TRIGEMINAL NEURALGIA

TRIGEMINAL NEURALGIA consists of extremely severe, lightning-like, electric, stabbing (“lancinating”) pain in the distribution of one or more divisions of the trigeminal nerve (1). The attacks, which typically last a few seconds each, arise either spontaneously or in response to a triggering stimulus, such as light touch in the cutaneous distribution of the trigeminal nerve, chewing, speaking, swallowing, or tooth-brushing. The pain is disabling and causes marked suffering; often, the sufferer is no longer able to eat. Thus, all patients with trigeminal neuralgia should be started on pharmacological treatment as soon as the condition is diagnosed. Between attacks, the patients are asymptomatic. Multiple attacks can occur daily for periods of weeks or months; in the early stage of the condition, they can also remit spontaneously for periods of weeks or months. The condition usually becomes increasingly severe over time. 29% of patients have only one episode in their lifetime, while 28% have three or more. 21% of patients have attacks every year in the first 5 years after the onset of the condition (2). There are no known factors enabling the physician to predict the long-term prognosis at the onset of the disease.

So-called idiopathic or classic trigeminal neuralgia is caused by vascular compression of the trigeminal nerve at its origin from the pons. Pain control can often be achieved by antiepileptic medication. Interventional procedures may be considered where symptom control is inadequate or side effects unacceptable. Methods: Selective literature review taking into account relevant guidelines. Results: Positive long term results following vascular decompression lend support to the rationale that vascular compression is etiological. Hence idiopathic trigeminal neuralgia has, in principle, become a curable condition. Patients unfit for surgery due to their age or medical condition may benefit from radiosurgery using the gamma knife. Percutaneous thermocoagulation and glycerol infiltration also have a place in these patients. Radiosurgery and percutaneous procedures share the disadvantage of sensory loss and a 50% recurrence rate within 5 years. Discussion: Greater tailoring of the intervention to the individual patient is now possible, but minimally invasive procedures are associated with high rates of sensory deficit and recurrence.

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Key words: trigeminal neuralgia, microvascular decompression, stereotactic radiosurgery, thermocoagulation, glycerol infiltration

Summary

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Current Treatments for Trigeminal Neuralgia – a Surgical Approach

Hans-Jakob Steiger, Gerhard Horstmann, Rainer Freynhagen

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The treatment of this condition has developed rapidly in the last few years. The positive long-term results of microvascular decompression of the trigeminal nerve have provided persuasive evidence that classic trigeminal neuralgia is caused by vascular compression; trigeminal neuralgia has thus become a curable disease (4). On the other hand, Gamma Knife radiosurgery has taken on an important role in the treatment of patients who are unfit for surgery because of their poor general condition or advanced age. In this article, we review the current results of the available therapeutic procedures and discuss their indications. To ascertain the long-term results of the various surgical and radiosurgical techniques, we performed a Medline search for journal article titles containing the term "trigeminal neuralgia." We included only controlled studies with multiple years of follow-up in our analysis. We considered the data on any particular treatment method to be reliable if the results of two or more studies were in close agreement. For recommendations regarding pharmacotherapy, we have made use of the meta-analyses of the Cochrane Data Bank and the guidelines of the German Neurological Society (Deutsche Neurologische Gesellschaft, DNG [8]).

Pharmacotherapy
Even in the year 2007, the primary management of trigeminal neuralgia remains conservative. In principle, a surgical intervention should be considered when maintenance therapy with medications causes major side effects or fails to relieve the pain adequately. The side effects of all medications that are effective against trigeminal neuralgia include dizziness, sleepiness, and cognitive impairment, ranging all the way to cerebral and cerebellar atrophy that are visible by MRI (9). The authors recommend surgery when the patient's quality of life is markedly impaired by inadequately controlled pain or by the side effects of medication.

Because each attack is very brief, analgesic treatment for the attacks as they occur is out of the question. The goal of pharmacotherapy is thus to prevent attacks (prophylaxis). Non-pharmacological treatments such as psychotherapy, acupuncture, and dental correction procedures have not been convincingly shown to have any effect (8, 10).

Medications should be dosed individually, titrating to their therapeutic effect and side effects (8). The dose should be raised slowly till the patient either is free of pain or else suffers intolerable side effects. If the effect of the medication diminishes over time, its dose will have to be increased accordingly. Conversely, if the patient has been free of pain for 4 to 6 weeks on a given dose, the dose should be reduced in stepwise fashion so that any possible remissions can be recognized early.

Classic trigeminal neuralgia usually responds, at least initially, to treatment with antiepileptic drugs (11). As the disease progresses, this treatment effect tends to wear off. In view of the small number of randomized, controlled trials, the choice of pharmacotherapy is still largely empirical rather than evidence-based (12, 13). Treatment with carbamazepine, preferably in sustained-release form, is still considered the gold standard worldwide, even though many antiepileptic drugs are available with a much better side effect profile. 90% of patients initially respond well to treatment with carbamazepine, and 50% continue to respond well for several years. The initial daily dose for trigeminal neuralgia is generally in the range of 200 to 400 mg; the prescribing physician can raise the dose daily by 50 mg to avoid producing fatigue, ataxia, and dizziness by raising the dose too quickly. Because enzyme autoinduction may occur, with consequent loss of drug effect, the carbamazepine dose may need to be raised still further during the first few weeks of treatment to provide an adequate beneficial effect. The dose required to control pain in these mostly elderly patients is usually between 600 and 1200 mg/day. The side effects include cognitive impairment, rare skin exanthems, thrombo- and leukocytopenia, hyponatremia, hepatic dysfunction, and cardiac arrhythmias.

Oxcarbazepine, a prodrug of carbamazepine, is similarly effective against trigeminal neuralgia (14, 15). It reaches its maximum serum concentration one hour after oral ingestion. The necessary daily dose ranges from 900 to 1800 mg. Its advantages over carbamazepine are a better cognitive side effect profile and the lack of autoinduction of drug metabolism. It has the disadvantage, however, that it causes hyponatremia somewhat more often (frequency ca. 23%) (16). The serum sodium concentration should be re-checked regularly, particularly in patients with clinical side effects such as confusion, headache, fatigue, or nausea. Oxcarbazepine can be switched to carbamazepine in...
a single day if necessary, or vice versa: the carbamazepine dose multiplied by 1.5 is the oxcarbazepine dose.

A number of other substances have been promoted for use against trigeminal neuralgia, including baclofen, lamotrigine, gabapentin, pregabalin, valproic acid, and tricyclic antidepressants. At the moment, however, only carbamazepine, gabapentin, and pregabalin have been approved for the treatment of trigeminal neuralgia and other types of neuropathic pain.

In the acute situation when rapid intervention is indicated, e.g., during an exacerbated attack, freedom from pain can often be attained in a short time with a slow intravenous infusion of 250 mg of phenytoin (1). The infusion rate should not exceed 1 mg per kg body weight per minute because of the cardiodepressive effect of the drug. The remainder of the loading dose can be given, if necessary, either intravenously or by the oral route (3 mg per kg body weight in 3 divided doses). There is convincing scientific evidence that antiepileptic drugs are effective against trigeminal neuralgia (11), but the evidence for other classes of medication is derived only from small-scale, non-randomized studies (8).

Microvascular decompression

According to the guidelines of the German Neurological Society, surgical treatment of classic trigeminal neuralgia is indicated when pharmacotherapy is ineffective or when its side effects markedly impair the patient’s quality of life (8). Because surgery is only held to be indicated once all medical treatment options have been exhausted, there are no studies comparing the effectiveness of medical and surgical treatment.

Microvascular decompression (the “Jannetta operation”) is performed under general anesthesia through an opening in the posterior cranial fossa via suboccipital craniotomy and is widely accepted as a curative treatment method (4). A contact between the trigeminal nerve and a blood vessel, usually the superior cerebellar artery, is found at the site where the nerve enters the brainstem (figures 1 and 2). The two contacting structures are separated by the insertion of a piece of synthetic material, e.g., a small Teflon sponge (a so-called “pledget”). After surgery, the success rate is high: 80% of patients are free of pain, and 15% are relieved of pain to some extent (4, 18). There are occasional early recurrences; thus, the definitive cure rate is approximately 70%. Early recurrences may occur because, in some patients, the neurovascular contact was not, in fact, the cause of the pain, and it was only the operative manipulation of the trigeminal nerve that led to transient relief. On the other hand, they may also occur if the interposed piece of material slips out of position postoperatively and the original neurovascular contact is restored. If pain recurs after microvascular decompression, the authors recommend another attempt to treat with medications as the initial management. If pain relief continues to be insufficient, or if the side effects are intolerable, then we recommend reoperation or recourse to one of the other interventional procedures described below.

Figure 1: Right-sided operative approach and view of the operative field in microvascular decompression (the “Jannetta operation”). Inspection of the origin of the trigeminal nerve (the fifth cranial nerve, marked “V”) from the pons reveals the typical finding of a loop of superior cerebellar artery (arrows) pressing on the nerve in the acute angle that it makes with the brainstem (the so-called “axilla” of the nerve).
Table 1 contains a summary of the results of the major publications on this subject. The follow-up interval varied from one study to another, but the larger studies relatively consistently found long-term cure rates from 70% to 80%. Studies with more than 5 years of follow-up found recurrence rates ranging from 10% to 30%. The rate of success after reoperation is less than that after initial operation; about 50% of patients are pain-free 5 years after repeated surgery.

Although microvascular decompression has been shown to yield excellent results in the hands of many different neurosurgeons, it does have occasional complications. The main danger is of ipsilateral deafness, which occurred in 1% of the patients of the team led by Jannetta (4). The operative mortality in the same series was 0.2%. Microvascular decompression can lead to a sensory deficit, though less commonly than the destructive methods (19). Dysfunction of other cranial nerves and cerebrospinal fluid fistulae occur only rarely.

Radiosurgical treatment
In the last few years, Gamma Knife treatment, a much less invasive technique, has acquired a role in the treatment of trigeminal neuralgia (20–22). The trigeminal nerve is radiated at
its root entry zone in a single, high dose of 80 to 90 Gy (figure 3). This is an ablative method that causes partial damage of the nerve; thus, 10% to 30% of the treated patients have a sensory deficit. The frequency of a disturbing sensory deficit ranges from 5% to 20%. The major published series are summarized in table 2. The pain improves initially in 70% to 90% of patients, though only after a latency of several days to weeks. Early recurrences are common, too, so that only about half of all patients are still pain-free at 5 years. The results vary markedly from series to series with respect to pain relief and sensory loss; therefore, it must be assumed that there are major technical differences in the way different centers perform this treatment. Factors such as correction for distortion in the planning MRI may play a large role, because the target volume is so small that the required precision is at the limit of what can be achieved with the radiosurgical apparatus.

Only a few reports have been published concerning repeated Gamma Knife treatment after pain recurrence. It seems that irradiation with an additional ca. 20 Gy leads to remission in about 50% of patients and partial improvement in 30%, although additional sensory deficits can be expected (23–25). In this respect, and also with regard to its high recurrence rate,

<table>
<thead>
<tr>
<th>Authors</th>
<th>Number of patients followed</th>
<th>Free of pain (%)</th>
<th>Partial improvement (%)</th>
<th>Recurrences (%)</th>
<th>Average duration of follow-up (years)</th>
<th>Maximal duration of follow-up (years)</th>
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<tr>
<td>Breeze, Igelnzi 1982 (e11)</td>
<td>51</td>
<td>85%</td>
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<td>15%</td>
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<td>van Loveren, Tew et al. 1982 (e12)</td>
<td>23</td>
<td>83%</td>
<td>-</td>
<td>17%</td>
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<td>-</td>
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<td>Barba, Alsone 1984 (e13)</td>
<td>23</td>
<td>91%</td>
<td>-</td>
<td>9%</td>
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<td>Kolluri, Heros 1984 (e14)</td>
<td>65</td>
<td>75.4%</td>
<td>-</td>
<td>24.6%</td>
<td>4.9</td>
<td>-</td>
</tr>
<tr>
<td>Piatt, Wilkins 1984 (e15)</td>
<td>81</td>
<td>74%</td>
<td>-</td>
<td>26%</td>
<td>4</td>
<td>-</td>
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<td>Apfelbaum 1988 (e16)</td>
<td>466</td>
<td>67%</td>
<td>14%</td>
<td>18%</td>
<td>6.4</td>
<td>20</td>
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<td>Burchiel, Clare et al. 1988 (e17)</td>
<td>36</td>
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<td>-</td>
<td>16.7%</td>
<td>2.1</td>
<td>-</td>
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<td>Steiger 1991 (19)</td>
<td>22</td>
<td>68%</td>
<td>-</td>
<td>32%</td>
<td>5</td>
<td>8</td>
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<td>Klun 1992 (e18)</td>
<td>167</td>
<td>93.4%</td>
<td>3.6%</td>
<td>3%</td>
<td>5.2</td>
<td>-</td>
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<tr>
<td>Yamaki, Hashi et al. 1992 (e19)</td>
<td>60</td>
<td>63.3%</td>
<td>21.7%</td>
<td>15%</td>
<td>3</td>
<td>5.5</td>
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<tr>
<td>Zakrzewska, Thomas 1993 (e20)</td>
<td>60</td>
<td>78%</td>
<td>12%</td>
<td>10%</td>
<td>5</td>
<td>9</td>
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<tr>
<td>Sun, Salto et al. 1994 (e21)</td>
<td>59</td>
<td>78%</td>
<td>-</td>
<td>22%</td>
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<td>Walchenbach, Voormolen et al. 1994 (e22)</td>
<td>58</td>
<td>64%</td>
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<td>29%</td>
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<td>Mendoza, Illingworth 1995 (e23)</td>
<td>124</td>
<td>68.6%</td>
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<td>5.3</td>
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<td>Barker, Janetta et al. 1996 (4)</td>
<td>1155</td>
<td>69.6%</td>
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<td>25%</td>
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<td>Rath, Klein et al. (e24)</td>
<td>135</td>
<td>73.1%</td>
<td>-</td>
<td>26.9%</td>
<td>2.5</td>
<td>5</td>
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<td>Kondo 1997 (e25)</td>
<td>226</td>
<td>86.1%</td>
<td>5.2%</td>
<td>8.7%</td>
<td>9.2</td>
<td>20</td>
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<td>Lee, Chang et al. 1997 (e26)</td>
<td>146</td>
<td>89%</td>
<td>-</td>
<td>11%</td>
<td>1</td>
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<td>Romansky, Stoianchev et al. 1998 (e27)</td>
<td>85</td>
<td>90.2%</td>
<td>3.7%</td>
<td>6.1%</td>
<td>1</td>
<td>-</td>
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<tr>
<td>Tronnier, Rasche et al. 2001 (e28)</td>
<td>225</td>
<td>63%</td>
<td>-</td>
<td>37%</td>
<td>10.9</td>
<td>20</td>
</tr>
<tr>
<td>Theodosopoulos, Marco et al. 2002 (e29)</td>
<td>420</td>
<td>72%</td>
<td>21%</td>
<td>7%</td>
<td>4.6</td>
<td>8</td>
</tr>
<tr>
<td>Li, Pan et al. 2004 (e30)</td>
<td>62</td>
<td>79%</td>
<td>-</td>
<td>21%</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Zakrzewska, Lopez et al. 2005 (e31.e32)</td>
<td>220</td>
<td>84%</td>
<td>-</td>
<td>16%</td>
<td>5</td>
<td>10</td>
</tr>
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<td>Sindou, Leston et al. 2006 (18)</td>
<td>362</td>
<td>80%</td>
<td>4.9%</td>
<td>15.1%</td>
<td>8</td>
<td>18</td>
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</tbody>
</table>

* The individual studies vary considerably with respect to methods of data assessment and comprehensiveness of follow-up.
radiosurgery is similar to the percutaneous techniques. The Gamma Knife can also be used to treat patients whose pain recurs after microvascular decompression (e1), but no precise figures are available concerning the success rate, which may or may not be comparable to that of primary radiosurgical treatment.

**Percutaneous techniques**

The percutaneous techniques, like radiosurgery, are destructive, but unlike it they produce a lesion in the Gasserian ganglion rather than in the prepontine root entry zone. The major long-term studies of treatment with percutaneous methods are summarized in table 3. A lesion is made in the trigeminal ganglion either with heat (thermocoagulation) or through a chemical effect (glycerol rhizolysis) (e2, e3). Local anesthesia or transient intravenous sedation is given, and a special type of needle is introduced freehand, under fluoroscopic guidance, at a cutaneous puncture site 2 to 3 cm from the corner of the mouth, through the cheek, and into the foramen ovale. The trigeminal ganglion is heated to 60° or 70°C for 60 to 70 seconds with a radiofrequency probe, producing a partial lesion, or else 0.3 to 0.4 mL of pure anhydrous glycerol is injected into Meckel’s cave through the needle. Balloon compression of the trigeminal ganglion is a third percutaneous technique that once enjoyed a certain degree of popularity: in this method, a 4 French Fogarty catheter is inserted through the foramen ovale and inflated for a few minutes with 0.75 to 1 mL of contrast medium (producing an intraluminal pressure of ca. 1500 mm Hg) (e4). Hardly any long-term follow-up studies of this technique are available.

Thermocoagulation and glycerol infiltration have proven their worth over the past 20 years. Either procedure can achieve total pain relief, at least initially, in 80% to 90% of patients. About half of all patients have a sensory deficit, which is disturbing in 10% to 15%. 50% to 60% of all patients are still pain-free at 5 years. The results of different clinical series are comparable to one another with respect to both initial and long-term results. Anesthesia dolorosa is about as common after either procedure (1.5% to 2%). The latter term denotes a type of denervation-induced pain that accompanies a sensory deficit. Septic and aseptic meningitis have been described as rare complications of glycerol rhizolysis.

All of these percutaneous techniques can be repeated in case the pain recurs. In general, however, the sensory deficit tends to worsen with each new intervention. The success rate of repeated procedures is also not as high as that of the initial procedure, though freedom from pain is still achieved in about 70% of patients. The question remains open whether the newly introduced “pulsed radiofrequency” technique, in which the nerve is heated only to a maximum temperature of 42°C, can truly fulfill the hopes that are being placed in it (e5, e6). One advantage of this technique appears to be that, because it is non-destructive, it is unlikely to cause new deafferentation pain.

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**Figure 3:**
Radiosurgical planning of a Gamma Knife treatment.
The target volume is centered on the trigeminal nerve and the radiation dose is 80-90 Gy. The brainstem must be meticulously spared.
Special situations

Trigeminal neuralgia in multiple sclerosis

Some 2% of all patients with multiple sclerosis develop trigeminal neuralgia at some point in the course of their illness. On the other hand, some 2.5% of all patients with trigeminal neuralgia have it as a manifestation of multiple sclerosis (e7). Traditionally, these patients were treated with destructive operative methods once all of the options for pharmacotherapy had been exhausted. The results appear to be no worse than when these procedures are performed for idiopathic trigeminal neuralgia (e8). More recently, microvascular decompression for the treatment of MS-associated trigeminal neuralgia has come under study. A certain degree of neurovascular conflict was found in nearly 50% of all cases in one clinical series, though one must bear in mind that the subjective component of the surgeon's intraoperative judgment necessarily plays a role in generating this figure (e9). The long-term postoperative results were significantly worse than those of microvascular decompression for idiopathic trigeminal neuralgia: only about 40% of patients enjoyed long-term improvement.

Atypical trigeminal neuralgia

This term refers to facial pain that does not meet the criteria listed above for typical trigeminal neuralgia. In particular, the affected patients have not only paroxysmal attacks of lancinating...
pain, but also continuous background pain, perhaps accompanied by dysesthesia and a sensory deficit. Atypical trigeminal neuralgia has many causes, including the following:

- a late development in an initially typical case of trigeminal neuralgia
- postherpetic neuralgia
- anesthesia dolorosa after destructive procedures on the trigeminal nerve
- some of the symptomatic types of trigeminal neuralgia.

Destructive procedures are considered to be of no value in patients who already have a sensory deficit. In such cases, electrical stimulation of the Gasserian ganglion through an implanted neurostimulator may be useful (e10). A recent study showed that microvascular decompression can also be highly effective against atypical trigeminal neuralgia, as long as a definite neurovascular conflict is demonstrated (18).

Conclusions
The most important developments in the treatment of trigeminal neuralgia in the past decade are the general acceptance of the vascular compression hypothesis for the etiology of this condition and the resulting opportunity for curative treatment with microvascular decompression. Elderly or frail patients can be alternatively treated with minimally invasive percutaneous methods or Gamma Knife radiosurgery. All of these techniques are destructive to neural tissue and therefore have the disadvantage of producing a sensory deficit. They are also associated with a greater likelihood of recurrent trigeminal neuralgia: whereas about half of all patients remain free of pain at 5 years. If the pain does recur, the percutaneous procedure can be repeated to good effect.

Conflict of Interest Statement
PD Dr. med. Freynhagen has received lecture and consulting fees from the following companies: Grünenthal, Janssen, Lilly, Böhringer, Mundipharma, Organon, Pfizer, and Schwarz-Pharm. Dr. med. Horstmann and Prof. Dr. med. Steiger declare that they have no conflict of interest as defined by the guidelines of the International Committee of Medical Journal Editors.

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REFERENCES
For e-references please refer to the additional references listed below.

ADDITIONAL REFERENCES

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