Trigeminal Neuralgia and Other Facial Neuralgias
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Chapter 127

TRIGEMINAL NEURALGIA AND OTHER FACIAL NEURALGIAS

Trigeminal Neuralgia

International Headache Society (IHS) code: 13.1
World Health Organization (WHO) code: G44.847 [G50.00]

Short description: Trigeminal neuralgia is a unilateral disorder characterized by electric-shock-like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve. Pain is commonly evoked by trivial stimuli including washing, shaving, smoking, talking, and/or brushing the teeth (trigger factors), and frequently occurs spontaneously. Small areas of the nasolabial fold and/or chin may be particularly susceptible to the precipitation of pain. The pains usually remit for variable periods (28).

Other terms: Tic douloureux

EPIDEMIOLOGY

Trigeminal neuralgia (TN) is relatively uncommon. The incidence rate of TN was estimated at 4.3 per 100,000 population in Rochester, Minnesota, between 1945 and 1984 (35). A more recent community-based survey of medical records of several general practices in London arrived at an annual incidence of 8 per 100,000 population and a lifetime prevalence of 70 per 100,000 (95% confidence index [CI] 40–100) (49). The incidence rate is higher in women (5.9) than in men (3.4). The incidence rates for both sexes increase with age and are highest in the age group of 60 years and older (35).

ANATOMY AND PATHOLOGY

Compression, distortion, or stretching of the trigeminal roots by arteries, veins, vascular malformation, skull base bony anomalies, or slowly growing tumors can cause the pain of TN (21). Compression of the nerve root by a blood vessel is often the cause in the majority of cases (10, 30, 38, 74). Jannetta initially reported an offending blood vessel in 100% of his TN cases (including those found at reoperation). Subsequently, many series cited a high percentage of vascular compression as well, ranging from 79 to 97 (10, 18, 30, 74, 77). By contrast, a tumor is found in 2 to 3% of cases (13, 18, 74) and a small arteriovenous malformation in 0.5 to 2% (18, 74).

Tumors, usually posterior fossa meningiomas or neuromas giving rise to symptoms indistinguishable from typical TN rather than numbness and atypical pain, are usually seen to distend the root, rather than invade it (13). Direct infiltration of the nerve or ganglion tends to give rise to sensory loss and nonparoxysmal pain.

Approximately 2 to 4% of patients with TN have multiple sclerosis (MS), although it is rarely the first manifestation of the disease (32, 69). MS should be considered in patients younger than 50, especially with history of bilateral TN. Very rarely, TN may accompany syringohydralia or develop after brainstem infarction (81). In all, symptomatic TN is likely to explain approximately 5 to 7% of cases in unselected populations; the rest are due to vascular compression of the root or the cause remains unknown.

Pathophysiology

There is general acceptance that because no or only minimal sensory loss is encountered in TN, most of the trigeminal pathways must remain anatomically intact. On the other hand, the pain in its most stereotyped presentation (including remissions) is likely to reflect relatively limited neuronal dysfunction either in the trigeminal nerve or its central connections. Two points are important to note. First, there is compelling evidence from large case
The pain of TN is almost always unilateral. Even in rare bilateral cases, the two sides react independently to various stimuli and the attacks come unsynchronized. Paroxysms are frequently followed by refractory periods, up to minutes (40), during which the previous stimulus, if repeated, fails to provoke an attack. TN has a tendency for weeks- or even months-long remissions, especially in the early phase of the condition. Not all TN starts with the classical features. Pretrigeminal
neuralgia, an intermittent unilateral trigeminal pain lacking some of the characteristics of TN pain, can occur (22). The pain, however, responds to carbamazepine and later develops into a definite form of TN. Conversely, it has been suggested that with time, many patients with a classical presentation develop other, less paroxysmal pain, in effect making “typical” cases “atypical,” with poorer treatment outcomes (11). If true, this will affect timing of treatment, which is discussed below.

Several other facial pain conditions bear similarities to TN and are reviewed in Table 127-1. This outlines the importance of obtaining a detailed pain description of the quality of pain (Table 127-1). There are several caveats that one should be aware of. About 10% of patients will not respond to carbamazepine, and up to 40% do not demonstrate trigger areas on clinical examination (72). Occasionally, patients describe redness and swelling of the face after a severe attack (60), and when the first division is involved, it is seen more frequently with SUNCT syndrome (short-lasting, unilateral, neuralgiform headache with conjunctival injection and tearing) can be difficult (27). Although several groups have shown sensory changes in TN (53,58), most unoperated patients show only minimal changes with conventional bedside testing. Indeed, substantial sensory loss should raise the possibility of a symptomatic TN, and investigations should be directed accordingly.

INVESTIGATIONS

Advanced imaging techniques form the backbone of etiologic assessment and also provide helpful information for the neurosurgeon in case operative treatment is being considered. Whenever possible, each patient with a new diagnosis of TN should undergo magnetic resonance imaging (MRI), preferably using 3-D reconstruction techniques to (a) rule out other causes than vascular compression (MS, tumor) and (b) assess the relationship of the nerve to the neighboring blood vessels. Whole-brain T2-weighted images are the minimum with suspected MS.

Several groups have shown that the identification of a treatable cause—compression of the nerve at the root entry zone by an overlying blood vessel—is possible with considerable reliability. In a pioneering study, Meanev and coworkers (1995) used 3-D fast imaging with steady-state precession (FISP) reconstruction images to demonstrate blood vessels as high signal intensity structures around the nerve in any orientation (52). While arteries were easily identifiable, veins could be properly visualized only after enhancement with intravenous gadolinium. The method was validated in 50 patients with 55 symptomatic nerves (5 patients had bilateral TN) who underwent posterior fossa exploration. In the 52 explorations carried out, neurovascular contact was confirmed in all of the 49 cases suggested by 3-D FISP. A further case with negative preoperative imaging was shown to lack any vascular contact at operation. There were two false-negative scans, and no false positives, leading to a sensitivity of 100% and a specificity of 96%. A similar study using identical imaging parameters confirmed these results (61).

Akimoto et al. combined 3-D FISP with 3-D constructive interference in steady state (CISS) to improve the visualization of the trigeminal nerve (1). This combined imaging technique improves the visualization of structures in the cerebrospinal fluid space due to its use of heavily T2-weighted source images. Patients who underwent microvascular decompression (MVD), there was excellent agreement between the preoperative imaging and operative diagnosis in all but one, and a compression was found by an undiagnosed vessel in this patient (1). Even if this is only an observational study without radiologic blinding, the results are impressive. Routine MRI techniques, although less time consuming, do not produce images accurate enough to determine the relationship of the nerve and blood vessels in its vicinity.

MRI provides the clinician with the anatomic substrate of clinically diagnosed TN but does not enable one to diagnose TN because vascular contacts are reported in 8% of asymptomatic nerves (52). In other words, the functional significance of a contact between a vessel and the trigeminal nerve is unclear in patients who do not have TN clinically. Also, false-negative findings remain a possibility, as small arteries (diameter <1 mm), arachnoidal thickenings, and similar less common causes are not detected using MRI (74). Despite these shortcomings, 3-D reconstruction MRI in trigeminal neuralgia, when used in conjunction with critical assessment of the quality of the pain, provides a rare opportunity for a far more precise diagnosis than is the norm in painful conditions involving the head and face.

Other methods, such as quantitative sensory testing and laser-evoked potentials, provide limited diagnostic benefit, mainly in quantifying afferent dysfunction, and do not have a therapeutic role, either in decision making or follow-up.

MANAGEMENT

TN remains one of the few chronic pain conditions for which there are several excellent therapeutic methods available. The obvious target is to tailor treatment to each individual’s clinical situation, such as severity of pain, degree of disability, general operability, and patient preferences (57,86). There has been a general shift away from the traditional approach of considering surgical treatment only when pharmacotherapy fails, in favor of early surgical intervention, especially in younger patients. It is believed that the risk of developing other types of neuropathic pain, which might be refractory, can develop if early intervention is not sought (11,57).
<table>
<thead>
<tr>
<th>Condition</th>
<th>Location of Pain</th>
<th>Duration of Pain or Attack</th>
<th>Shooting Pain or Paroxysms</th>
<th>Autonomic Symptoms</th>
<th>Pain Relief with Carbamazepine</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trigeminal neuralgia</td>
<td>Usually 2nd and/or 3rd division</td>
<td>&lt;1 second to minutes</td>
<td>Yes</td>
<td>Variable, mild</td>
<td>Good</td>
<td>Trigger areas, mechanical trigger factors</td>
</tr>
<tr>
<td>Glossopharyngeal neuralgia</td>
<td>Pharynx, ear</td>
<td>&lt;1 second to minutes</td>
<td>Yes</td>
<td>None</td>
<td>Good</td>
<td>Pain provoked by swallowing, syncope attacks</td>
</tr>
<tr>
<td>Nervus intermedius neuralgia</td>
<td>Deep in ear</td>
<td>Seconds or minutes</td>
<td>Yes</td>
<td>None</td>
<td>Good</td>
<td>Rare, maybe confused with glossopharyngeal neuralgia</td>
</tr>
<tr>
<td>SUNCT</td>
<td>Forehead, retrobulbar</td>
<td>5 seconds to several minutes</td>
<td>Yes</td>
<td>Prominent</td>
<td>None</td>
<td>Almost exclusively in women; rare</td>
</tr>
<tr>
<td>Cluster headache</td>
<td>Retrobulbar, cheek, chin</td>
<td>20 minutes to several minutes</td>
<td>Only superimposed on deep dull pain</td>
<td>Prominent</td>
<td>Modest</td>
<td>Triptans help</td>
</tr>
<tr>
<td>CPH</td>
<td>Forehead, retrobulbar</td>
<td>2 to 45 minutes</td>
<td>No</td>
<td>Prominent</td>
<td>None</td>
<td>Alcohol provokes</td>
</tr>
<tr>
<td>Cracked tooth syndrome</td>
<td>Upper jaw</td>
<td>Seconds</td>
<td>Yes</td>
<td>None</td>
<td>None</td>
<td>Provided on biting and chewing</td>
</tr>
<tr>
<td>Jabs and jolts syndrome</td>
<td>Anywhere in the head</td>
<td>Seconds</td>
<td>Yes</td>
<td>None</td>
<td>None</td>
<td>No precipitating factors</td>
</tr>
<tr>
<td>Postherpetic neuralgia</td>
<td>Forehead, eye, cheek (rarely)</td>
<td>Continuous</td>
<td>Superimposed on background pain</td>
<td>Variable, mild</td>
<td>Usually modest</td>
<td>History of shingles, Tactile allodynia, Sensory impairment</td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td>Forehead, neck, temples</td>
<td>Continuous</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Jaw claudication</td>
</tr>
</tbody>
</table>

CPH, chronic paroxysmal hemicrania; SUNCT, short-lasting, unilateral neuralgiform headache with conjunctival injection and tearing.

Sources: Bruyn (8), Goadsby and Lipton (26), Nurmikko and Eldridge (59), Sjaastad et al. (76), and Zakrzewska (87).
Pharmacotherapy

A summary of drugs commonly used in the treatment of TN is listed in Table 127-2. Carbamazepine remains the only drug that has been subjected to several placebo-controlled randomized controlled trials in large patient populations (12,37,55). All three trials showed its superiority over placebo. One study demonstrated a reduction in intensity and number of bursts (12) while the other two indicated benefit on global pain relief (37,55). The composite numbers needed to treat calculated from these data is 1.7 (95% CI, 1.3, 2.2) (75). Baclofen was superior to placebo in a cross-over trial of 10 subjects only (23), and lamotrigin as an add-on medication in another with 13 participants (88).

Comparative studies suggest that oxcarbazepine is as effective as carbamazepine with a similar side effect profile (4,90). Indeed, oxcarbazepine is licensed for TN in some countries (4). Similarly, tocainide in a single cross-over trial of 10 subjects only (23), and lamotrigin as an add-on medication in another with 13 participants (88).

Comparative studies suggest that gabapentin, sodium valproate, clonazepam, and intravenous lidocaine may be effective, but the data are too limited for definite conclusions (2,6,14,75). In severe exacerbations of TN, intravenous phenytoin, fosphenytoin, and sodium valproate appear useful. Misoprostol has been reported to be useful in a small case series of patients with MS and TN (17).

Customarily, TN treatment commences with carbamazepine and many patients respond within 48 hours, after which the dose is titrated to the lowest level controlling the pain. A single-blind study suggested that best results are obtained at a serum concentration of 20 to 40 µmol/L (82). In practice, up-titration is usually associated with increasing side effects, and balancing the two is recommended for best results. Combination therapy with lower doses of two preparations is used successfully in some patients but no level I studies support this strategy. In a long-term follow-up study, half of the patients originally on carbamazepine monotherapy had stopped at 10 years due to lack of effect or problems with tolerability (80).

Neuroablative Procedures

The trigeminal nerve can be interrupted at several sites during its course including the peripheral nerve, ganglion, root, and mesencephalon. In practice, the ganglion and the root remain the main targets for these procedures. In some particular circumstances, peripheral procedures (e.g., cryosurgery, neuroectomy, alcohol blockade) can be considered, but the results are clearly inferior to other available treatments and are not discussed further in this chapter. Similarly, medullary tractotomy, while of great historical interest, is not among treatments that are applied today. There are four major methods that can be used to interrupt afferent pathways at the level of the ganglion or root. In balloon compression, a small-sized Fogarty catheter is passed through a needle inserted just through the foramen ovale and inflated for 1 to 5 minutes, filling the Meckel cave to its maximum capacity. With glycerol gangliolysis, 0.1 to 0.4 mL of mildly neurotoxic glycerol is injected into the trigeminal cistern. The radiofrequency method
entails stimulation of the retroganglionic fibers corre- 

csponding to the painful division(s), and their thermoco- 

agulation. Stereotactic radiosurgery is carried out by first 

identifying the root and the putative root entry zone, and 

then targeting it with several hundred intercepting beams 

of gamma radiation. All these procedures lead to a varying 

degree of sensory loss and weakness of the masseter mus- 

cle. Following balloon compression and radiofrequency le-

sioning, the effect is immediate, while glycerol gangliolysis 

may take up to 7 days to work. The effect from stereotactic 

radiosurgery takes usually 2 to 3 months to peak.

Large case series with variable follow-up have been pub- 

lished. A systematic review suggests that these methods 

are relatively similar in terms of immediate pain relief, rate of 

recurrence, and adverse effects (45). The choice between 

them is likely to depend more on clinical factors than on 

local circumstances and surgeons’ preferences.

Neuroablative procedures provide only temporary re-

lief, and most cases recur in a few years: 40 to 50% ex-

perience a recurrence at 36 months (45). Also, all these 

procedures are associated with a risk of troublesome post-

treatment dysesthesia that may significantly reduce the pa-

tient’s quality of life, and which may be more common in 

radiofrequency lesioning than other methods (45,63). The 

problem with post procedural dysesthesia is that symptom 

treatment is difficult. A further problem arises if and when 

the neuralgia recurs, as repeat ablations are likely to carry 

an increased risk of worsening of dysesthesia. Anesthesia 

dolorosa is seen in less than 1% of patients who have un-

dergone a neuroablative procedure. Furthermore, corneal 

sensory loss and keratitis have been reported and are more 

common after radiofrequency lesioning than other treat-

ments (45,63). Finally, pain relief following neuroablution 

is less complete in atypical than typical TN, and sensory 

complications are more common (7,43,56,89).

Microvascular Decompression

MVD, which was popularized by Jannetta (30), is the most 

invasive treatment for TN but is the only therapy that pur-

tports to directly address the underlying cause of the parox-

ysmal pain. Pain relief following MVD is almost always 

immediate, and its long-term results are superior to other 

procedures. At 1 to 2 years, 75 to 80% of patients remain 

without pain. Eight to 10 years post-MVD, 58 to 64% are 

asymptomatic and 4 to 12% suffer minor recurrence only 

(3,10,30,38,64).

MVD involves exposing the trigeminal nerve and iden-

tifying the offending blood vessel(s). The most common 

finding is a segment of the superior cerebellar artery com-

pressing the nerve at the root entry zone (30,74). After free-

ing the offending vessel, the operator places a piece of felt 

between it and the nerve to ensure a permanent separation.

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ing the offending vessel, the operator places a piece of felt 

between it and the nerve to ensure a permanent separation.

Which Treatment to Choose?

Treatment options for TN are many, and a well-informed 

patient should participate in treatment decisions. Also, the 

decision process should address the patient's quality of life, 

which depends on many factors in addition to pain relief 

(86).

Barring no universal guidelines for the management of 

TN, it is suggested to perform imaging studies, which 

can provide a definite etiologic answer and can categorize 

many cases into either typical or atypical neuralgia. Sub-

sequently, treatment choices are recommended based on 

the best balance in the individual patient between safety, 

risk of adverse effects, likelihood of recurrence, and patient 

preference. For example, an undisputed compression in a 

patient with no major operative contraindications would 

be a valid indication for either pharmacotherapy, neurode-

structive procedures, or MVD. In atypical TN with no evi-

dence on MRI of vascular compression, pharmacotherapy 

remains the safest first-line treatment.

There is no consensus regarding the choice of treatment 

for recurrent trigeminal neuralgia. Neurodestructive pro-

cedures can be repeated, although the effect may be of 

shorter duration and deafferentation as such may lead to 

increasing pain. Stereotactic radiosurgery may be repeated 

once. Repeat MVD may occasionally reveal a missed ves-

sel, although the increasing use of preoperative MRI likely 

diminishes this problem.

Glossopharyngeal Neuralgia

HIS code: 13.2

Short description: Glossopharyngeal neuralgia is a severe 

transient stabbing pain experienced in the ear, in the 

base of the tongue, in the tonsillar fossa, or beneath the 

angle of the jaw. The pain is therefore felt in the dis-

tribution of the auricular and pharyngeal branches of 

the vagus and glossopharyngeal nerves. It is commonly 

provoked by swallowing, talking, and coughing, and 

may remit and relapse similar to trigeminal neuralgia.

Other terms: Vagoglossopharyngeal neuralgia
Pain has all of the following characteristics:

- Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 minutes and fulfilling criteria B
- Attract attacks are stereotyped in the individual patient.
- There is no clinically evident neurologic deficit.
- Not attributed to another disease (see Note).

Note: Other causes have been ruled out by history, physical examination, and/or special investigation.

Glossopharyngeal neuralgia is in many ways similar to trigeminal neuralgia. Its average age of onset is about 50 years. The attacks of pain come in paroxysms and are lightninglike. The pain occurs in the region of the base of the tongue, in the tonsillar fossa, under the angle of the jaw, or in the ear. Usual triggers are swallowing, chewing, and talking. In addition to sharp pains, clicking, scratching, or foreign body sensations in the throat are reported (70). Spontaneous remissions are common, especially in mild cases (36). Glossopharyngeal neuralgia may be associated with sick sinus syndrome, severe bradycardia, or asystole, resulting in syncope or convulsions (19).

As with TN, the diagnosis of glossopharyngeal neuralgia is based on a history of the characteristic paroxysms of lancinating pain in a patient where neurologic, dental, and imaging studies are normal (8). Some investigators differentiate otalgic and oropharyngeal forms on the basis of the dominant pain (50,62). Case series describe patients with more continuous pain, or with a burning quality to it, and some authors have also used the "typical versus "atypical" dichotomy (67).

With advanced MRI technologies, vascular compression is an increasingly recognized cause of glossopharyngeal neuralgia (5,20,34,54), but to date, estimates of specificity and sensitivity of preoperative MRI remain unknown.

Similarly to TN, medical therapy of glossopharyngeal neuralgia includes carbamazepine, oxcarbazepine, baclofen, phenytoin, gabapentin, and lamotrigine either alone or in combination (2,54). Neurodestructive procedures and microvascular decompression are both advocated (39,67,79). Both open rhizotomy (often involving rhizotomy of the vagus nerve in addition to that of the glossopharyngeal nerve) and MVD carry high success rates in recent series. In one series, none of the 12 patients who underwent rhizotomy experienced recurrence on a mean follow-up of 10 years (79). Similarly, Kondo reported no failures in 16 of the 20 patients who were available for follow-up at a mean of 11 years (39). Also, Sampson et al. had only one recurrence in 29 patients of the original 47 who were followed for a median of 13 years (71). These data contrast with two case series that demonstrated complete pain relief in 79% and 59%, and partial relief in further 10% and 18% of patients, respectively, at a mean follow-up of 4 years (42,67).

Operative mortality is up to 5%. Also, early neurologic and other complications (dysphagia, hoarseness of
voice, and swallowing disturbances) are common, are usu-
ally mild, but may not resolve in 10% of all operated
cases. Percutaneous radiofrequency lesioning has been mostly
used in symptomatic glossopharyngeal neuralgia with im-
pressive results (25).

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Nervus Intermedius Neuralgia

IHS code: 13.3

WHO code: G44.847 [G51.80]

Short description: Nervus intermedius neuralgia is a rare
disorder characterized by brief paroxysms of pain that
are felt deep in the auditory canal. Constant pain and
referred pain to deep facial structures may accompany
the paroxysms.

Other terms: Geniculate neuralgia, Hunt neuralgia

ANATOMY AND PATHOLOGY

The intermedius nerve forms a small sensory branch of
the facial nerve. Both visceral afferent and somatic sen-
sory fibers travel proximally to reach the solitary and
trigeminal nucleus, respectively. The cell bodies of the
sensory afferents are located in the geniculate ganglion
and their peripheral axons reach the skin in the exter-
nal meatus and just behind the ear, overlying the mastoid
process.

PATHOPHYSIOLOGY

Evidence of the role of the geniculate ganglion and the in-
termidius nerve in the generation of neuralgic pain is rel-
atively scanty (9,50). Since Hunt's original suggestion in
1907 (29) that facial palsy, vestibulocochlear symptoms,
pain, and vesicles in the auditory canal represent herpetic
inflammation, interest in this condition has waxed and
waned. Some case series have been published in which
the predominant symptom is pain deep in the ear, and the
pain is mostly, but not exclusively, sharp and intermittent
(48,68). There is no direct evidence that the intermedius
nerve and/or geniculate ganglion are the origin of neu-
ralgic pain. Still, based on the original hypothesis, some
surgeons have carried out extensive neurodestructive pro-
cedures with moderately successful results. It is of note
that the published case series describe procedures that al-
most invariably approached several nerves (intermedius
and branches of the glossopharyngeal and vagal nerves) dur-
ing operation (48,68). Surgeons have argued that there
is significant overlap in sensory innervation of the region,
and single nerve approaches are unlikely to yield good re-
results (48,68).

CLINICAL FEATURES

IHS revised criteria (ICHD-II) for nervus intermedius neu-
ralgia are as follows:

A. Pain paroxysms of intermittent occurrence lasting for
seconds or minutes in the depth of the ear

B. Presence of a trigger area in the posterior wall of the
auditory canal

C. Not attributed to another disease (see Note)

Note: Other causes, in particular a structural lesion, have
been ruled out by history, physical examination, and spe-
cial investigations.

Many case reports agree with Hunt's original descrip-
tion of combined otalgia (pain in the ear) and deep and
vaguely described facial pain (originally named prosopal-
gia by Hunt). The IHS classification has set out criteria for
the neuralgia, which require the presence of both parox-
ysmal pain and a trigger area in the posterior wall of the
auditory canal. Reports conform to these criteria. The clin-
ical diagnosis is almost entirely based on pain description
and ruling out other neurologic and otorhinologic causes.
No report has been published on the use of MRI to identify
putative neurovascular compression.

MANAGEMENT

Conventional pharmacotherapy similar to that with other
cranial neuralgias should be tried and surgical manage-
ment considered a last resort.

OTHER CRANIAL NEURALGIAS

Infection (e.g., herpes zoster) and injury can lead to neu-
ropathic pain, affecting various branches of the trigemi-
nal nerve. The most common of these is undoubtedly pos-
therpetic neuralgia affecting the ophthalmic branch of the
trigeminal nerve (13.15.2 [B02.02]). Injury to any termi-
nal branch of the trigeminal nerve may lead to a painful
aftermath, and the supraorbital or nasociliary branches
of the ophthalmic nerve, infraorbital branch of the max-
illary nerve, and lingual branch of the mandibular seem
most commonly affected. Wisdom tooth removal is associ-
ated with injury to the inferior alveolar nerve, although
it is rarely painful. Common to all these conditions is
that they represent focal painful neuropathies and do not
usually share the paroxysmal characteristics of primary
cranial neuralgias. The pain is usually described as con-
tinuous, fluctuating, sharp, or burning, with some lanci-
nating pain superimposed. Clinical examination confirms
sensory impairment and cutaneous allodynia, with ten-
derness at the exit foramina and a positive Tinel sign in
some. Management is similar to that in neuropathic pain
conditions in general and includes tricyclics, opioids, gabapentinoids, capsaicin, and lidocaine patch. In intractable cases, trigeminal nerve stimulation or intracranial stimu-
lation may be considered. On occasion, patients may present with neurologic pains confined to the territory of one single branch (e.g., auriculotemporalis or infraorbitalis). Local pathology may be responsible for some of these cases, although a cause frequently cannot be established. It is debated whether to consider these cases under separate entities. Indeed, some will evolve into classical TN.

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