

Microvascular decompression for primary trigeminal neuralgia: long-term effectiveness and prognostic factors in a series of 362 consecutive patients with clear-cut neurovascular conflicts who underwent pure decompression

MARC SINDOU, M.D., PH.D.,¹ JOSÉ LESTON, M.D., M.Sc.,¹ EVELYNE DECULLIER, PH.D.,²
AND FRANÇOIS CHAPUIS, M.D., PH.D.²

¹Department of Neurosurgery, Hôpital Neurologique “Pierre Wertheimer,” University of Lyon; and ²Department of Medical Information and Clinical Epidemiology Resources, Hospices Civils de Lyon, University of Lyon, France

Object. The purpose of this study was to evaluate the long-term efficacy of microvascular decompression (MVD) and to identify the factors affecting outcome in patients treated for primary trigeminal neuralgia (TN). Only the cases with a clear-cut neurovascular conflict (vascular contact and/or compression of the root entry zone of the trigeminal nerve) found at surgery and treated with “pure” MVD (decompression of the root without any additional lesioning or cutting of the adjacent rootlets) were retained.

Methods. The study included 362 patients who were followed up over a period of 1 to 18 years (median follow-up 7.2 years). A Kaplan–Meier survival analysis was generated at 1 and 15 years of follow-up for all of the considered factors. According to Kaplan–Meier analysis, the success rate (defined as pain-free patients without any medication) was 91% at 1 year and estimated to be 73.38% after 15 years of follow-up.

Results. None of the following patient-related factors played any significant role in prognosis: sex, patient age at surgery, history of systemic hypertension, duration of neuralgia before surgery, or history of failed trigeminal surgery. Patients with atypical neuralgia (a baseline of permanent pain) had the same outcome as those with a typical (purely spasmodic) presentation. In addition, the side and topography of the trigeminal nerve did not play a role, whereas involvement of all three divisions of the nerve had a negative effect on outcome. Concerning anatomical factors, neither the type of the compressive vessel nor its location along or around the root was found to be significant. However, the severity of compression was important—the more severe the degree of compression, the better the outcome ($p = 0.002$). The authors also found that presence of focal arachnoiditis had a negative influence on outcome ($p = 0.002$).

Conclusions. Pure MVD can offer patients affected by a primary TN a 73.38% probability of long-term (15 years) cure of neuralgia. The presence of a clear-cut and marked vascular compression at surgery (and possibly—although not yet reliably—on preoperative magnetic resonance imaging) is the guarantee of a higher than 90% success rate. (DOI: 10.3171/JNS-07/12/1144)

KEY WORDS • Kaplan–Meier analysis • microvascular decompression • neurovascular compression • outcome • trigeminal neuralgia

BASED on the hypothesis that vascular compression is the cause of most cases of so-called “primary” TN,^{11–13,15} MVD^{16,17} has become widely accepted as a logical and valuable method for curing the disease. However, MVD is not always successful; failure rates of 15 to 35% have been reported in the literature.^{4,5,37,48,50} We therefore chose to undertake this study to investigate the factors influencing outcome after MVD to improve patient selection and surgical strategies. Most publications on outcome have considered prognosis, but only a few have focused on

a detailed analysis of the spectrum of prognostic factors.^{4,17,22,44} Furthermore, in some reports, not all patients had clear-cut vascular compression, and not all underwent “pure” vascular decompression, that is, without additional lesioning of adjacent rootlets.

The aim of this study was to assess the long-term effectiveness of pure MVD on neuralgia and to identify the main factors affecting prognosis. The present study was conducted in a series of 362 consecutive patients treated between 1983 and 1999, who all had clear-cut vascular compression and were all treated with pure MVD. All patients underwent follow-up for longer than 1 year (range 1–18 years), with an average follow-up of 8 years and a median of 7.2 years.

Abbreviations used in this paper: MVD = microvascular decompression; NVC = neurovascular conflict; TN = trigeminal neuralgia.

Clinical Material and Methods

Because our aim was to evaluate the effectiveness of pure MVD, only patients with clear-cut NVCs observed at surgery who underwent decompression without any additional lesioning to the rootlets were included in the study.

Patient Selection

Between 1983 and 1999, 2232 patients with a diagnosis of refractory TN (Fig. 1) were referred to our institution for surgery. The patients retained for the present study were only those with primary TNs (either typical or atypical) and clear-cut vascular compression of the root found at surgery. All included patients had to undergo “pure” MVD (without any additional cutting or coagulation of the adjacent rootlets) and attend follow-up for at least 1 year postoperatively.

Of the 448 patients who underwent posterior fossa exploration, 86 patients were excluded. Forty patients were not retained (despite having received sufficient follow-up) because of a lack of important information in their operative record or initial chart, specifically a sufficiently precise description of their trigeminal pain or of the anatomical findings at surgery. The percentage of patients with pain relief in this group was not different from that of the selected patient group. In 23 patients with vertebrobasilar arterial compression, decompression was incomplete and associated with a partial sectioning or coagulation of the adjacent rootlets. Of these 23 patients, 19 achieved lasting pain relief but continued to have some degree of numbness and paresthesia. In 21 patients, no vascular compression was found (4.7% of the overall series). In these cases, no lesioning of the root was done to investigate the cause, and surgery consisted of simply freeing the surrounding arachnoid mater. All patients in this group experienced persistence or early recurrence of their pain and required a subsequent thermorhizotomy. Two patients were excluded because they died of hemorrhagic cerebellar infarction within the first months after surgery and thus could not receive sufficient follow-up.

Assessment of the Effect of MVD on Pain

All 362 patients were assessed in person by the senior surgeon (M.S.) at discharge (generally postoperative Day 10), at the first outpatient visit (usually around Day 70 postoperatively), and then at 1 year. In addition, at 1 year of follow-up, all 362 patients attended independent outpatient visits with their referring neurologists. There were no patients lost to follow-up after 1 year.

For this study (completed in 2001), all patients, or families of patients who had died, were contacted by mail (or by telephone if there was no response) for an interview to answer the study questionnaire. This survey was performed by an independent observer.²⁷ The surviving 337 patients were asked to confirm the cure or whether pain persisted or recurred, to describe the residual and/or new pain, and to explain how this pain was controlled. For the 25 patients who died, relatives and the family doctor were asked to provide the necessary information.

Neuralgia was considered cured—and consequently the MVD a success—when relief was complete and all medication could be withdrawn. The result was considered a failure when pain persisted in any form, either spasmodic

or constant aching pain, even when no medical therapy was necessary.

Pain was considered recurrent when, after an initial success, the patient reported trigeminal pain again, necessitating resumption of medication, and if this medical therapy was not sufficient, underwent a lesioning procedure (usually a percutaneous thermorhizotomy in our department).

Failures and recurrences were listed according to when they occurred and are presented in a Kaplan–Meier survival curve at 1 and 15 years (Fig. 2).

Prognostic Factors

The prognostic factors considered were classified into the following groups: patient-related factors, neuralgia-related factors, and anatomical findings. The patient-related factors included sex, age at surgery, history of systemic hypertension, duration of neuralgia before surgery, and role of previous failed surgical procedures. It is important to mention, for the latter factor, that the patients who had undergone destructive surgery for TN that led to neuropathic pain and/or produced a significant degree of hypesthesia were not retained for undergoing MVD. The factors related to neuralgia included operated side, topography of the trigeminal nerve, extent of surgery, and clinical presentation of pain (typical or atypical). The factors related to intraoperative anatomical findings included type of the compressing vessels, NVC severity, location along the length of the root and around the circumference of the root, the presence of global atrophy of the root, and the presence of focal arachnoiditis adhesive to the root.

Correlations between those factors and outcomes were then calculated, first at 1 year of follow-up for all patients, and then at 15 years through a Kaplan–Meier analysis. Correlations at the latest follow-up visits were not studied because of variability of follow-up length among patients.

Statistical Analysis

Data analysis was performed using commercially available software (SAS). Results were expressed as percentages for qualitative data and means for quantitative data. Comparisons were performed using the chi-square test.

The time between surgery and failure was calculated, and survival time was limited to 15 years. Patients with no failure were censored at the date of latest follow-up. A Kaplan–Meier survival analysis was performed and log-rank tests were used to compare survival curves. Risk factors for failure were evaluated using the Cox proportional hazard method. Probability values less than 0.05 were considered statistically significant.

Results

In all 362 patients retained in the study, the pain was neuralgic and of primary origin. The neuralgic nature was attested by the fact that the pain responded well initially to anticonvulsant agents in all patients. The primary origin was confirmed by the fact that imaging results were nondiagnostic with the exception of the eventual demonstration of vessel (or vessels) in close contact with the trigeminal root. Classification of the neuralgia into typical or atypical—sometimes difficult to establish—was done independently by the senior neurosurgeon (M.S.) together with the

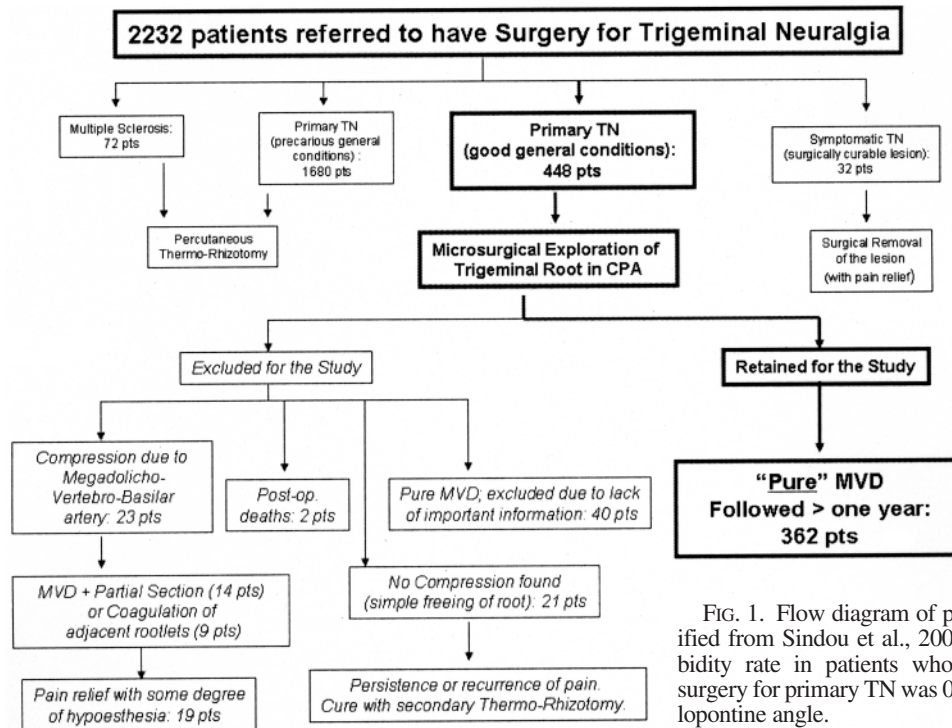


FIG. 1. Flow diagram of patient selection, modified from Sindou et al., 2006. Note that the morbidity rate in patients who had posterior fossa surgery for primary TN was 0.44%. CPA = cerebellopontine angle.

referring neurologist at the preoperative examination, in accordance with the descriptions given in the classical handbooks on pain surgery by William Sweet, the senior author's mentor.^{41,42} Typical TN was defined as characterized by paroxysms of electrical, shooting pain; atypical TN was defined as paroxysmal pain (at least at the beginning of the disease) but later accompanied by a constant deep aching or burning pain.

In all included patients, pain had become refractory to medical treatment as directed by the patient's neurologist, including anticonvulsant agents,⁹ and general health conditions were estimated as acceptable for open surgery by the neuroanesthesiologist. All patients were informed of the risks and gave informed consent for MVD.

All the retained patients underwent pure MVDs. In all cases, surgery was performed by the same neurosurgeon (M.S.) using classical microsurgical techniques with some personal modifications so that the decompression would be as atraumatic as possible, avoiding a new compression of the root with the prosthesis. The technique has already been described in detail in previous publications.^{31-34,37} In summary, maximum care was taken to: 1) explore the whole root from the porus to the trigeminal root entry zone so as not to miss multiple NVCs and to free the entire root from all arachnoid adhesions, while allowing a straight trajectory for the root without angulations, kinking, or twisting; 2) decompress the nerve with minimal—if any—manipulation; and 3) keep the compressive vessel apart from the nerve by means of a prosthesis, which was not in contact with the root (or at least did not compress it). Decompression was performed without any prosthesis contact with the root in 249 patients (71.1%). It was impossible to avoid allowing the prosthesis to touch the root in the 101 other patients (28.9%).

Characteristics of the Series

Patient Demographics. Patient age at surgery ranged from 28 to 84 years (average 61 years); 109 patients (30.1%) were younger than 50 years of age, 231 patients (63.8%) were between 50 and 70 years of age, and 22 patients (6.1%) were 70 years or older. One hundred seventy-two patients (47.5%) were men, and 190 (52.5%) were women. In 97 patients (26.8%) there was a history of systemic hypertension. The duration of symptoms before surgery ranged from 1 to 36 years (average 6.4 years). Sixty patients had had previous surgery for neuralgia as follows: 42 patients (11.6%) had had one or several thermorhizotomies, 13 (3.6%) had undergone one or several balloon compression procedures, and five (1.4%) had undergone an unsuccessful MVD at another institution. All 60 patients who had undergone surgery previously reported residual or recurrent neuralgic pain (with electric, shooting paroxysms). The clinical presentation was purely typical in 41 patients and atypical in the other 19 patients.

Neuralgic Pain. There were 224 patients (61.9%) with pain on the right side and 138 (38.1%) with pain on the left side. In 10 patients (2.7%) only the ophthalmic division (V1) was affected, in 77 patients (21.3%) only the maxillary division (V2) was affected, and in 55 patients (15.2%) only the mandibular division (V3) was affected. In 80 patients (22.1%) both the V1 and V2 divisions were affected, in 111 patients (30.7%) both V2 and V3 were affected, and in 29 patients (8%) all three divisions were affected. The number of divisions involved were as follows: only one division in 140 patients (38.7%), two in 193 patients (53.3%), and all three divisions in 29 patients (8%). The clinical presentation of neuralgia was only of the paroxysmal type and was therefore considered strictly typical in

Microvascular decompression for trigeminal neuralgia

237 patients (65.5%). In the other 125 patients (34.5%) neuralgia was atypical because the paroxysmal crises were accompanied by a baseline of predominantly aching/burning pain in 69 patients (19.1%), by vasomotor manifestations in 38 patients (10.5%), and by both in 18 patients (4.9%).

Anatomical Findings. The entire trigeminal root from the porus of Meckel cave to the root entry zone at the pons was explored under the microscope. The anatomical data collected included the types of the compressing vessels, NVC severity, and NVC location in relation to the nerve root. Eventual (global) root alterations and surrounding abnormalities were noted. Because detailed descriptions have been given in previous publications,^{35,36} anatomical data will only be summarized here. Multiple compressing vessels were frequently found in the same individual. The superior cerebellar artery alone was involved in 269 patients (74.3%), the anteroinferior cerebellar artery alone in 22 patients (6.1%), and both in 59 patients (16.3%); there were arterial NVCs in 350 patients (96.7%). A vein was found more or less embedded in the nerve in 96 cases (26.5%), 84 of which were associated with an arterial compressive vessel, and only 12 (3.3%) of which were exclusively venous. The degree of NVC severity was established in the 350 cases with arterial compression; when several compressing vessels were found, the major conflict alone was considered. Severity was rated on a degree scale of I through III. In the 48 patients (13.3%) with Degree I severity, the vessel was in contact with the root without any visible indentation to the root. In the 184 patients (50.8%) with Degree II severity, there was displacement and/or distortion of the root, and in 118 patients (32.6%) with Degree III severity there was a marked indentation in the root.

The NVC location along the root was noted for all clear-cut arterial and venous conflicts, either alone or as a component of several conflicts in the same patient. The location was noted as juxtapontine when the NVC was located at the trigeminal root entry zone, at the brainstem or less than 5 mm from the entry into the pons; midcisternal when the NVC was at the middle third of the root; and juxtapetrous when it was where the root exists from the porus of Meckel cave. Arterial NVCs (in 350 patients) were juxtapontine in 190 patients (54.3%), midcisternal in 141 patients (40.3%), and juxtapetrous in 19 patients (5.4%). Venous NVCs (in 96 patients) were juxtapontine in 27 patients (28.1%), midcisternal in 41 patients (42.7%), and juxtapetrous in 28 patients (29.2%).

As for the location of the NVCs around the circumference of the root, only the 350 cases with arterial NVCs were retained, and if several conflicting vessels were found in the same patient, only the major conflict was considered. The NVC location was superomedial to the root in 214 patients (61%), superolateral in 98 patients (28%), and inferior in 38 patients (11%).

Besides eventual segmental indentations on the nerve at the sites of the compressing vessels, alterations in the entire trigeminal root were frequently observed. In 95 patients (26.3%) the nerve had a significant degree of global atrophy such that the caliber of the nerve was diminished by one-third or more. In 48 patients (13.2%) the atrophy was considerably marked, with a caliber reduction to approximately two-thirds of the normal size. The nerve appeared

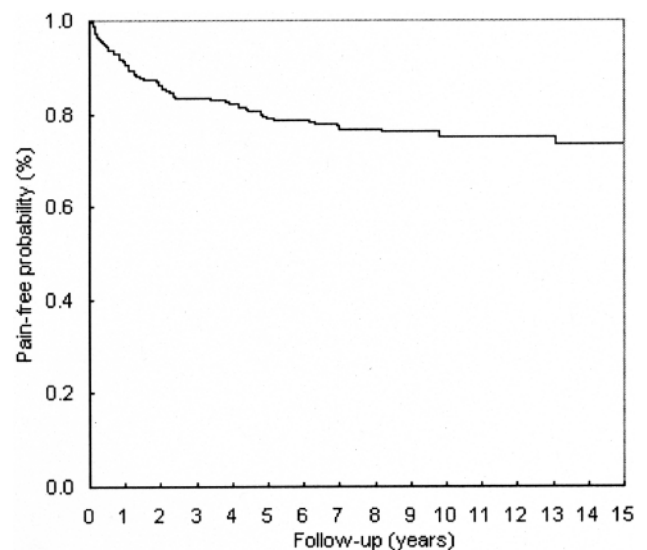
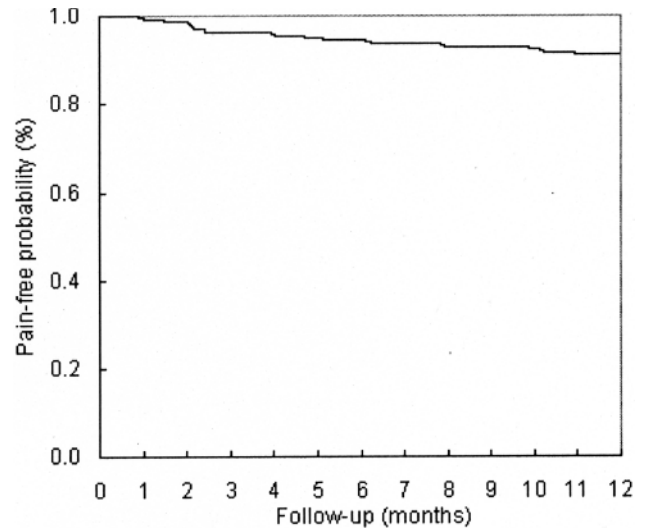


FIG. 2. Graphs of global results of pain-free patients at 1 year (upper) and at 15 years (lower). Note that patients in whom paroxysmal pain was relieved but who continued to experience some background pain even though no medication was required were considered to have had failed surgery.

ribbon-shaped and grayish in color. Among the 42 patients who had previously undergone thermorhizotomy, 26 had marked atrophy. Thus, there were 66 patients (18.2%) who had not had any previous surgery and in whom the trigeminal nerve had atrophy. Thickening of the arachnoid mater in the vicinity of the root was found in 83 patients (22.9%), with strong adhesions to the nerve (considered severe) in 46 patients (12.7%).

Follow-Up. Some patients experienced complications resulting in permanent disturbances or deficits. One patient (0.27%) had severe gait disturbances due to cerebellar/brainstem ischemia that was related to excessive surgical manipulation and vasospasm of the superior cerebellar artery. Three patients (0.83%) had diplopia due to trochlear nerve palsy, three (0.83%) had facial nerve palsy, and seven (1.9%) had hearing loss. Eleven patients (3.04%) com-

plained of permanent trigeminal nerve sensory disturbances (paresthesias, dysesthesias, and/or hemifacial numbness). Among the overall series of 448 patients who underwent trigeminal root exploration in the cerebellopontine angle, two patients died (0.44%) of hemorrhagic cerebellar infarctions.

All patients were followed up for at least 1 year. In the 337 who were still alive, follow-up continued until the study ended in 2001 and until death in the 25 patients who died of concurrent disease or “advanced age” before the end of the study. Follow-up data are summarized in Table 1. The results are presented at discharge, after the first outpatient visit, and at 1 year of follow-up for all of the patients. The Kaplan–Meier curves are then given at 1 and 15 years of follow-up.

Pain Relief Over Time, up to 1 Year

At discharge, on the 10th day, 310 patients (85.6%) were pain-free. Eleven patients (3%) still had some background of pain; these patients were not considered pain-free although their residual pain did not necessitate medical treatment. In 41 patients (11.4%) the procedure was considered a failure because of the persistence of paroxysms necessitating medical therapy. In these patients, paroxysmal pain was pure in 37 and associated with a background of deep pain in four patients.

At the outpatient visit on Day 70 postoperatively, 300 patients were pain-free—a success rate of 83.1%. Eighteen patients (4.9%) still had some (bearable) background of pain. In 44 patients, surgery failed (12.2%), and paroxysmal pain persisted as the sole component in 40 patients, with a background of deep pain in the four others.

At the 1-year postoperative visit, the success rate was 81.2%, corresponding to 294 totally pain-free patients. Thirteen patients (3.6%) had a bearable background of pain. Failures amounted to 15.2% (55 patients) with persistent paroxysmal pain—pure in 50 and associated background pain in five.

Kaplan–Meier Analysis of Outcome

The Kaplan–Meier curves of pain-free patients have been established. For the calculation, the patients with only a residual background of pain were considered together with the cases considered true failures. At the 1-year follow-up examination, an estimated 91% of patients were pain-free. The corresponding curve is presented in Fig. 2 upper.

Because “latest follow-up” was very different in length from one patient to another (range 1–18 years), the long-term effect of MVD on pain (at 15 years of follow-up) was evaluated by calculating a Kaplan–Meier survival curve.

The percentage of patients with the probability of being totally pain-free at 15 years was established at 73.38%. The curve is presented in Fig. 2 lower, and the corresponding data in Table 1.

Prognostic Factors

A study of prognostic factors was performed by correlating selected factors and postsurgical outcomes after 1 and 15 years of follow-up. The results are detailed in Tables 2, 3, and 4 and summarized in the next section.

Patient Characteristics. The success rate was not significantly different between male and female patients either at 1 or 15 years of follow-up. Analysis of data concerning patient age at the time of surgery demonstrated that the older the patient, the higher the success rate at 1 and 15 years postoperatively (Fig. 3). However, the probability value calculation for this data showed only a statistical tendency ($p = 0.09$) at both follow-ups. Patient history of systemic hypertension, duration of preoperative neuralgia (Fig. 4), or history of previous failed trigeminal surgery did not make the MVD significantly less effective or influence prognosis.

Neuralgia-Related Factors. The side (right or left) of neuralgia had no influence on the outcome. When the V1 division was involved, the success rate at 15 years was better than when V1 was not involved (84% compared with 72.7% for pure V2, and 71.1% for V3 only and V2 and V3). However, this finding did not reach statistical significance ($p = 0.19$).

When the neuralgia included all three trigeminal divisions the outcome was worse—75.9% success at 1 year (compared with 93.3 and 91.4% for neuralgia involving two and one divisions, respectively) and 69% at 15 years (compared with 81.9 and 75.7%, respectively). This difference was significant at 1 year ($p = 0.007$) but not at 15 years ($p = 0.11$).

Clinical presentation did not appear to play a role in prognosis. Patients with atypically manifested neuralgia had approximately the same outcome as those with typical neuralgia at both 1 year and 15 years of follow-up. This finding did not reach statistical significance.

Factors Related to Anatomical Findings. The type of compressing vessels did not affect the prognosis after either 1 or 15 years of follow-up ($p = 0.44$ and 0.43 , respectively). However, a lower success rate was recorded for pure venous compression over arterial compression—66.7% at 15 years for venous compression compared with 81% for arterial compression. This finding was not statistically significant. The location of the NVC along and around the root did not influence the outcome. The degree of severity of

TABLE 1
Summary of follow-up data*

Patient Characteristic	Time Since Op												
	0 Days	10 Days	70 Days	1 Yr	2 Yrs	4 Yrs	6 Yrs	8 Yrs	10 Yrs	12 Yrs	14 Yrs	15 Yrs	
pain free	362	310	300	294	285	234	172	121	84	55	24	14	
w/ failure	0	52	62	68	49	62	71	75	77	77	78	78	
LTF	0	0	0	0	28	66	119	166	201	230	260	270	

* LTF = lost to follow-up.

Microvascular decompression for trigeminal neuralgia

TABLE 2
Summary of patient characteristics

Characteristic (no. of patients)	No. of Patients w/ Success at 1 Yr (%)	p Value	Estimated	
			No. of Patients w/ Success at 15 Yrs (%)	p Value
sex		0.93		0.41
M (172)	157 (91.3)		139 (80.8)	
F (190)	173 (91.1)		145 (76.3)	
age at op		0.09*		0.09*
<50 yrs (58)	49 (84.5)		40 (69)	
50–70 yrs (252)	231 (91.7)		199 (79)	
>70 yrs (52)	50 (96.2)		45 (86.5)	
history of hypertension		0.29		0.52
present (265)	239 (90.2)		205 (77.4)	
absent (97)	91 (93.8)		79 (81.4)	
symptom duration before MVD		0.62		0.67
<2 yrs (60)	54 (90.0)		44 (73.3)	
2–6 yrs (184)	166 (90.2)		148 (80.4)	
>6 yrs (118)	110 (93.2)		92 (78)	
previous (failed) op		0.78		0.54
no op (302)	275 (91.1)		236 (78.1)	
op (60)	55 (91.6)		48 (80)	

* Although not statistically significant, these values indicate a trend.

the compression proved significant with respect to the outcome. The more severe the degree of preoperative compression, the better the outcome at 1 year ($p = 0.002$) and at 15 years of follow-up ($p = 0.001$) (Fig. 5). At 1 and 15 years of follow-up, patients with Degree III compression had cure rates of 96.6 and 88.1%, respectively. Patients with Degree II compression had 90.2 and 78.3% cure rates, and those with Degree I compression had 83.3 and 58.3% cure rates.

Neither global atrophy of the root (Fig. 6) nor the presence of local arachnoiditis adhesive to the root had an effect on the prognosis at 1 year postoperatively ($p = 0.77$ and 0.23 , respectively). However, over the long term, the presence of severe arachnoiditis was found to have a significant negative effect, with a success rate of only 58.7% at 15 years ($p = 0.002$) (Fig. 7); atrophy did not have a significant negative effect (15-year success rate 72.9%, $p = 0.36$) (Fig. 5).

Discussion

The purpose of this study was to identify the factors likely to play a role in the long-term effectiveness of MVD when clear-cut vascular compression is found at surgery and a pure decompression performed.

Long-Term Effect on Pain

The effect of MVD on pain was evaluated with a questionnaire that we set up in 1983 and revised in 2000, before the recommendations and Zakrzewska's questionnaire to assess patient satisfaction after trigeminal surgery^{49,51} were published. According to a Kaplan–Meier analysis of our data, a successful outcome (defined as complete pain relief of both paroxysms and deep background pain, and total withdrawal of medications) was achieved in 73.3% of patients at 15 years. In addition, we noticed a rather good stability of pain relief after 1 year of follow-up.

We recently published a detailed analysis of the progress

TABLE 3
Summary of neuralgia-related factors

Factor (no. of patients)	No. of Patients w/ Success at 1 Yr (%)	p Value	Estimated	
			No. of Patients w/ Success at 15 Yrs (%)	p Value
op side		0.64		0.78
rt (224)	203 (90.6)		174 (77.7)	
lt (138)	127 (92)		110 (79.7)	
topography		0.82		0.19
all V1 (119)	110 (92.4)		100 (84)	
pure V2 (77)	70 (90.9)		56 (72.7)	
V3 & V2 + V3 (166)	150 (90.4)		128 (71.1)	
extent		0.007*		0.11
1 division (140)	128 (91.4)		106 (75.7)	
2 divisions (193)	180 (93.3)		158 (81.9)	
3 divisions (29)	22 (75.9)		20 (69)	
clinical presentation		0.25		0.98
typical (237)	219 (92.4)		187 (78.9)	
atypical (125)	111 (88.8)		97 (77.6)	

* Highly significant.

in our 362 patients up to their last follow-up.³⁷ Among the patients who were pain-free at their 3-month postoperative visit, 17% had recurrence of neuralgia. Conversely, 13% of patients who continued to suffer neuralgic pain at 3 months postsurgery showed a late cure. The possibility of a late cure after MVD for TN has rarely been reported in litera-

TABLE 4
*Summary of anatomical findings**

Factor (no. of patients)	No. of Patients w/ Success at 1 Yr (%)	p Value	Estimated	
			No. of Patients w/ Success at 15 Yrs (%)	p Value
compressing vessel		0.44		0.43
SCA (269)	245 (91.1)		208 (77.3)	
AICA (22)	19 (86.4)		18 (81.8)	
SCA + AICA (59)	56 (94.9)		50 (84.7)	
pure venous (12)	10 (83.3)		8 (66.7)	
NVC severity		0.002†		0.001†
I (48)	40 (83.3)		28 (58.3)	
II (184)	166 (90.2)		144 (78.3)	
III (118)	114 (96.6)		104 (88.1)	
site of NVC along root		0.37		0.24
TREZ (190)	174 (91.6)		149 (78.4)	
midcisternal (141)	127 (90.1)		109 (77.3)	
juxtapesitrous (19)	19 (100)		18 (94.7)	
NVC location around root		0.47		0.15
superomed (214)	198 (92.5)		174 (81.3)	
superolat (98)	89 (90.8)		70 (71.4)	
inferior (38)	33 (86.8)		32 (84.2)	
root atrophy		0.77		0.36
absent (267)	243 (91)		213 (79.8)	
mild (47)	44 (93.6)		36 (76.6)	
severe (48)	43 (89.6)		35 (72.9)	
arachnoiditis		0.23		0.002†
absent (279)	256 (91.8)		226 (81)	
mild (37)	35 (94.6)		31 (83.8)	
severe (46)	39 (84.8)		27 (58.7)	

* AICA = anteroinferior cerebellar artery; SCA = superior cerebellar artery; superolat = superolateral; superomed = superomedial; TREZ = trigeminal root entry zone.

† Indicates a high level of statistical significance.

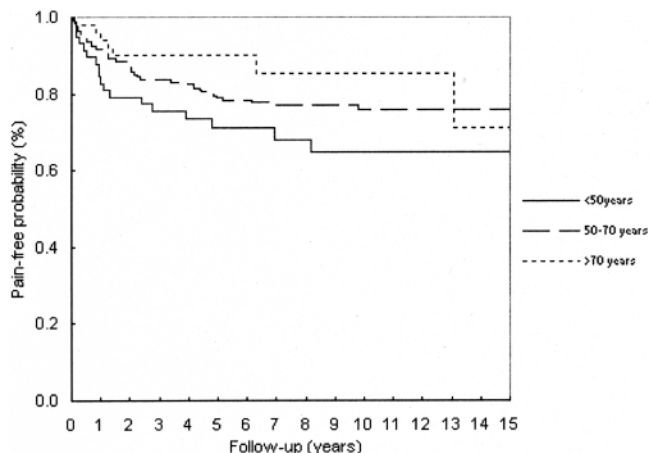


FIG. 3. Kaplan–Meier curve (at 15 years of follow-up) of pain-free patients according to age at time of surgery. Outcome was better in older patients than in younger ones. This difference is not statistically significant, but does demonstrate a trend ($p = 0.09$).

ture, in contrast to MVD for hemifacial spasm.³⁸ Failures after MVD, whether initial or as a recurrence, reached 15.1% at the latest follow-up (1–18 years, 8 years on average). Patients with recurrence were treated with medication, and if that proved insufficient, some were referred for a secondary surgery (thermorhizotomy at our institute).

The 73.38% probability of total cure at 15 years by pure MVD in the present study is well within the range of other published results, which give a 63 to 86% success rate at 5 years.^{1,7,10,18–23,25,26,29,30,39,40,44–48,50,52} In the largest reported series of 1204 patients, 69.6% of the patients had excellent results 10 years after MVD.⁴ In the other publications with Kaplan–Meier analyses, such as those by Tronnier et al.,⁴⁵ Zakrzewska and colleagues,⁵⁰ Broggi et al.,⁷ and Bederson and Wilson,⁵ cure rates were 63% at 10.9 years, 84% at 5 years, 84.7% at 3.2 years, and 75% at 5.1 years, respectively.

Prognostic Factors in the Literature

A review of the literature does not bring to light unani-

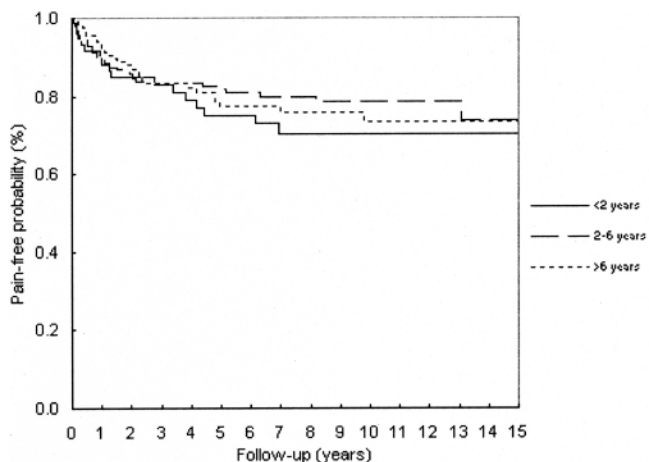


FIG. 4. Kaplan–Meier curve (at 15 years of follow-up) of pain-free patients according to preoperative duration of neuralgia. No statistical significance was found ($p = 0.67$).

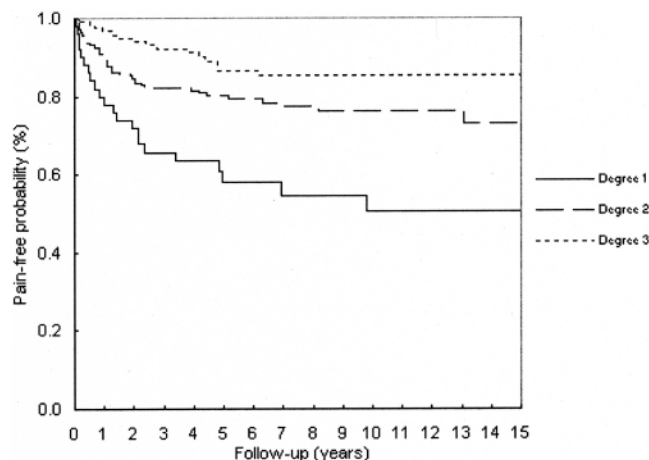


FIG. 5. Kaplan–Meier curve (at 15 years of follow-up) of pain-free patients according to degree of compression; the more severe the degree, the better the outcome ($p = 0.001$).

mously recognized factors that affect outcome in patients after MVD. There are many reasons for this. Some publications dealt only with a small number of patients and/or did not make statistical calculations; others only studied a limited number of factors. Furthermore—and puzzlingly enough—the comparison between large series, even those with statistical analysis, shows contradictory results.

Women were noted to have a higher rate of failures or recurrences in some studies^{5,17,19,43,44} but not in others.^{14,18,39,40,48} Pain on the left side in men was found to account for a higher degree of recurrence.⁴⁴ Young age at the time of surgery was found to have a negative effect on outcome in the study by Theodosopoulos et al.⁴⁴ ($p < 0.01$), whereas this was not the case in other studies.^{39,40} A longer duration of symptoms before surgery had a harmful influence according to a wide number of authors,^{1,3,5,17,19,22,28,44} but several others have reported no influence.^{18,26,39,40,48} A previous failed destructive procedure at the trigeminal nerve was reported as unfavorable by several authors,^{3,9,24,28,39,43} but was not of any significance in the studies by Theodosopoulos et al.,⁴⁴ Jannetta,¹⁷ and Tronnier and associates,⁴⁵ at least for the tic-like component of pain. The distribution and number of trigeminal divisions affected by neuralgia was not significant in the prognosis in any series studied.^{5,6,17,39,40,44}

Atypical manifestations added to paroxysmal pain in the clinical presentation had a significant negative effect in the report of Li et al.²² in 62 patients ($p < 0.01$).

With regard to operative findings, arterial conflicts were found to be associated with a better outcome compared with venous conflicts in most series studied.^{9,14,17,18,22,40}

The role of the degree of compression on the outcome was diversely appreciated. Severe compression (“grooving”) of the nerve was found to correlate with a better outcome,^{9,40} a worse outcome,²² or to not play any role in outcome.⁴⁴ This factor was not examined in the large series by Jannetta.¹⁷

Prognostic Factors in the Current Study

Data from our study, especially results of the Kaplan–Meier analysis at 15 years of follow-up, have to be careful-

Microvascular decompression for trigeminal neuralgia

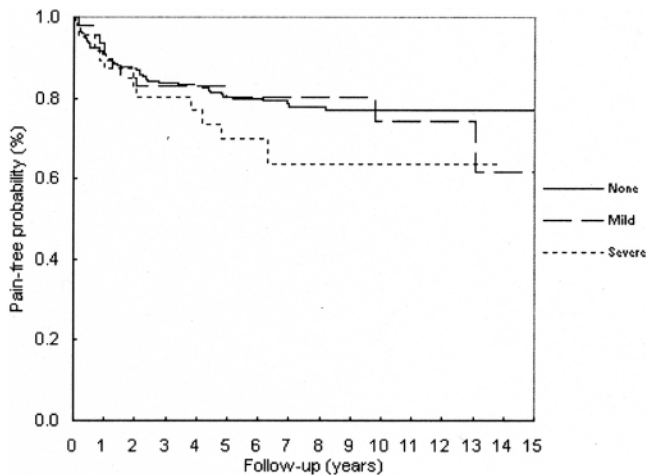


FIG. 6. Kaplan–Meier curve (at 15 years of follow-up) of pain-free patients according to degree of atrophy. Curve shows a worse outcome with severe atrophy; however, the difference is not statistically significant ($p = 0.36$).

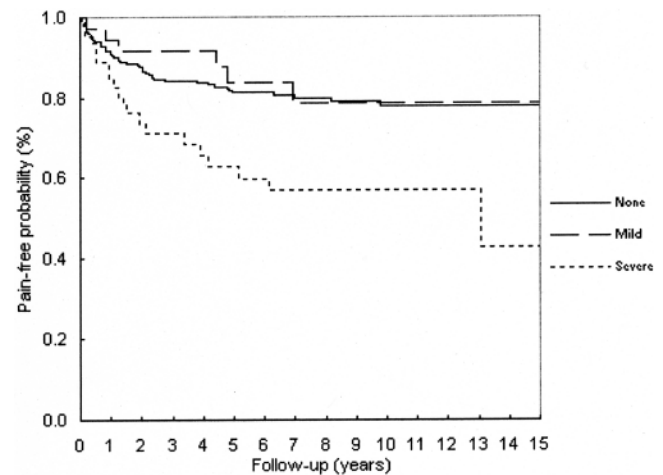


FIG. 7. Kaplan–Meier curve (at 15 years of follow-up) of pain-free patients according to degree of arachnoiditis. Severe arachnoiditis was revealed to have a significant negative effect on outcome (in the long term only; $p = 0.002$).

ly discussed because their consideration may have practical consequences. With regard to patient age at surgery, we found that older patients had a better outcome than younger ones (Table 2). Although this finding was not statistically significant ($p = 0.09$), these figures plead for not excluding elderly people from MVD if other conditions are good (Fig. 3). Amazingly, the duration of neuralgia before MVD did not influence the outcome at all ($p = 0.67$); therefore, the argument that delaying surgery could compromise its effectiveness does not seem justified (Fig. 4).

We also found that patients who had undergone a previous failed surgery did not have significantly worse effects after MVD, at least when paroxysmal pain was the main component of persistent neuralgia ($p = 0.54$). This should not, however, be used as an argument for using MVD as a second choice after lesioning procedures have failed, because any sensory deficit and subsequent dysesthesias produced by these procedures would not be improved by MVD surgery.

Patients with neuralgias involving the ophthalmic division of the trigeminal nerve had better long-term results than those without V1 distribution (cure rate 84 and 72%, respectively). Although this difference is not significant ($p = 0.19$), we believe that V1 implication is a reason to prefer MVD surgery to a percutaneous destructive method that would almost inevitably produce some degree of corneal hypesthesia and risk keratitis.

The extent of the neuralgia to two divisions had no negative impact on outcome compared with the extent to one division only. When all three divisions were affected, the success rate was lower; this was significant at the 1-year follow-up ($p = 0.007$) but not at 15 years ($p = 0.11$). We assume that involvement of all three divisions could mean a severe alteration of the trigeminal nerve, perhaps an underlying neuropathy, which is hardly ever reversible.

Contrary to current belief, we found that a clinical presentation with atypical manifestations did not have a negative effect on prognosis. The percentage of long-term cures in atypical neuralgia (77.6%) was about the same as that in patients with typical neuralgia (78.9%; $p = 0.98$). In most

patients the permanent background of pain and, if present, the vasomotor manifestations disappeared together with the paroxysmal pain component. The effectiveness of MVD for atypical TN in our series could, at least partly, be explained by the fact that our criteria for defining atypical TN was very restrictive—only patients with a clear paroxysmal component and whose pain responded to anticonvulsant therapy (at least initially)³⁷ were retained. All so-called “atypical facial pain” syndromes^{8,41,42} were excluded from MVD surgery, as well as from any other type of trigeminal surgery.

The study of the outcome according to the various types of compressing vessels did not bring out any statistical difference between types ($p = 0.43$). The long-term cure rate for arterial offending vessels was in the range of 77.3 to 84.7% depending on the arteries involved. The long-term cure rate for (pure) venous compression was 66.6%; this rather good percentage of cure compared with that in literature can probably be explained by the fact that only veins that engrooved the root with a marked atrophy at the cross-compression level were retained.

The location of the NVC along the root did not affect the outcome ($p = 0.24$). The same cure rate was achieved when the location was in the middle third or laterally at the root, as in the trigeminal root entry zone. In addition, the location of the NVC around the root did not play any role either ($p = 0.15$).

The more severe the degree of compression, the better the results ($p = 0.001$). Therefore, the hypothesis might be that, when an NVC of Degree I is found, some other (adjuvant) factor must be the cause of the neuralgia (Fig. 5).

Global atrophy of the root was found in 95 patients and was severe in 48. This might testify to an underlying neuropathy. However, this finding did not correlate with a poor outcome ($p = 0.36$; Fig. 6). On the contrary, the presence of focal arachnoiditis adhesive to the root (found in 83 patients, 46 of whom had a severe form) indicates a negative long-term effect. This is shown in the Kaplan–Meier analysis prediction of a cure rate of only 58.7% after 15 years of follow-up ($p = 0.002$; Fig. 7) in such patients.

Conclusions

Consideration of the prognostic factors is interesting for practical reasons in terms of patient selection and surgical management. Older patients should not be eliminated as candidates for MVD solely on the basis of age. Neither should the presence of atypical manifestations accompanying TN exclude a patient from selection for MVD. We found that not only the spasmodic pain, but also atypical manifestations, respond well to surgery. Likewise, having had previous failed surgery for TN does not mean that patients with persistent neuralgia will not benefit from MVD, especially for treating the paroxysmal component. Patients in whom a lesioning procedure caused damage to the trigeminal nerve and caused neuropathic pain should of course not be considered for MVD, or for any other procedure destructive to the trigeminal nerve. A long duration of the neuralgia before surgery does not have negatively impact MVD effectiveness.

Concerning anatomical findings at surgery, the existence of a marked cross-compression on the root correlates with a high percentage of cure. A low degree of compression or the presence of focal arachnoiditis has a negative effect on outcome. If surgery fails in such situations, a secondary percutaneous procedure or radiosurgical treatment should be considered without delaying too long.

Consideration of these data together with the use of new magnetic resonance imaging technology that may show evidence of compressive vessels (although not yet reliably) should help achieve better patient selection for MVD, percutaneous lesioning, or radiosurgical procedures.

References

1. Apfelbaum RJ: Surgical management of disorders of the lower cranial nerves, in Schmidek HH, Sweet WH (eds): **Operative Neurosurgical Techniques. Indications, Methods, and Results, ed 2**. New York: Grune & Stratton, 1988, pp 1097–1109
2. Ashkan K, Marsh H: Microvascular decompression for trigeminal neuralgia in the elderly: a review of the safety and efficacy. **Neurosurgery** **55**:840–850, 2004
3. Barba D, Alksne JF: Success of microvascular decompression with and without prior surgical therapy for trigeminal neuralgia. **J Neurosurg** **60**:104–107, 1984
4. Barker FG, Jannetta PJ, Bissonnette DJ, Larkins MV, Jho HD: The long-term outcome of microvascular decompression for trigeminal neuralgia. **N Engl J Med** **334**:1077–1083, 1996
5. Bederson JB, Wilson CB: Evaluation of microvascular decompression and partial sensory rhizotomy in 252 cases of trigeminal neuralgia. **J Neurosurg** **71**:359–367, 1989
6. Breeze R, Ignelzi RJ: Microvascular decompression for trigeminal neuralgia. Results with special reference to the late recurrence rate. **J Neurosurg** **57**:478–490, 1982
7. Broggi G, Ferroli P, Franzini A, Servello D, Dones I: 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
8. Burchiel KJ: A new classification for facial pain. **Neurosurgery** **53**:1164–1166, 2003
9. Burchiel KJ, Clarke H, Hagland M, Loeser JD: Long-term efficacy of microvascular decompression in trigeminal neuralgia. **J Neurosurg** **69**:35–38, 1988
10. Burchiel KJ, Slavin KV: On the natural history of trigeminal neuralgia. **Neurosurgery** **46**:152–155, 2000
11. Dandy WE: Treatment of trigeminal neuralgia by the cerebellar route. **Ann Surg** **96**:787–795, 1932
12. Gardner WJ: Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. **J Neurosurg** **19**:947–958, 1962
13. Gardner WJ, Miklos MV: Response of trigeminal neuralgia to decompression of sensory root. Discussion of cause of trigeminal neuralgia. **JAMA** **170**:1773–1776, 1959
14. Hamlyn PJ, King TT: Neurovascular compression in trigeminal neuralgia: a clinical and anatomical study. **J Neurosurg** **76**:948–954, 1992
15. Jannetta PJ: Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. **J Neurosurg** **26**:159–162, 1967
16. Jannetta PJ: Microsurgical approach to the trigeminal nerve for tic douloureux, in Krayenbuhl H, Maspes PE, Sweet WH (eds): **Progress in Neurological Surgery**. Basel: Karger, 1975, pp 180–200
17. Jannetta PJ: Outcome after microvascular decompression for typical trigeminal neuralgia, hemifacial spasm, tinnitus, disabling positional vertigo, and glossopharyngeal neuralgia (honored guest lecture). **Clin Neurosurg** **44**:331–383, 1996
18. Klun B: Microvascular decompression and partial sensory rhizotomy in the treatment of trigeminal neuralgia: personal experience with 220 patients. **Neurosurgery** **30**:49–52, 1992
19. Kolluri S, Heros RC: Microvascular decompression for trigeminal neuralgia. **Surg Neurol** **22**:235–240, 1984
20. Kondo A: Follow-up results of microvascular decompression in Trigeminal Neuralgia and Hemifacial spasm. **Neurosurgery** **40**:46–52, 1997
21. Lee KH, Chang JW, Park YG, Chung SS: Microvascular decompression and percutaneous rhizotomy in trigeminal neuralgia. **Stereotact Funct Neurosurg** **68**:196–199, 1997
22. Li ST, Pan Q, Liu N, Shen F, Liu Z, Guan Y: Trigeminal Neuralgia: what are the important factors for good operative outcomes with microvascular decompression. **Surg Neurol** **62**:400–405, 2004
23. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick D: Microvascular decompression of cranial nerves lesions learned after 4400 operations. **J Neurosurg** **90**:1–8, 1999
24. Meglio M, Cioni B, Moles A, Visocchi M: Microvascular decompression versus percutaneous procedures for typical trigeminal neuralgia: personal experience. **Stereotact Funct Neurosurg** **54**:76–79, 1990
25. Mendoza N, Illingworth RD: Trigeminal neuralgia treated by microvascular decompression: a long-term follow-up study. **Br J Neurosurg** **9**:13–19, 1995
26. Piatt JH, Wilkins RH: Treatment of tic douloureux and hemifacial spasm by posterior fossa exploration: therapeutic implication of various neuromuscular relationships. **Neurosurgery** **14**:462–471, 1984
27. Pierga E: **Névràlgie Essentielle du Trijumeau par Conflit Vasculo-nerveux Opérée par Décompression Vasculaire Microchirurgicale Pure. Résultats à Long Terme et Facteurs Prognostiques sur une Série de 362 Patients. Thèse présentée à l'Université Claude Bernard, Lyon I**. Lyon: Faculté de Médecine Lyon Nord, 2001
28. Puca A, Meglio M, Cioni M, Visocchi M, Vari R: Microvascular decompression for trigeminal neuralgia: prognostic factors. **Acta Neurochir Suppl (Wien)** **58**:165–167, 1993
29. Rath SA, Klein HJ, Richter HP: Findings and long-term results of subsequent operations after failed microvascular decompression of trigeminal neuralgia. **Neurosurgery** **39**:933–940, 1996
30. Romansky K, Stoianchev E, Dinev E, Iliev I: Results of treatment of trigeminal neuralgia by microvascular decompression of the Vth nerve at its root entry zone. **Arch Physiol Biochem** **106**:392–396, 1998
31. Sindou M: Microvascular decompression for trigeminal neuralgia, in Kaye AH, Black PM (eds): **Operative Neurosurgery**. London: Churchill-Livingstone, 2000, Vol 2, pp 1595–1614
32. Sindou M, Acevedo G: Microvascular decompression of the trigeminal nerve. **Operative Tech Neurosurg** **4**:110–126, 2001
33. Sindou M, Amrani F, Mertens P: Does microsurgical vascular decompression for trigeminal neuralgia work though a neocompres-

Microvascular decompression for trigeminal neuralgia

- sive mechanism? Anatomical-surgical evidence for a decompressive effect. **Acta Neurochir Suppl (Wien)** **52**:124–127, 1991
34. Sindou M, Amrani F, Mertens P: [Microsurgical vascular decompression in trigeminal neuralgia. Comparison of 2 technical modalities and physiopathologic deductions. A study of 120 cases.] **Neurochirurgie** **36**:16–26, 1990 (Fr)
 35. Sindou M, Chiha M, Mertens P: Anatomical findings in microsurgical vascular decompression for trigeminal neuralgia. Correlations between topography of pain and site of the vascular conflict. **Acta Neurochir Suppl** **64**:125–127, 1995
 36. Sindou M, Howeidly T, Acevedo G: Anatomical observations during microvascular decompression for idiopathic trigeminal neuralgia. Prospective study in a series of 579 patients. **Acta Neurochir (Wien)** **144**:1–13, 2002
 37. Sindou M, Leston J, Decullier E, Chapuis F: Micro-vascular decompression for primary trigeminal neuralgia (typical or atypical). Long-term effectiveness on pain; prospective study with survival analysis in a consecutive series of 362 patients. **Acta Neurochir (Wien)** **148**:1235–1245, 2006
 38. Sindou M, Polo G, Fischer C, Vial C: Neurovascular conflict and hemifacial spasm. **Suppl Clin Neurophysiol** **58**:274–281, 2006
 39. Steiger HJ: Prognostic factors in the treatment of trigeminal neuralgia. Analysis of a differential therapeutic approach. **Acta Neurochir (Wien)** **113**:11–17, 1991
 40. Sun T, Saito S, Nakai O, Ando T: Long-term results of microvascular decompression for trigeminal neuralgia with reference to probability of recurrence. **Acta Neurochir (Wien)** **126**:144–148, 1994
 41. Sweet WH: Atypical facial neuralgia, in White JC, Sweet WH (eds): **Pain and the Neurosurgeon: Forty Years of Experience**. Springfield, IL: Charles C Thomas, 1969, pp 408–409
 42. Sweet WH: Atypical trigeminal neuralgia, in Gybels JM, Sweet WH (eds): **Neurosurgical Treatment of Persistent Pain: Physiological and Pathological Mechanisms of Human Pain**. Basel: Karger, 1989, pp 41–42
 43. Szapiro J Jr, Sindou M, Szapiro J: Prognostic factors in microvascular decompression of trigeminal neuralgia. **Neurosurgery** **17**:920–929, 1985
 44. Theodosopoulos PV, Marco E, Applebury C, Lamborn KR, Wilson CB: Predictive model for pain recurrence after posterior fossa surgery for trigeminal neuralgia. **Arch Neurol** **59**:1297–1302, 2002
 45. Tronnier VM, Rasch D, Hamer J, Kienle AL, Kunze S: Treatment of idiopathic trigeminal neuralgia: comparison of long-term outcome after radiofrequency rhizotomy and microvascular decompression. **Neurosurgery** **48**:1261–1268, 2001
 46. Van Loveren H, Tew JM, Keller JT, Nurre M: A 10 year experience in the treatment of trigeminal neuralgia. **J Neurosurg** **57**:757–764, 1982
 47. Walchenbach R, Voromolen JHC, Hermans J: Microvascular decompression for trigeminal neuralgia. A critical reappraisal. **Clin Neurol Neurosurg** **96**:290–295, 1994
 48. Yamaki T, Hashi K, Niwa J, Tanabe S, Nakagawa T, Nakamura T, et al: Results of reoperation for failed microvascular decompression. **Acta Neurochir (Wien)** **115**:1–7, 1992
 49. Zakrzewska JM, Lopez BC: Quality of reporting in evaluations of surgical treatment of trigeminal neuralgia: recommendations for future reports. **Neurosurgery** **53**:110–122, 2003
 50. Zakrzewska JM, Lopez BC, Kim SE, Coakham HB: Patients reports of satisfaction after microvascular decompression and partial sensory rhizotomy for trigeminal neuralgia. **Neurosurgery** **56**:1304–1312, 2005
 51. Zakrzewska JM, Lopez BC, Kim SE, Varian EA, Coakham HB: Patient satisfaction after surgery for trigeminal neuralgia—development of a questionnaire. **Acta Neurochir (Wien)** **147**:925–932, 2005
 52. Zakrzewska JM, Thomas DGT: Patient's assessment of outcome after three surgical procedures for the management of trigeminal neuralgia. **Acta Neurochir (Wien)** **122**:225–230, 1993

Manuscript submitted July 7, 2006.

Accepted May 17, 2007.

Address correspondence to: Marc Sindou, M.D., Ph.D., Department of Neurosurgery, Hôpital Neurologique "Pierre Wertheimer" 59, Bd Pinel, 69003 Lyon, France. email: marc.sindou@chu-lyon.fr.