SPECIAL ARTICLE

# Practice Parameter: The diagnostic evaluation and treatment of trigeminal neuralgia (an evidence-based review)

## Report of the Quality Standards Subcommittee of the American Academy of Neurology and the European Federation of Neurological Societies

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## ABSTRACT

**Background:** Trigeminal neuralgia (TN) is a common cause of facial pain.

**Purpose:** To answer the following questions: 1) In patients with TN, how often does routine neuroimaging (CT, MRI) identify a cause? 2) Which features identify patients at increased risk for symptomatic TN (STN; i.e., a structural cause such as a tumor)? 3) Does high-resolution MRI accurately identify patients with neurovascular compression? 4) Which drugs effectively treat classic and symptomatic trigeminal neuralgia? 5) When should surgery be offered? 6) Which surgical technique gives the longest pain-free period with the fewest complications and good quality of life?

**Methods:** Systematic review of the literature by a panel of experts.

**Conclusions:** In patients with trigeminal neuralgia (TN), routine head imaging identifies structural causes in up to 15% of patients and may be considered useful (Level C). Trigeminal sensory deficits, bilateral involvement of the trigeminal nerve, and abnormal trigeminal reflexes are associated with an increased risk of symptomatic TN (STN) and should be considered useful in distinguishing STN from classic trigeminal neuralgia (Level B). There is insufficient evidence to support or refute the usefulness of MRI to identify neurovascular compression of the trigeminal nerve (Level U). Carbamazepine (Level A) or oxcarbazepine (Level B) should be offered for pain control while baclofen and lamotrigine (Level C) may be considered useful. For patients with TN refractory to medical therapy, Gasserian ganglion percutaneous techniques, gamma knife, and microvascular decompression may be considered (Level C). The role of surgery vs pharmacotherapy in the management of TN in patients with MS remains uncertain. ***Neurology*® 2008;71:1183–1190**

## GLOSSARY

**AAN** = American Academy of Neurology; **CBZ** = carbamazepine; **CTN** = classic TN; **EFNS** = European Federation of Neurological Societies; **MS** = multiple sclerosis; **NNH** = number needed to harm; **NNT** = number needed to treat; **OXC** = oxcarbazepine; **RCT** = randomized controlled trial; **RFT** = radiofrequency thermocoagulation; **STN** = symptomatic TN; **TN** = trigeminal neuralgia.

This practice parameter was developed as a joint venture of the American Academy of Neurology (AAN) and the European Federation of Neurological Societies (EFNS) to aid clinicians in the treatment of trigeminal neuralgia (TN).

The International Association for the Study of Pain defines TN as sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve.[1] The annual incidence of TN is 4 to 5 in 100,000.[2]

The latest classification of the International Headache Society[3] distinguishes between classic and symptomatic TN. Classic TN (CTN) includes all cases without an established etiology (i.e., idiopathic, as well as those with potential vascular compression of the fifth cranial nerve). The diagnosis of classic

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Quality Standards Subcommittee members, AAN classification of evidence, Classification of recommendations, Conflict of Interest Statement, and Mission Statement of the Quality Standards Subcommittee (appendices e-1 through e-5), as well as tables e-1 through e-9 and references e1 through e38, are available as supplemental data on the *Neurology*® Web site at www.neurology.org.

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TN also requires that there be no clinically evident neurologic deficit. The diagnosis of symptomatic TN (STN) is made when investigations identify a structural abnormality other than potential vascular compression affecting the trigeminal nerve. Such abnormalities include multiple sclerosis (MS) plaques, tumors, and abnormalities of the skull base.

This practice parameter addresses the following diagnostic questions:

1. How often does routine neuroimaging (CT, MRI) identify a structural cause of TN (excluding vascular contact with compression of the fifth cranial nerve)?

2. Which clinical or laboratory features accurately identify patients with STN?

3. For patients with CTN, does high-resolution MRI accurately identify patients with neurovascular compression?

The pharmacologic portion of this parameter addresses the following questions:

1. Which drugs effectively treat CTN?

2. Which drugs effectively treat STN?

3. Is there evidence of efficacy of intravenous drugs in acute exacerbations of TN?

The surgical portion of this parameter addresses the following questions:

1. When should surgery be offered?

2. Which surgical technique gives the longest pain-free period with the fewest complications and good quality of life?

3. Which surgical techniques should be used in patients with MS?

## DESCRIPTION OF THE ANALYTIC PROCESS

The AAN and EFNS assembled a panel of experts who searched MEDLINE, EMBASE, and the Cochrane library. Searches extended from the time of database inception to December 2007. All searches used the following synonyms for TN: trigeminal neuralgia, tic douloureux, facial pain, or trigeminal neuropathy. The primary search was supplemented by a secondary search using the bibliography of retrieved articles and knowledge from the panel. Only full-length original communications were accepted. Panel members reviewed abstracts and titles for relevance. Two panel members reviewed papers meeting inclusion criteria. An additional panel member arbitrated disagreements.

The methods of classifying evidence adopted by AAN and EFNS are essentially identical (appendix e-2, A–C, on the *Neurology*® Web site at www.neurology.org). The methods for determining the strength of the recommendations—though largely compatible—differ in a few points. The present article uses the AAN's method for determining the strength of recommendation (appendix e-3).

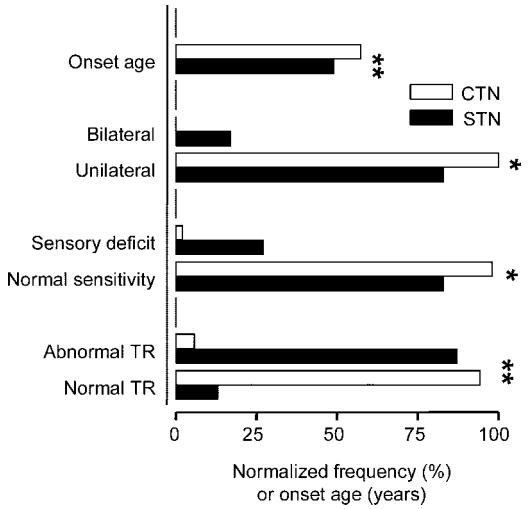
## ANALYSIS OF EVIDENCE

**For patients with TN, how often does routine neuroimaging (CT, MRI) identify a structural cause (excluding vascular contact with compression of the fifth cranial nerve)?** ***Evidence.*** Five articles (one graded Class IV) reported the results of head imaging on consecutive patients diagnosed with TN with normal neurologic examinations (table e-1).[4-8] Four studies included cohorts of patients with TN assembled at tertiary centers with an interest in TN. Because more complicated and potentially less representative patients with TN get treated at such centers, these studies were judged to be at risk for referral bias and thus were graded Class III.[4–7] Yields of brain imaging ranged from 10 to 18%. The most commonly identified abnormalities were cerebello-pontine angle tumors and MS plaques. Combining Class III studies results in a pooled estimate of yield of 15% (95% CI, 11–20).

***Conclusions.*** For patients with TN, routine neuroimaging may identify a cause in up to 15% of patients (four Class III studies). These reported yields are most representative of those expected from referral centers.

**For patients with TN, which clinical or laboratory features accurately identify patients with STN?** ***Evidence.*** We found seven papers (one graded Class IV) studying the diagnostic accuracy of clinical characteristics potentially distinguishing STN from CTN (table e-2).[4-6,8-11] Clinical characteristics studied included the presence of sensory deficits, age at onset, first division of trigeminal nerve affected, bilateral trigeminal involvement, and unresponsiveness to treatment. One study was graded Class III because of a case control design with a narrow spectrum of patients.[9] Four studies were judged to have a moderately low risk of bias because of a cohort design with a broad spectrum of patients. However, these studies collected data retrospectively and were thus graded Class II.[5,6,8,10] We found one prospective Class I study.[4] In these studies, involvement of the first trigeminal division and unresponsiveness to treatment were not associated with a significant increase in the risk of STN. Younger age was significantly associated with increased risk of STN. However, there was considerable overlap in the age ranges of patients with CTN and STN. Thus, although younger age increases the risk of finding STN, the diagnostic accuracy of age as a predictor of STN was too low to be clinically useful in discriminating CTN and STN. The presence of trigeminal sensory deficits and bilateral involvement

**Figure 1** **Mean age and relative frequency of clinical characteristics and abnormal trigeminal reflexes in classic (CTN) and symptomatic trigeminal neuralgia (STN)**




Response to treatment and involvement of first trigeminal division are similar in the two populations. Onset age is lower in CTN than STN ($p < 0.0001$). Bilateral neuralgia and sensory deficits only occur in STN ($p < 0.001$). Trigeminal reflexes (TR) are abnormal in STN (87%) and normal in CTN (94%) ($p < 0.0001$). Data from 10 trials (Class I–III) in 628 patients, detailed in tables e-2 and e-3. *$p < 0.001$; **$p < 0.0001$.

was significantly more common in patients with STN. However, many patients with normal sensation and unilateral involvement of the trigeminal nerve were found to have STN (figure 1).

Five studies addressed the accuracy of trigeminal reflex testing in distinguishing STN from CTN (table e-3).[4,12–15] Trigeminal reflex testing included measurement of the blink reflex. The latency and amplitude of ipsilateral and contralateral facial muscle contractions are measured following stimulation of the trigeminal nerve (usually V1) using standard EMG equipment. One study used a prospective design and was graded Class I.[4] The remaining studies either used a case control design with a narrow spectrum of patients or employed retrospective data collection and were graded Class II or III.[12–15] The diagnostic accuracy of trigeminal reflexes for identifying patients with STN in most studies was relatively high (sensitivity range 59 to 100%, specificity range 93 to 100%). Pooled sensitivity was 94% (95% CI, 91–97); pooled specificity was 87% (95% CI, 77–93).

Four studies addressed the accuracy of trigeminal evoked potentials (table e-4). Two of these studies attained a grade of Class II and two a grade of Class III.[12,16–18] Evoked potentials did not distinguish STN from CTN with high accuracy (sensitivity 60 to 100%, specificity 49 to 76%). Pooled sensitivity was 84% (95% CI, 73–92); pooled specificity was 64% (95% CI, 56–71). Many patients with STN had normal evoked potentials and many patients with CTN had abnormal evoked potentials.

***Conclusions.*** For patients with TN, younger age (one Class I and three Class II studies) and abnormal trigeminal nerve evoked potentials (two Class II and two Class III studies) are probably associated with an increased risk of STN. However, there is too much overlap in patients with CTN and STN for these predictors to be considered clinically useful.

The presence of trigeminal sensory deficits or bilateral involvement of the trigeminal nerves probably increases the risk of STN. However, the absence of these features does not rule out STN (one Class I and two Class II studies).

Involvement of the first division of the trigeminal nerve and unresponsiveness to treatment are probably not associated with an increased risk of STN (one Class I and two Class II studies).

Because of a high specificity (94%) and sensitivity (87%), abnormal trigeminal reflexes are probably useful in distinguishing STN from CTN (one Class I and two Class II studies).

**For patients with classic TN, does high-resolution MRI accurately identify patients with neurovascular compression?** ***Evidence.*** Sixteen papers, the latest published in 2006, studied patients with TN with high-resolution MRI. Nine studies were graded Class IV because they relied on the unmasked findings of the operating surgeon to determine the presence of vascular contact. Table e-5 lists the characteristics of the seven higher-quality studies.[19–25] One study employed a case control design with a narrow spectrum of patients and another was retrospective (Class III).[19,20] Five studies were masked cohort surveys with prospective data collection (Class I).[21–25] The most common reference standard in these Class I studies was the masked comparison of the MRI of the symptomatic side to the asymptomatic side.

Sensitivities and specificities in the Class I through III studies varied widely (sensitivity 52 to 100%; specificity 29 to 93%) and in three Class I studies the association was not significant. The heterogeneity in results may have resulted from differences in the MRI techniques employed.

***Conclusions.*** Because of inconsistency of results, there is insufficient evidence to support or refute the usefulness of MRI to identify vascular contact in CTN or to indicate the most reliable MRI technique.

**Which drugs effectively treat CTN pain?** ***Evidence.*** The literature search identified 15 randomized controlled trials (RCTs) studying medications for TN. In three

of these, the number of patients (less than seven) was too small for meaningful statistical analysis. Of the remaining 12 studies, eight were placebo-controlled trials[26–33] (table e-6) and four used carbamazepine as the comparator[34–37] (table e-7).

Four placebo-controlled studies (Class I or II) totaling 147 patients demonstrated the efficacy of carbamazepine (CBZ).[26–29] The doses of CBZ used ranged from 300 to 2,400 mg a day. The treatment response in these trials was robust, with 58 to 100% of patients on CBZ attaining complete or near-complete pain control as compared to 0 to 40% of patients on placebo. The number needed to treat (NNT) to attain important pain relief was less than 2. CBZ reduced both the frequency and intensity of painful paroxysms and was equally efficacious for spontaneous and trigger-evoked attacks. CBZ was sometimes poorly tolerated with numbers needed to harm (NNHs) of 3 for minor and of 24 for severe adverse events.

Two Class II masked RCTs including a total of 130 patients compared oxcarbazepine (OXC) 600–1,800 mg a day to CBZ in patients with CTN.[36,37] The reduction in pain was equally good for both CBZ and OXC (88% of patients achieving a reduction of attacks by >50%).

Other drugs have each been studied in single trials: baclofen (40 to 80 mg a day) was superior to placebo in reducing the number of painful paroxysms (Class II)[30]; lamotrigine (400 mg a day) was effective as add-on therapy on a composite index of efficacy (Class II)[31]; pimozide (4 to 12 mg a day) was more effective than CBZ (Class II)[35]; and tocainide (12 mg a day) was as effective as CBZ (Class III).[34] Tizanidine was better than placebo in a small study but its effect diminished within 1 to 3 months (Class III).[32]

Small open-label studies (Class IV) have suggested therapeutic benefit from other antiepileptic drugs[38] (e.g., phenytoin, clonazepam, gabapentin, valproate).

Topical ophthalmic anesthesia was ineffective in a Class I placebo-controlled RCT.[33]

***Conclusions.*** Carbamazepine is established as effective for controlling pain in patients with CTN (multiple Class I and II studies). Oxcarbazepine is probably effective for treating pain in CTN (three Class II studies). Baclofen, lamotrigine, and pimozide are possibly effective for controlling pain in patients with CTN (single Class II study for each drug). Topical ophthalmic anesthesia is probably ineffective for controlling pain in patients with CTN (single Class I study). There is insufficient evidence to support or refute the efficacy of clonazepam, gabapentin, phenytoin, tizanidine, topical capsaicin, and valproate for controlling pain in patients with CTN.

**Which drugs effectively treat STN pain?** ***Evidence.*** There are no placebo-controlled studies in patients with STN. The existing studies are small, open-label trials (Class IV) of MS-associated TN using lamotrigine,[39] gabapentin,[40,e1,e2] topiramate,[e3] or misoprostol.[e4,e5]

***Conclusion.*** There is insufficient evidence to support or refute the effectiveness of any medication in treating pain in STN (Class IV studies).

**Is there evidence of efficacy of IV administration of drugs in acute exacerbations of TN?** ***Evidence.*** We were unable to find published RCTs on the use of IV medications to treat TN pain. One Class IV study[e6] reported three patients who responded quickly to IV fosphenytoin.

***Conclusion.*** There is insufficient evidence to support or refute the efficacy of IV medications for the treatment of pain from TN (Class IV study).

**When should surgery be considered?** ***Evidence.*** There are no studies dealing specifically with this issue. Two Class IV studies surveying patients who already underwent surgery determined that the majority of these patients stated they would have preferred to have surgery earlier.[e7,e8]

***Conclusion.*** There is insufficient evidence to allow conclusions as to when surgery should be offered (two Class IV studies).

**Which surgical technique gives the longest pain-free period with the fewest complications and good quality of life?** Surgical interventions are best classified according to the principal target: peripheral techniques targeting portions of the trigeminal nerve distal to the Gasserian ganglion, percutaneous Gasserian ganglion techniques targeting the ganglion itself, gamma knife radiosurgery targeting the trigeminal root, and posterior fossa vascular decompression techniques.

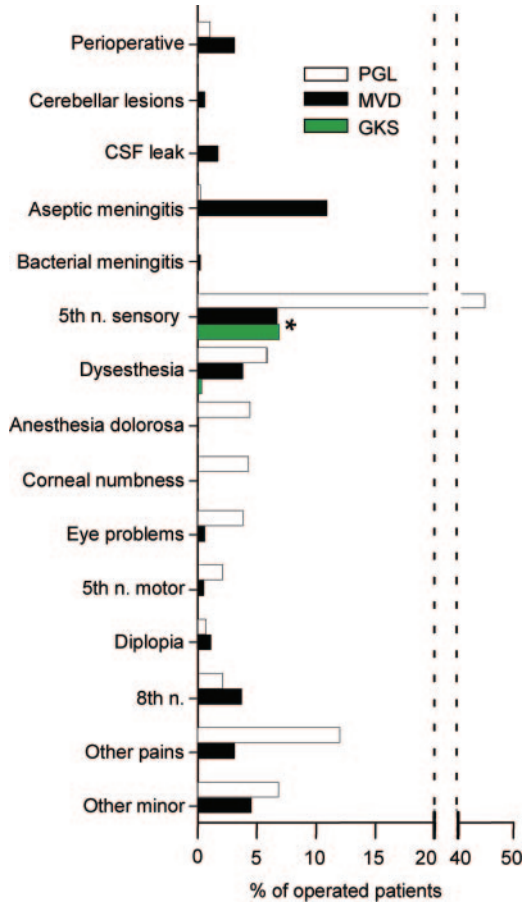
***Evidence.*** Our literature search revealed three Class I RCTs, one Class II prospective cohort study, and a handful of Class III studies where the outcome was independently assessed (explicitly stated). The majority of the evidence was Class IV.

Additionally, the evidence from direct comparisons between different surgical procedures is insufficient.[e9,e10] Study characteristics and semiography of the patients included in our analysis can be found in table e-8 and complications in table e-9 and figure 2.

*Peripheral techniques.* These techniques involve blocking or destruction of portions of the trigeminal nerve distal to the Gasserian ganglia.

Two small RCTs (Class I) on the use of streptomycin and lidocaine compared with lidocaine alone showed no effect on pain.[e11,e12] Other peripheral le-

**Figure 2** Complications of surgery




Frequency (%) of complications with surgical procedures for trigeminal neuralgia. PGL = percutaneous Gasserian lesions (includes radiofrequency thermocoagulation, glycerol rhizotomy, balloon compression); MVD = microvascular decompression; GKS = Gamma knife surgery. Perioperative complications: pneumonia and deep vein thrombosis. Data from 14 trials (Class III) in 2,785 operated patients, detailed in table e-9. *Many Class IV studies on GKS report trigeminal sensory disturbances in 9–37% of patients.

sions (including cryotherapy, neurectomies, alcohol injection, phenol injection, peripheral acupuncture, radiofrequency, and thermocoagulation) have all been reported as case series with no independent outcome assessment (Class IV). These studies showed that 50% of patients had a recurrence of pain after 1 year. The morbidity associated with the peripheral procedures was low.

*Percutaneous procedures on the Gasserian ganglion (rhizotomies).* These techniques[e13] involve penetration of the foramen ovale with a cannula and then controlled lesion of the trigeminal ganglion or root by various means: thermal (radiofrequency thermocoagulation [RFT]),[e14] chemical (injection of glycerol),[e15] or mechanical (compression by a balloon inflated into Meckel's cave).[e16]

We only found uncontrolled case series of the effectiveness of these percutaneous procedures. Only two reports on RFT, one on glycerol injection and one on balloon compression, employed independent outcome assessors (Class III).[e16-e19] Ninety percent of patients attain pain relief from the procedures. At 1 year 68 to 85% of patients will be pain free, but by 3 years the number has dropped to 54 to 64%. At 5 years around 50% of patients undergoing RFT are still pain free.

Sensory loss after these percutaneous procedures is present in almost half of patients (figure 2). Less than 6% develop troublesome dysesthesias. The incidence of anesthesia dolorosa is around 4%. Postoperatively, 12% of patients report a discomfort described as burning, heavy, aching, or tiring. Corneal numbness, with the risk of keratitis, occurs in 4% of patients. Problems with other cranial nerves are uncommon, and the major perioperative complication is meningitis, mainly aseptic (0.2%). Up to 50% of patients undergoing balloon compression suffer temporary and rarely chronic masticatory problems.[e17] Mortality is extremely low.[e20]

*Gamma knife surgery.* This procedure aims a focused beam of radiation at the trigeminal root in the posterior fossa. There is one Class I RCT comparing two different regimes.[e21] This study showed no major differences between the gamma knife techniques used.

We found three case series (Class III) which used independent outcome assessment and provided long-term follow-up.[e22-e24] One year after gamma knife therapy complete pain relief with no medication occurs in up to 69% of patients. This falls to 52% at 3 years. Pain relief can be delayed for a mean of 1 month.[e25]

In the Class III studies, sensory complications occur in an average of 6% of patients. In large Class IV series, facial numbness is reported in 9 to 37% of patients (though it tends to improve with time) and troublesome sensory loss or paresthesias are reported in 6 to 13% (whereas anesthesia dolorosa is practically absent).[e25-e28] No complications outside the trigeminal nerve have been reported. Quality of life improves and 88% are satisfied with the outcome.[e24]

*Microvascular decompression.* This is a major neurosurgical procedure that entails craniotomy to reach the trigeminal nerve in the posterior fossa. Vessels compressing the nerve are identified and moved out of contact.

Five reports were identified which used independent outcome assessment (Class III).[e7,e28-e31] Ninety percent of patients obtain pain relief. Over 80% will still be pain free at 1 year, 75% at 3 years, and 73% at 5 years.

The average mortality associated with the operation is 0.2% though it may rise to 0.5% in some reports.[e20,e32] Postoperative morbidity is lowest in

high volume units.[e32] Up to 4% of patients incur major problems such as CSF leaks, infarcts, or hematomas. Aseptic meningitis is the most common complication (occurring in 11% of patients). Diplopia is often transient and facial weakness is rare. Sensory loss occurs in 7% of patients. The major long-term complication is hearing loss which can occur in as many as 10% of patients.

***Conclusions.*** Percutaneous procedures on the Gasserian ganglion, gamma knife, and microvascular decompression are possibly effective in the treatment of TN (multiple Class III studies). The evidence about peripheral techniques is either negative (two Class I studies about streptomycin/lidocaine) or insufficient (Class IV studies for all the other peripheral techniques).

Indirect comparisons of multiple Class III studies suggest that patients undergoing microvascular decompression have a longer duration of pain control than patients undergoing other surgical interventions. However, the lack of direct comparative studies prohibits formal conclusions regarding the relative efficacy of the surgical techniques.

**Which surgical techniques should be used in patients with MS?** ***Evidence.*** There are only small case series reporting treatment outcomes in patients with MS, with a general tendency toward lesser efficacy in this population as compared to patients with CTN.[e33,e34]

***Conclusion.*** There is insufficient evidence to support or refute the effectiveness of the surgical management of TN in patients with MS.

**RECOMMENDATIONS** For patients with TN, routine imaging may be considered to identify STN (Level C).

The presence of trigeminal sensory deficits or bilateral involvement of the trigeminal nerves should be considered useful to identify patients with STN. However, because of poor specificity, the absence of these features is not useful for excluding STN (Level B).

Measuring trigeminal reflexes in a qualified electrophysiologic laboratory should be considered useful for distinguishing STN from CTN (Level B).

Younger age at onset, involvement of the first division of the trigeminal nerve, unresponsiveness to treatment, and abnormal trigeminal evoked potentials should be disregarded as useful for accurately identifying patients with STN (Level B).

To control pain in patients with TN: carbamazepine should be offered (Level A), oxcarbazepine should be considered (Level B), baclofen, lamotrigine, and pimozide may be considered (Level C), and topical ophthalmic anesthesia should not be considered (Level B).

For patients with TN refractory to medical therapy: early surgical therapy may be considered (Level C), and percutaneous procedures on the Gasserian ganglion, gamma knife, and microvascular decompression may be considered (Level C).

**PUTTING THE EVIDENCE INTO A CLINICAL CONTEXT** The initial diagnostic evaluation of a patient with TN naturally focuses on those clinical characteristics known to identify patients with STN. Those characteristics include the presence of trigeminal sensory deficits and bilateral involvement. If after the initial evaluation the clinician remains suspicious of STN, further testing is desirable. Based upon cost, local expertise and availability, and patient preferences, obtaining trigeminal reflex testing or head imaging are both reasonable next steps. Because of a high diagnostic accuracy, MRI might reasonably be foregone in a patient with normal trigeminal reflexes.

The two drugs to consider as first-line therapy in TN are CBZ (200–1,200 mg/day) and OXC (600–1,800 mg/day). Although the evidence for CBZ is stronger than for OXC, the latter may pose fewer safety concerns.[e35]

There is little evidence to guide the clinician on the treatment of patients with TN who fail first-line therapy. Some evidence supports add-on therapy with lamotrigine or a switch to baclofen (pimozide being no longer in use). The effect of other drugs commonly used in neuropathic pain is unknown. There are no published studies directly comparing polytherapy with monotherapy.[e36]

Referral for a surgical consultation seems reasonable in patients with TN refractory to medical therapy. Some TN experts believe patients with TN failing to respond to first-line therapy are unlikely to respond to alternative medications and suggest early surgical referral.[e36]

**RECOMMENDATIONS FOR FUTURE RESEARCH** To establish a better estimate of the yield of routine brain imaging in identifying patients with STN, we need a population-based study of consecutive, newly diagnosed patients with TN all undergoing head imaging.

To improve our knowledge of the diagnostic accuracy of clinical characteristics and electrophysiologic studies to distinguish STN from CTN, we need prospective cohort surveys of large populations of patients with TN all undergoing standardized diagnostic assessments reported using STARD criteria.[e37]

It would also be useful to determine if finding a neurovascular contact on high-resolution MRI accurately identifies patients who will respond to microvascular decompression. This question could be

answered with a prospective study comparing long-term outcomes in patients with TN undergoing microvascular decompression with and without neurovascular contact identified on preoperative high-resolution MRI.

The efficacy of new drugs and, in particular, surgical interventions, needs to be determined in well-designed RCTs. Although double-blinded studies are impractical for surgical trials, randomized treatment allocation and independent outcome assessment would go a long way to establish the efficacy of the surgical techniques.

The optimal timing of surgical referral remains a crucial question. How many different drugs should be tried before referring a patient for surgery? What is the likelihood that a patient with TN failing OXC or CBZ will respond to alternative drugs? These are questions that could be answered by a large prospective cohort survey of patients with TN treated in a standardized, stepwise fashion.

## DISCLOSURE

The authors report the following conflicts of interest: Dr. Gronseth has received speaker honoraria from Boeheringer Ingelheim, Pfizer, and GlaxoSmithKline, and has been compensated by Ortho-McNeil for serving on a safety monitoring committee; Dr. Cruccu has given lectures for Pfizer and estimates 5% of his clinical effort is spent on trigeminal reflex testing; Dr. Alksne estimates 10% of his clinical effort is spent on surgery for trigeminal neuralgia; Dr. Argoff estimates 25% of his clinical effort is spent on Medtronic Pump, 25% on trigger point injections, 20% on epidural steroid injections, and 30% on botulinum toxin injections; Dr. Brainin has nothing to disclose; Dr. Burchiel holds equity in Medtronic, Boston Scientific, and Pfizer; Dr. Nurmikko holds financial interests in Boehringer Ingelheim, Pfizer UK, Grunenthal, Sanofi Pasteur MSD, and UCB/Scwarz Pharma; Dr. Nurmikko estimates 10% of his clinical effort is spent on MRI; Dr. Zakrzewska holds financial interests in UCB Pharma and has received research support from the Bupa Foundation for decision analysis in trigeminal neuralgia.

## DISCLAIMER

This statement is provided as an educational service of the American Academy of Neurology. It is based on an assessment of current scientific and clinical information. It is not intended to include all possible proper methods of care for a particular neurologic problem or all legitimate criteria for choosing to use a specific procedure. Neither is it intended to exclude any reasonable alternative methodologies. The AAN recognizes that specific patient care decisions are the prerogative of the patient and the physician caring for the patient, based on all of the circumstances involved. The clinical context section is made available in order to place the evidence-based guideline into perspective with current practice habits and challenges. No formal practice recommendations should be inferred.

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