Chapter 127

Trigeminal Neuralgia and Other Facial Neuralgias

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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 syringobulbia 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

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series that simple decompression at the dorsal root entry zone renders most patients pain-free for years—in effect, cures them. Second, typical (or classical) features of TN are exceedingly rare in conditions that affect the trigeminal pathways outside the dorsal root entry zone. Even in MS-related TN, there is a plaque near the dorsal root entry zone, effectively disrupting the functions of the central terminals (24).

The myelin at the dorsal root entry zone is derived from astrocytes and is considered less tolerant to compression than that produced in the peripheral nerve by Schwann cells. Neuropathologic studies of operative specimens from the dorsal root entry zone in patients with vascular compression and in patients with MS and no compression show significant demyelination and axons in direct opposition without intervening glial processes (16,46,47,66). This is thought to favor ephaptic transmission, that is, the transfer of nerve impulses from one set of fibers to another (15,46,47,59). Should this happen between fibers mediating tactile impulses and those mediating painful impulses from trigger zones, the result expectedly would be what is seen clinically, touch-evoked pain. From experimental nerve models it is hypothesized that affected axons acquire a state of hyperexcitability, which renders them capable of producing intense firing for seconds or minutes when stimulated (15,16,46). Compression by a blood vessel in this regard is likely to be significant; intraoperative electrophysiologic studies showed delayed transmission in the affected trigeminal root, which normalized immediately after decompression (43). It has also been suggested that secondary changes at the level of the ganglion cells (whether due to hitherto unknown mediators or secondary degenerative processes in ganglion cell somata) make these cells susceptible to cross-excitation (15,59,65). If this were a synchronized event, it could lead to the simultaneous activation of several cells, producing the explosive pain pattern that TN represents (15).

Although far from proven, this hypothesis explains why many treatments are effective in TN. Decompression removes the cause, neuroablative procedures interfere with the cell-to-cell cross-talk and hyperexcitability, and medication effectively prevents excessive firing. Many aspects of TN remain unknown, however, including reasons for spontaneous remissions, why compression in only a minority leads to TN, and the mechanism of TN in the small minority of patients without structural abnormality.

CLINICAL FEATURES

TN remains a clinical diagnosis, based on the patient's description of pain. Crucial features include severe stabbing, or electric-shock–like pains, usually unilateral and located in the distribution of the trigeminal nerve. IHS diagnostic criteria (Revised International Classification of Headache Disorders [ICHD-II]) for TN are as follows:

A. Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions in the trigeminal nerve and fulfilling criteria B and C.
B. Pain has at least one of the following characteristics:
  1. Intense, sharp, superficial, or stabbing
  2. Precipitated from trigger areas or by trigger factors
C. Attacks are stereotyped in the individual patient.
D. There is no clinically evident neurologic deficit.
E. Not attributed to another condition

The IHS criteria of "symptomatic" TN are identical to those of "classical" TN, except for the demonstration of a nonvascular lesion (MS, tumor, etc.) in classical TN and the allowance of sensory impairment. Vascular compression, however, is considered part of the "classical" category.

Sometimes, TN is categorized either as "typical" and "atypical" irrespective of its cause, while pain arising from the trigeminal nerve but without all characteristics of TN and frequently demonstrating features common to any neuropathic pain is referred to as *painful trigeminal neuropathy*. This practice has developed from observations that the treatment results are better in typical versus atypical cases, irrespective of the surgical method chosen, while most are contraindicated in trigeminal neuropathy (7,43,84,89).

Generally, the key to the right diagnosis is careful history taking that includes both the patient's spontaneous description of the pain and a semistructured practitioner's interview, probing the specific pain characteristics. Not all patients volunteer that their pain is "electric-shock–like," "lightninglike," lancinating, or stabbing, and many will insist that it is exceedingly variable and lasts well beyond the 2 minutes allowed by the IHS criterion. To the patient, the experience of the intense paroxysm may be more relevant than differentiating the duration of a single stab. A very poorly controlled trigeminal pain may be described as continuous by the patient attempting to highlight the relentlessness of the rapidly recurring paroxysms.

Paroxysm trigger factors are mechanical by nature, although rarely, extreme cold, smell, or taste has been implicated (73). Sometimes, patients develop methods of avoiding pain triggers, including avoiding eating and drinking, which leads to dehydration in severe cases.

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 time 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*

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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 diagnosed on the basis of history. This underlines 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, a distinction from 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, Meaney 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. In 24 consecutive 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).

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TABLE 127-1 Differential Diagnosis of Trigeminal Neuralgia

Condition	*Location of Pain*	*Duration of Pain or Attack*	*Shooting Pain or Paroxysms*	*Autonomic Symptoms*	*Pain Relief with Carbamazepine*	*Comments*
Trigeminal neuralgia	Usually 2nd and/or 3rd division	<1 second to minutes	Yes	Variable, mild	Good	Trigger areas, mechanical trigger factors
Glossopharyngeal neuralgia	Pharynx, ear	<1 second to minutes	Yes	None	Good	Pain provoked by swallowing, syncope attacks
Nervus intermedius neuralgia	Deep in ear	Seconds or minutes	Yes Also aching pain	None	Good	Rare, may be confused with glossopharyngeal neuralgia
SUNCT	Forehead, retrobulbar	5 seconds to several minutes	Yes	Prominent	None	Almost exclusively in women; rare
Cluster headache	Retrobulbar, cheek, chin	20 minutes to hours	Only superimposed on deep dull pain	Prominent	Modest	Triptans help Alcohol provokes
CPH	Forehead, retrobulbar	2 to 45 minutes	No	Prominent	None	Responsive to indomethacin
Cracked tooth syndrome	Upper jaw Lower jaw	Seconds	Yes	None	None	Provoked on biting and and chewing
Jabs and jolts syndrome	Anywhere in the head	Seconds	Yes	None	Good	No precipitating factors
Postherpetic neuralgia	Forehead, eye, cheek (rarely)	Continuous	Superimposed on background pain	Variable, mild	Variable, usually modest	History of shingles Tactile allodynia Sensory impairment
Giant cell arteritis	Forehead, neck, temple	Continuous	None	None	None	Jaw claudication

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).

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TABLE 127-2 Summary of Drugs Commonly Used in Trigeminal Neuralgia

Drug (Refs.)	*Initial Dose*	*Maintenance Dose*	*Adverse Effects*
Carbamazepine (12,37,55) (CBZ)	200 mg/day	400–1200 mg/day	Sedation, dizziness, cognitive impairment, headache, GI symptoms, allergic rash, leucopenia, folate deficiency, hyponatremia, several drug interactions, warfarin
Oxcarbazepine (90) (OXC)	300 mg/day	600–1200mg/day	Better tolerated than CBZ, sedation, dizziness, cognitive impairment, hyponatremia, rash
Gabapentin (14)	300 mg/day	600–2400 mg/day	Sedation, memory disturbances, peripheral edema
Phenytoin (6) (PHT)	300 mg/day	200–400 mg/day	Sedation, ataxia, behavioral change, cognitive impairment, lymphadenopathy, osteopenia, acne, gingival hypertrophy, rash, folate deficiency, liver failure, several drug interactions
Lamotrigine (88) (LTG)	25–50 mg/day	200–400 mg/day	Allergic rash (necessitates immediate discontinuation), sedation, dizziness, headache, ataxia, significant interactions with other anticonvulsants
Baclofen (23) (BAC)	10 mg/day	30–80 mg/day	Sedation, ataxia, fatigue, GI symptoms, muscle weakness

GI, gastrointestinal.
Comments: CBZ, 90% will respond initially (within 48 hours) (Sato 2004); PHT, licensed in many countries for trigeminal neuralgia (TN), narrow therapeutic range, IV treatment possible, therapeutic effect lost quickly, no randomized controlled trials; OXC, licensed for TN in some countries, effect similar to CBZ but slightly better tolerated, cross-allergy with CBZ in 25% of cases; GBP, limited data, well tolerated, may work as monotherapy as well as in combination therapy; LTG, combination therapy with CBZ only shown effective, slow dose escalation with fortnightly dose increments; BAC, best suited for combination therapy, do not withdraw quickly (risk of convulsions).

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 lamotrigine 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. Similarly, tocainide in a single cross-over trial of 21 patients had efficacy on par with carbamazepine (44) but has since been withdrawn because of serious side effects. Pimozide showed superiority over carbamazepine in a cross-over study involving 48 patients but the results have never been replicated (42). Tizanidine was less efficacious than carbamazepine in one study (85).

Open-label 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, neurectomy, 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 3 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

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entails stimulation of the retroganglionic fibers corresponding to the painful division(s), and their thermocoagulation. Sterotactic 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 muscle. Following balloon compression and radiofrequency lesioning, 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 published. 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 relief, and most cases recur in a few years; 40 to 50% experience a recurrence at 36 months (45). Also, all these procedures are associated with a risk of troublesome posttreatment dysesthesia that may significantly reduce the patient'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 control 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 undergone a neuroablative procedure. Furthermore, corneal sensory loss and keratitis have been reported and are more common after radiofrequency lesioning than other treatments (45,63). Finally, pain relief following neuroablation 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 purports to address directly the underlying cause of the paroxysmal 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 identifying the offending blood vessel(s). The most common finding is a segment of the superior cerebellar artery compressing the nerve at the root entry zone (30,74). After freeing the offending vessel, the operator places a piece of felt between it and the nerve to ensure a permanent separation. Most patients leave the hospital within a few days.

Similar to other forms of surgery, patients with atypical neuralgia do worse than those with typical TN. Indeed, one study showed that 80% of typical cases experience significant pain relief at 5 years postprocedure as compared to 51% of the atypical group (84). Nevertheless, these results remain superior to the ones quoted after radiofrequency lesioning and stereotactic radiosurgery (7,83).

Elderly patients tolerate MVD well, provided they are in reasonable health and can receive general anesthetics (31). Operative or postoperative death occurs in 0 to 0.6% of cases (3,33,41,51,78). Other surgical complications include cerebellar injury (0.45%), eighth nerve injury (0.8%), and cerebrospinal fluid leak (1.85%) (51).

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. Subsequently, 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, neurodestructive procedures, or MVD. In atypical TN with no evidence 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 procedures 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 vessel, although the increasing use of preoperative MRI likely diminishes this problem.

Glossopharyngeal Neuralgia

IHS code: 13.2
WHO code: G44.847.1
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 distribution 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

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EPIDEMIOLOGY

The age- and gender-adjusted annual incidence rate of glossopharyngeal neuralgia in Rochester, Minnesota, for 1945 through 1984, was 0.8 per 100,000 population (36). Although significantly lower than the incidence of TN, the Rochester incidence is much higher than in any previous study, possibly because mild cases were included. Rarely, in less than 5% of cases of glossopharyngeal neuralgia, the patient has concomitant TN. There are no good data on the incidence or prevalence of symptomatic glossopharyngeal neuralgia.

ANATOMY AND PATHOLOGY

The glossopharyngeal nerve is a mixed nerve that contains motor, somatosensory, visceral sensory, and parasympathetic fibers. It communicates with the sympathetic trunk, vagus, and facial nerves. Although complex and variable, its somatosensory functions can be divided into two components: (1) the auricular or tympanic branch, which contains afferents innervating the mastoid, auricle, and external auditory meatus, and (2) the pharyngeal branch, which supplies the pharyngeal mucosa. There is variable communication between pharyngeal and vagal afferents, and together they provide somatic sensory innervation to the base of the tongue, tonsil, and soft palate. Somatic afferents from both nerves terminate in the spinal trigeminal nucleus. There are a number of connections between somatic and autonomic medullary nuclei, and between visceral afferents of the two nerves. These communications provide the anatomic substrate for syncopal attacks sometimes seen in glossopharyngeal neuralgia.

The list of pathologic conditions affecting the glossopharyngeal nerve and causing secondary neuralgia is fairly similar to that in trigeminal neuralgia (e.g., neoplasms, infections, trauma, MS, and structural abnormalities such as an elongated styloid process) (8). Reports of vascular compression found at operation provide a reasonably strong case for etiologic significance, which Bruyn predicted over 20 years ago (8,62,67,71).

CLINICAL FEATURES

IHS revised criteria (ICHD-II) for glossopharyngeal neuralgia are as follows:

A. Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 minutes and fulfilling criteria B and C.
B. Pain has all of the following characteristics:
  1. Unilateral location
  2. Distribution within the posterior part of the tongue, tonsillar fossa, and pharynx or beneath the angle of the lower jaw and/or the ear
  3. Sharp, stabbing, and severe
  4. Precipitated by swallowing, chewing, talking, coughing, and/or yawning
C. Attacks are stereotyped in the individual patient.
D. There is no clinically evident neurologic deficit.
E. 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 include 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 (62,67).

Operative mortality is up to 5%. Also, early neurologic and other complications (dysphagia, hoarseness of

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voice, and swallowing disturbances) are common, are usually mild, but may not resolve in 10% of all operated cases.

Percutaneous radiofrequency lesioning has been mostly used in symptomatic glossopharyngeal neuralgia with impressive results (25).

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 sensory 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 external meatus and just behind the ear, overlying the mastoid process.

PATHOPHYSIOLOGY

Evidence of the role of the geniculate ganglion and the intermedius nerve in the generation of neuralgic pain is relatively 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 neuralgic pain. Still, based on the original hypothesis, some surgeons have carried out extensive neurodestructive procedures with moderately successful results. It is of note that the published case series describe procedures that almost invariably approached several nerves (intermedius and branches of the glossopharyngeal and vagal nerves) during 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 results (48,68).

CLINICAL FEATURES

IHS revised criteria (ICHD-II) for nervus intermedius neuralgia 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 special investigations.

Many case reports agree with Hunt's original description of combined otalgia (pain in the ear) and deep and vaguely described facial pain (originally named prosopalgia by Hunt). The IHS classification has set out criteria for the neuralgia, which require the presence of both paroxysmal pain and a trigger area in the posterior wall of the auditory canal. Reports conform to these criteria. The clinical 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 management considered a last resort.

OTHER CRANIAL NEURALGIAS

Infection (e.g., herpes zoster) and injury can lead to neuropathic pain, affecting various branches of the trigeminal nerve. The most common of these is undoubtedly postherpetic neuralgia affecting the ophthalmic branch of the trigeminal nerve (13.15.2 [B02.02]). Injury to any terminal 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 maxillary nerve, and lingual branch of the mandibular seem most commonly affected. Wisdom tooth removal is associated 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 continuous, fluctuating, sharp, or burning, with some lancinating pain superimposed. Clinical examination confirms sensory impairment and cutaneous allodynia, with tenderness at the exit foramina and a positive Tinel sign in some. Management is similar to that in neuropathic pain

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conditions in general and includes tricyclics, opioids, gabapentinoids, capsaicin, and lidocaine patch. In intractable cases, trigeminal nerve stimulation or intracranial stimulation may be considered.

On occasion, patients may present with neuralgic 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 eventually evolve into classical TN.

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