the trigeminal

ganglion. **J Neurosurg** 94: 913–917, 2001.

76. Sojqvist T: Eine neue operations methode bei trigeminus-neuralgie: Durchscheneidung tractus spinalis trigemini. **Zbl Neurochir** 5:247, 1937.

77. Spiller WG, Frazier CH: The division of the sensory root of the trigeminus for relief of tic douloureux; an experimental, pathological, and clinical study, with a preliminary report of one surgically successful case. **University of Pennsylvania Medical Bulletin** 14:342–352, 1901, and **Philadelphia Medical and Physical Journal** 8:1039–1049, 1901.

78. Sun T, Saito S, Nakai O, et al: Long-term results of microvascular decompression for trigeminal neuralgia with reference to probability of recurrence. **Acta Neurochir (Wien)** 126:144–148, 1994.

79. Sweet WH, Wepsic JG: Controlled thermocoagulation of trigeminal ganglion and rootlets for differential destruction of pain fibers. **J Neurosurg** 40:143–156, 1974.

80. Szapiro J, Sindou M, Szapiro J: Prognostic factors in microvascular decompression for trigeminal neuralgia. **Neurosurgery** 17:920–929, 1985.

81. Taarnhoj P: Decompression of the trigeminal root and the posterior part of the ganglion as a treatment in trigeminal neuralgia; preliminary communication. **Neurosurgery** 9:288–290, 1952.

82. Taarnhoj P: Decompression of the posterior trigeminal root in trigeminal neuralgia: A 30-year follow-up review. **J Neurosurg** 57:14–17, 1982.

83. Taha JM, Tew JM: Comparison of surgical treatments for trigeminal neuralgia: reevaluation of radiofrequency rhizotomy. **Neurosurgery** 38:865–871, 1996.

84. Taptas N: Les injections d’alcool dans le ganglion de Gasser travers le trou ovale. **Presse Med** 19:798, 1911.

85. Tew JM Jr, Keller JT: The treatment of trigeminal neuralgia by percutaneous radiofrequency technique. **Clin Neurosurg** 24:557–578, 1977.

86. Vilming ST, Lyberg T, Lastate X: Tizanidine in the management of trigeminal neuralgia. **Cephalalgia** 6:181–2, 1986.

87. Waxman SG, Ritchie JM: Hyperexcitability of pathologically myelinated axons and positive symptoms in multiple sclerosis, in Stephen G, Waxman SG, Ritchie JM (eds), *Demyelinating Diseases, Basic and Clinical Electrophysiology*. New York, Raven Press, 1981, pp 289–297.

88. Yamaki T, Hashi K, Niwa J, et al: Results of reoperation for failed microvascular decompression. **Acta Neurochir (Wien)** 115:1–7, 1992.

89. Zheng LG, Xu DS, Kang CS, Zhang ZY, Li YH, Zhang YP, Liu D, Jia Q: Stereotactic radiosurgery for primary trigeminal neuralgia using the Leksell Gamma unit. **Stereotact Funct Neurosurg** 76:29–35, 2001.

90. Zorman G, Wilson CB: Outcome following microvascular decompression or partial sensory rhizotomy in 125 cases of trigeminal neuralgia. **Neurology** 34:1362–1365, 1984.

FIG. 37.1 Graph illustrating the cumulative proportion of completely pain-free patients. *A*, idiopathic TN. *B*, TN in patients with MS.

FIG. 37.2 Patient's positioning and skin incision.

FIG. 37.3 Craniectomy.

FIG. 37.4 Fifth cranial nerve exposed through a supracerebellar approach avoiding the use of retractors. A neurovascular conflict impinging the trigeminal root entry zone is shown.

FIG. 37.5 See trigeminal nerve atrophy observed during MVD in one patient who underwent previous TRZ.

FIG. 37.6 Percutaneous balloon microcompression (PMC).

FIG. 37.7 Treatment plan with CyberKnife: dose distribution. (Courtesy of Dr. Fariselli, Radiosurgical Center, INNCB, Milan, Italy.)