Introduction

Trigeminal neuralgia (TN) can be managed by medical and surgical treatment. Half of the patients are cured by drugs (particularly carbamazepine) without side effects, 25% are cured but have intolerable side effects and 25% are not cured [1]. Then, half of the patients require surgical treatment. When drugs become ineffective or not tolerated, the question of which intervention (percutaneous or open) is appropriate becomes an important issue. Among modern operations, three are percutaneous (trigeminal percutaneous radiofrequency thermorhizotomy (TPRT) has an established place because of its safety in elderly patients, while microvascular decompression (MVD) has appeal in younger patients because of its non-destructive nature and because it attacks what is believed to be the primary etiology of tic douloureux. Nevertheless, MVD is a successful operation only when true neurovascular conflict (NVC) is ascertained, rather than a simple arterial loop and neurovascular contact. Probably, many immediate failures and early relapses are the consequence of the inadequate patient selection for MVD on the presumption that this operation is in any case the ideal cure. The inadequate selection can be explained by the difficult preoperative diagnosis of NVC in the past. Indeed, angiography and computed tomography showed the neurovascular contact but not the size of compression. Fortunately, today magnetic resonance imaging is a reliable instrument to ascertain NVC. So, the diatribe between the supporters of percutaneous techniques and MVD can be concluded with the following: (1) percutaneous techniques are indicated for patients without demonstrated NVC (including patients with TN in multiple sclerosis) and in those with NVC if MVD is contraindicated by ill-health or refused by the informed patient; and (2) MVD is indicated for patients with ascertained NVC who are in good health and who, informed of the surgical risk, favor this operation desiring no sensory deficit.

Key words Trigeminal neuralgia • Neurovascular conflict • Trigeminal percutaneous radiofrequency thermorhizotomy • Microvascular decompression

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Choice of open or percutaneous procedures in the surgical treatment of trigeminal neuralgia

Abstract The aim of this study was to define criteria for the selection of patients for percutaneous or open operations for the cure of drug-resistant trigeminal neuralgia (TN). Trigeminal percutaneous radiofrequency thermorhizotomy (TPRT) has an established place because of its safety in elderly patients, while microvascular decompression (MVD) has appeal in younger patients because of its non-destructive nature and because it attacks what is believed to be the primary etiology of tic douloureux. Nevertheless, MVD is a successful operation only when true neurovascular conflict (NVC) is ascertained, rather than a simple arterial loop and neurovascular contact. Probably, many immediate failures and early relapses are the consequence of the inadequate patient selection for MVD on the presumption that this operation is in any case the ideal cure. The inadequate selection can be explained by the difficult preoperative diagnosis of NVC in the past.
radiofrequency thermorhizotomy (TPRT) [2], retrogasserian glycerol injection (RGI) [3] and percutaneous compression of the trigeminal ganglion (PCTG) [4]) and two are open (partial sensory rhizotomy (PSR) [5] and microvascular decompression (MVD) [6]).

The debate between the supporters of open techniques (particularly MVD) [7–14] and of percutaneous techniques (particularly TPRT) [15–24] has lasted many years. The former sustain that MVD is the “definitive physiologic rather than symptomatic operative treatment of patients with trigeminal neuralgia” [25] and the only non-destructive surgical treatment which produces pain control without sensory impairment nor postoperative dysesthesia [26, 27]. The latter point out that TPRT avoids the risks of craniectomy, it is repeated easily if tic pain recurs, morbidity is minimal and there is essentially no risk of mortality. Burchiel et al. [28] sustained that MVD should be considered because it attacks what is believed to be the primary etiology of tic douloureux, the trigeminal nerve is preserved, postoperative pain relief does not depend upon the production of sensory deficit and it may have a greater potential to produce long-lasting pain relief. Möbius et al. [29] affirmed that MVD should especially be recommended for patients in whom all other forms of therapy including TPRT have failed. Walchenbach et al. [30] stated that as neither TPRT nor MVD is an unequivocally more effective treatment, the less invasive procedure should be preferred. Morley [31] put the question if “it is justified to place the patient at a 1% risk of death and a 10% risk of significant, sometimes grave morbidity for the treatment of a condition that is never fatal when other procedures are available that are effective and carry virtually no risk of death or neurologic morbidity other than planned sensory impairment”. Soyka [32] affirmed that the principle of MVD is either an interruption of the pain-conducting fibers or a non-specific manipulation at the gasserian ganglion or the sensory root with the result of an interruption of abnormal ephapses and short-circuits and that the operation should not be considered to be a specific and causal therapeutic approach as well as the therapy of first choice for all cases. Taha and Tew [33, 34] affirmed that TPRT is the procedure of choice for most patients undergoing first surgical treatment and that MVD is recommended for healthy patients who desire no sensory deficit. On the contrary, Kunze and Steiner [9] affirmed that in every case of typical TN there is an indication for MVD, provided that pharmacotherapy has proved ineffective and anesthesia carries no increased risk due to old age or ill-health. For patients under 65 years, Broggi et al. [35] proposed PCTG or, if neuroradiological evidence of neurovascular conflict (NVC) is given, MVD; for patients older than 65 years, these authors proposed TPRT.

Considering these different opinions and the lack of firm evidence for choosing the surgical treatment, the aim of this study was to define criteria for the selection of patients undergoing percutaneous or open operations through an up to date review of the literature on the outcome of TPRT (the more classic percutaneous surgical treatment of TN) and MVD.

### Materials and methods

Out of all published reports of patients treated by TPRT or MVD, papers with an average follow-up of 3 years or longer were selected. According to this criterium, 18 studies including 7473 TN patients treated by TPRT (Table 1) with a follow-up from 3 to 9.3 years (average 5.8 years) and 17 studies including 3611 patients treated by MVD (Table 2) with a follow-up from 3 to 8.5 years (average 5 years) were considered. The results are indicated as the average of the values of the different papers.

### Results

Immediate pain relief was achieved in 92.5% of patients treated with TPRT and 90.4% of patients undergoing MVD. Relapses of pain were observed in 24.4% of TPRT cases and in 21% of MVD cases. The predictable postoperative pain relief examined with the Kaplan-Meier plot [36] showed a half-life of 3 years [37], 5 years [38] or more than 5 years [39] for TPRT and of 10 years [40] for MVD. Facial sensory deficit was a common and wanted finding in TPRT cases and an unwanted remark in 8.9% of MVD cases.

Dysesthesia was reported by 15.6% of patients treated by TPRT: it was mild (non-disturbing and non-requiring treatment) in 7.9% of cases, moderate (disturbing but controlled by drugs) in 5.9% and severe (disturbing and not controlled by drugs) or anesthesia dolorosa in 1.8%. In MVD-treated patients, dysesthesia was not quoted among the complications.

Motor deficits figured in 7.3% of patients treated with TPRT. Damage to cranial nerves other than V figured in 0.4% of TPRT cases (particularly, IV and VI with transient diplopia) and in 7.4% of MVD cases (particularly, VII and VIII with bradycusia or deafness).

Fritz et al. [41] observed that out of 21 patients examined audiometrically before and after MVD, five (23.8%) had postoperative hearing impairment. Fuse and Miller [42] observed a delayed and progressive hearing loss after MVD, which they interpreted to be the result of reactive scar tissue and atrophy of the auditory nerve. Vestibular nerve injuries were also described [43] and, moreover, penduncular hallucinosis [44, 45].

Other possible major neurological complications after MVD are: cerebellar hemorrhage, edema or infarction, arterial air embolism, acute mental status change, status epilepticus, acute epidural hematoma, chronic subdural hematoma,
Table 1 Outcome of trigeminal percutaneous radiofrequency thermorhizotomy (TPRT) for 7473 patients in 18 studies with a follow-up of 3 years or longer, presented in chronological order from 1974 to 1995. Values are percentages of patients unless otherwise indicated.

| Patients, n | Average follow-up years | Pain relief | Complications |
|-------------|-------------------------|-------------|---------------|
|             |                         | Immediate   | Relapses      | Corneal anesthesia | Keratitis | Dysesthesia | Motility disturbance | Diplopia | Death |
|             |                         |             |               | L | M | S |               |             |       |       |
| 214         | 4.2                     | 91          | 22            | 10 | 0.5 | 3<sup>a</sup> | – | 1 | NG | NG | 0 |
| 50          | 3.0                     | 80          | 40            | NG | 0 | 12<sup>a</sup> | – | 0 | 20 | NG | 0 |
| 1000        | 6.7                     | 98          | 21            | 21 | 6 | 17 | 13 | 3 | 1 | NG | 0 |
| 400         | 3.5                     | 93          | 6.7           | 32 | 0 | NG | NG | 0.2 | NG | NG | 0 |
| 700         | 6.0                     | 99          | 20            | NG | 4 | 22 | 5 | NG | NG | NG | 0 |
| 96          | 5.0                     | 90          | 50            | 41 | 2 | 13.5 | 12.5 | 1 | 5.2 | 5 | 0 | 0 |
| 229         | 3.8                     | 94          | 21            | NG | 4 | 6<sup>a</sup> | – | 9 | 1 | 1.5 | 0.3 |
| 98          | 4.5                     | 69          | 44            | 26 | 4.4 | NG | NG | 2.9 | 0 | 0 | 0 |
| 122         | 7.0                     | 92          | 27            | NG | NG | NG | NG | NG | NG | NG |
| 533         | 6.5                     | 97          | 10            | 3 | 1.9 | 15 | NG | 1.5 | 3 | 0.2 | 0 |
| 700         | 3.0                     | NG          | 25            | 1 | NG | NG | NG | 0.6 | 8 | 0.1 | NG |
| 1000        | 9.3                     | 95          | 18            | 20 | 0.5 | NG | 5.2 | 1.5 | 10.5 | 0.5 | 0 |
| 124         | 3.7                     | 95          | 28            | 5 | 2 | 17 | 3 | 3 | 3 | 1 | 0 |
| 1070        | 9.2                     | 96          | 22            | 3.5 | 0.4 | 6<sup>a</sup> | – | 0.5 | 26 | 0.2 | 0.2 |
| 568         | 6.0                     | 97          | 16            | NG | NG | NG | NG | NG | NG | NG |
| 240         | 4.11                    | 96          | 28            | 3.7 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 175         | 5.0                     | NG          | 20            | 17 | 2 | NG | NG | 1.8 | 3 | NG | 0 |
| 154         | 15.0                    | 99          | 21            | 18.8 | 1.9 | 23<sup>a</sup> | – | NG | 14.2 | NG | 0 |
| Average result |                        | 5.8         | 92.5          | 24.4 | 13.3 | 1.9 | 7.9 | 5.9 | 1.8 | 7.3 | 0.4 | 0.03 |

NG, data not given; L, mild dysesthesia (not requiring treatment; M, moderate dysesthesia (controlled by drugs); S, severe dysesthesia or anesthesia dolorosa (not controlled by drugs)

<sup>a</sup> Data refer to mild (L) and moderate dysesthesia
Table 2: Outcome of microvascular decompression (MVD) for 3611 patients in 17 studies with a follow-up of 3 years or longer, presented in chronological order from 1982 to 1986. Values are percentages of patients unless otherwise indicated.

| Patients, n | Follow-up, years | Pain relief | Complications |
|-------------|------------------|-------------|---------------|
|             | Range | Mean | Immediate | Relapses | Cranial nerve damage | General complications<sup>a</sup> | Death |
|             |       |      |           |          | V | IV, VI, VIII |                      |      |
| Van Loveren et al. [24] | 23    | –    | 3         | 96       | 13 | 13 | 9 | NG | 0 |
| Apfelbaum [104] | 289   | –    | 4.6       | 94       | 28 | 0  | 3 | NG | 1 |
| Barba, Alksne [106] | 37    | –    | 3.6       | NG       | 27 | NG | NG | NG | NG |
| Kolluri, Heros [107] | 72    | –    | 4.9       | 96       | 18 | 12 | 25 | NG | 0 |
| Piatt, Wilkins [62, 63] | 103   | –    | 4         | 77       | 23 | 10.6 | 16.5 | 8.7 | 0.9 |
| Burchiel et al. [40] | 36    | 7.5–11.5 | 8.5      | 90       | 47 | 20 | NG | NG | NG |
| Bederson, Wilson [57] | 252   | –    | 5.1       | 83       | 12 | 3  | 4.8 | 7.9 | 0 |
| Dahle et al. [108] | 57    | 3–7  | 3.1       | NG       | 20 | 12 | 3.6 | 8.7 | 1.7 |
| Jannetta [25] | 703   | 1–11.8 | 6.5      | 79       | 27 | 0  | 1.3 | 7   | 0.6 |
| Klun [52] | 178   | 0.5–12 | 5.2       | 96       | 6  | 1.3 | 7.2 | 1.3 | 1.3 |
| Sindou, Mertens [53] | 420   | NG   | <30 (NG) | 91       | 6  | NG | 2.3 | 0.4 | 0.4 |
| Suwa et al. [66] | 29    | 4.2  |           | 96       | 7  | NG | NG | NG | NG |
| Sun et al. [67] | 61    | 1–12 | 6.6       | 100      | 18 | NG | NG | NG | NG |
| Walchenbach et al. [30] | 58   | 1–12 | 6.4       | 80       | 29 | NG | NG | NG | 0 |
| Meneses et al. [11] | 48    | 1–8  | 4.5       | 98       | 16 | NG | NG | NG | 0 |
| Mendoza, Illingworth [109] | 60   | 0.5–15 | 5        | NG       | 29 | NG | NG | NG | NG |
| Barker et al. [7] | 1185  | <20  | 6.2       | NG       | 30 | 17 | 1  | NG | 0.2 |

Average result  5.0  90.4  21  8.9  7.4  5.6  0.6

<sup>a</sup> General complications include cerebellar hemorrhage, edema or infarction; arterial air embolism; acute mental status change; status epilepticus; acute epidural or chronic subdural hematoma; subarachnoid hemorrhage; aseptic or bacterial meningitis; CSF leak; hydrocephalus; infarction of the brain stem; NG, data not given
subarachnoid hemorrhage, aseptic or bacterial meningitis, cerebrospinal fluid leak (CSF) leak, hydrocephalus and infarction of the brain stem. Finally, operative death occurred in 0.03% and 0.6% of patients undergoing TPRT and MVD, respectively.

Considering the outcome of TPRT and MVD in multiple sclerosis-related TN (MS-TN), Siegfried [46] affirmed that pain can be successfully eliminated by operation. Brisman [47] found that the probability of recurrence showed no significant differences in 16 patients with MS-TN compared with 219 patients without MS. Only Broggi and Franzini [48] observed a high recurrence rate in the MS-TN group (40% at 1–4 years of follow-up). On the other hand, Resnick et al. [49] affirmed that MVD fails to provide adequate or reliable pain relief in MS-TN.

Discussion

TPRT, the selective thermoablation of the trigeminal ganglion and root, has the faculty to control preoperatively by electrostimulation the trigeminal division(s) involved and, to some extent, to modulate the lesion itself in order to have hypalgesia or analgesia without anesthesia. On the other hand, MVD, which is not a neurolesive operation, consists of the separation of the retrogasserian root away from the impinging vessel using an interposed foreign body or by the transposition of vessel without interposition of synthetic material [50]. The operation is based on the assumption that TN is caused by NVC.

The average immediate postoperative pain relief and long-term recurrence are the same for TPRT and MVD. For both operations, the percentage of recurrence is different in the various series. The different percentages of recurrence after TPRT (from 6.7% in the paper of Schvarcz [43] to 50% in the paper of Latchaw et al. [38]) depend on:

1. The different pre-determined end-points of operation: analgesia or hypalgesia. In the paper by Latchaw et al. [38], at 5 years relapses occurred in 26% of the patients in which the operation produced analgesia or anesthesia, in 54% of patients in which it produced hypalgesia, and in 100% of patients in which no sensory deficits were caused.

2. The technical differences. Recurrence (particularly early relapses) can be due to an approximate technique. Performed by a skilled operator, TPRT allows the precise location of the cannula within the retrogasserian division and the lesion can be accurately confined. TPRT is relatively time consuming: it takes up to 2 hours and must be performed according to the following operative steps [51]:

   (a) Identification by fluoroscopy in oblique projection of the sector of the oval foramen (the medial third for trigeminal division I-II and the middle third for III): this step takes only a few minutes;

   (b) Localization of the trigeminal cistern (this structure can be more or less deeply located depending on the length of the mandibular branch);

   (c) Electrophysiological identification of the target (this step is time consuming because at any attempt the operator must wait for the patient to awake prior to stimulation and for subsequent sedation prior to moving the electrode);

   (d) Progressive thermoablation and verification of the obtained pin prick hypalgesia-analgesia.

The different percentages of recurrence after MVD (from 6% in the study of Klun [52] and of Sindou and Mertens [53] to 47% in the study of Burchiel et al. [40]), apart from the operator’s different technical experience, can reflect the different criteria in the selection of the patients or, merely, the casual inclusion of patients with or without true NVC.

If one considers the different surgical risks (operative death of MVD is 20-times greater than that of TPRT), 21% of recurrence after MVD can be regarded as a disappointing outcome in comparison with 24.4% after TPRT. The recurrence rate after MVD is so high that terms such as “failed microvascular decompression” [54–56] and “failed posterior fossa exploration” [57] were coined. In order to define indications for MVD, we must explain if MVD is the etiologic and definitive treatment for TN and if it really does not provoke facial sensory impairments nor postoperative dysesthesia.

Is MVD the etiologic treatment for TN?

MVD is the etiologic treatment when NVC occurs. Nevertheless, whereas most TN patients have NVC, some do not [24, 58]. Moreover, NVC is a common finding in asymptomatic patients [59], in some TN patients it is contralateral to the pain [60] and, finally, many nerve roots are physiologically in contact with arteries and veins in the skull [61]. In order to make clear the concept of NVC and to study its pathogenetic role, the papers of Hardy and Rhoton [59] and Haines et al. [58] are quoted. Hardy and Rhoton bilaterally examined the trigeminal roots in 25 corpses of subjects who never had TN. They found that in 30 of 50 nerves, there was contact between an artery and the trigeminal root: 26 of them were with the superior cerebellar artery (SCA) and 4 were with the antero-inferior cerebellar artery (AICA). Quoting this study, Adams and Chir [61] observed that arterial loop and neurovascular contact were present at least on one side in every examined subject and that as “each patient had a vessel in contact with one or other trigeminal nerve … one can conclude that it is unusual not to find some
vascular contact in asymptomatic patient at or near the root entry zone". In other words, arterial loop and neurovascular contact seem to be physiological. Haines et al. [58] examined 80 corpses: 40 without and 40 with a history of TN. These authors found, respectively, neurovascular contact and compression in 48% and 10% of the subjects without TN and in 28% and 80% of the subjects with TN. They concluded that arterial loop and neurovascular contact are common but cause TN only if the vessel exerts a “significant compression” on the root. At surgical exploration the root is “distorted” and shows a groove where the artery pulses. Piatt and Wilkins [62, 63] verified clinically these anatomical findings and correlated the surgical outcome with the nerve-vascular relation type. They found that MVD cured TN in 83%, 62% and 42% of the cases if, respectively, distortion of the root by an artery, contact of the root with an artery without distortion or contact-distortion of the root with a vein were present. Hence, the type of neurovascular relationship is crucial and arterial loop and neurovascular contact are to be distinguished by the compression and distortion of the root (or NVC). The latter has pathogenetic relevance whereas the simple arterial loop and neurovascular contact are unimportant anatomical abnormalities. Of the same opinion were Niwa et al. [64] who pointed out the difference between contact which is asymptomatic and compression which is symptomatic. Moreover, as NVC is not present in every TN patient, it is not the only cause of “essential” TN but one of the possible causes [65]. As demyelination is constantly present in every patient, natural aging can figure among the other causes of neuralgia [66].

Is MVD the definitive treatment for TN?

MVD is the definitive treatment for TN if there is NVC and there is not a severe neuropathic lesion of trigeminal root. Recurrence can be ascribed to the postoperative fibrotic adhesions formed around the nerve [56], to the intrinsic lesion [67] and especially to the absence of NVC.

Does MVD not provoke facial sensory impairments?

Facial sensory impairment occurred in an average of 8.9% of all patients treated with MVD and in 20% in the series of Burchiel et al. [40]. Searching for corneal sensitivity after neurosurgical interventions on the trigeminal nerve, Ackermann-Kaorner and Draeger [68] found that MVD may lead to a severe decrease of corneal sensitivity. In terms of complications concerning sensory loss of cornea, TPRT was the less risky treatment, followed by RGI, MVD and retro-gasserian rhizotomy according to Frazier [69]. Hence, it seems a hazard to guarantee patients that MVD does not cause sensory deficits: nevertheless, severe dysesthesia is an unusual complication.

Selection of patients

It is current opinion that TPRT has an established place because of its safety, particularly in elderly patients, and that MVD has an appeal in younger patients [70–76] because of its non-destructive nature. However, there are presently no clear guidelines for the choice of percutaneous or open operation. In order to establish such guidelines, it is crucial to preoperatively distinguish NVC from the innocent arterial loop and neurovascular contact and to recognize the type (reversible or irreversible) of intrinsic nerve lesion. The preoperative distinction between NVC and arterial loop was difficult in the past because angiography and computed tomography (CT) showed the arterial loop and neurovascular contact but not the size of compression. At operation, Tew et al. [60] found a “significant” impingement in less than half of 50 patients in whom preoperative study showed arterial loop and neurovascular contact. Fortunately, today magnetic resonance imaging (MRI) is a reliable technique to study neurovascular interaction at the trigeminal root entry zone [64, 77–93] and it must be performed in every TN patient candidate to MVD. Meaney et al. [86], sustaining that MRI is “an extremely sensitive and specific method for demonstrating vascular compression in TN”, concluded that “open surgical procedures can be recommended with confidence”.

Allowing the choice of MVD only at the presence of a demonstrated nerve compression, we can expect that the accuracy in the identification of NVC with MRI will reduce the percentage of postoperative failures and recurrence. Today, it is possible to demonstrate NVC but unfortunately there is no available method to preoperatively recognize the reversibility or irreversibility of the intrinsic nerve lesion in TN and consequently to identify the patients who surely will improve after MVD. This uncertain diagnosis remains the most frequent cause of failure, because if there is irreversible nerve damage, decompression does not ameliorate the symptoms and neurolesion is the only available treatment.

MVD is a successful operation when the indication is correct. Probably, many failures and relapses are the consequence of an inadequate selection of patients, after operations performed on the presumption that MVD is in any case the ideal cure for TN. With the availability of MRI, it can be stated that MVD is not an alternative of percutaneous techniques, favorable if the patients can tolerate a major opera-
tion: it has firm indications (Fig. 1) and the diatribe between the supporters of percutaneous techniques and MVD can be concluded with the following statements: (1) percutaneous techniques are indicated for patients without demonstrated NVC (including patients with TN-MS) and in those with NVC if MVD is contraindicated by a concomitant illness or is refused by the informed patient, and (2) MVD is indicated in patients with ascertained NVC, without severe trigeminal neuropathy and in good health who, informed of the surgical risk, favor this operation desiring no sensory deficit and hoping to be among the more than 80% subjects who have no postoperative sensory deficits.

Fig. 1 Choice of open or percutaneous surgical procedures for trigeminal neuralgia. NVC, neurovascular conflict; MVD, microvascular decompression

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