

THE LADY WITH “ELECTRICAL” FACIAL PAIN

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Case History

A 38-year-old female, referred by her primary-care doctor, presented to a multidisciplinary pain center complaining of facial pain. The pain was described as a sharp, electrical pain localized to the left face. It was reported as being centered around a tooth in the maxilla that had recently been restored with a composite filling material. The patient, a school teacher, reported the pain as being triggered by talking, eating, as well as when she brushed her teeth. The pain had been present for 6 months and had been partially responsive to carbamazepine at a dose of 400 mg per day. She reported she could no longer teach as the pain prevented her from speaking. She had lost 10 pounds in the 6 months since the pain's inception.

She had been in a long-lasting relationship that she described as happy and fulfilling. She had however noted she was shying away from intimacy, as kissing would aggravate the pain. This was creating issues in the relationship that had not previously been present. The patient was convinced there was a problem with the tooth, despite seeing her general dentist, an endodontist, and an oral surgeon, who were all at a loss for the cause. The oral surgeon said he would extract the tooth if she insisted, although he did not feel it would be beneficial.

Questions on the Case

Please read the questions, try to answer them, and reflect on your answers before reading the author's discussion.

- What is the likely diagnosis?
- What is the relevance of the dental therapy?
- What is the relevance of her age?
- How does one investigate the pain? Is there a need for special tests?

- What are the therapeutic options? How would you approach this short term and what considerations would you give this for the long term?

Case Discussion

The differential diagnosis for facial pain presenting as an intermittent sharp, electrical pain should include dental, neuropathic, and neurovascular etiologies. Musculoskeletal etiologies are not usually sharp and electrical, and especially do not last seconds to minutes. An organ system classification has been used to help delineate facial pain and is summarized in Table 40-1.

Extracranial Pain

Tooth pain is one of the “extracranial” structures and the most common cause of orofacial pain. Most frequently, dental pain is related to dental caries, presenting as a reversible pulpitis. The reversible pulpitis is characterized by poorly localized pain, often sensitive to hot or cold stimuli. The reaction to the noxious stimulus (hot or cold) disappears soon after its removal. Eventually, when the carious lesion invades the pulp, an irreversible pulpitis begins. This is characterized by a lingering reaction to noxious stimuli such as

Table 40-1. Organ System Classification for Orofacial Pain

Organ	Presence	Quality
A. Extracranial	Continuous or intermittent	Dull/variable
B. Intracranial	Continuous	Variable
C. Psychogenic	Variable	Variable
D. Neurovascular	Intermittent	Throbbing
E. Neuropathic	Intermittent	Sharp, shooting, electric
F. Musculoskeletal	Continuous	Burning
	Continuous	Dull, aching

hot or cold. If the microorganisms and inflammatory products invade the area around the root apex (periapical), then this is called a periodontitis and may present with toothache associated with chewing, touch, and percussion sensitivity. Periapical pathology may be observed as an area of increased radiolucency on radiograms. The tooth may have an abnormal response to pulp testing, where applying heat, cold, or electrical stimulus is not perceived. In clinical practice, differentiating reversible and irreversible pulpitis is difficult. In situations where the diagnosis is not obvious, careful observation over days or weeks is recommended. Too often, endodontic therapy is performed when not indicated.

An intermittent pain that is triggered by biting on an offending tooth characterizes a cracked tooth syndrome. Unfortunately, the cracks are often difficult to find and do not show on all radiograph images. This pain is often confused with pulpitis or trigeminal neuralgia (TIC), resulting in frustration and unnecessary treatment. Tomographic imaging 1 mm apart through the tooth's long axis may be beneficial in defining the crack. Further careful clinical examination, including meticulous bite tests on each tooth cusp or staining, may be useful.

In this patient, the pain started following a dental procedure. The tooth in question had been filled with an amalgam (silver) filling and was leaking, requiring the dentist to replace it. No evidence on the pre-procedure radiograph indicated any dental pathology. Specifically, there were no caries, usually seen as a radiolucent area in the tooth; also, there was no periapical pathology, usually seen as a thickening of the radiolucent periodontal space, or a loss of bone around the tooth apex. The procedure, according to the patient, was uneventful and the pain started a few days later. Subsequent radiographs also showed no pathology.

It is important to point out that the periapical radiographic changes may take up to 4 months to develop. The changes seen require the infection to erode the cortical plate. Until this occurs, radiographs may not show any abnormality. Other clinical signs and symptoms therefore are important to review. Percussion is very useful as it helps define the infected tooth. Using the back of a dental mirror or pen-shaped object, and gently tapping the teeth, will elicit a marked increased response if pulpal pathology is present. A cracked tooth may only be identified using bite pressure directed in a particular direction. Using a tongue blade, placed carefully so that bite pressure is transmitted to an individual cusp, and then asking the patient to bite and release, may evoke sharp, electrical pain on release. This is indicative of a cracked tooth. A "tooth slooth" is a specific instrument designed for this function.

On examination, the patient displayed no sensitivity to percussion. She also had a normal response to cold stimulus applied to her teeth, indicating vitality and no pulpal pathology.

Neurovascular Pain

An alternative diagnosis could include neurovascular pains. There are two disorders that may present with intermittent sharp, electrical pain in this category: chronic paroxysmal hemicrania (CPH), and short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT).

PAROXYSMAL HEMICRANIA

There are cases of episodic paroxysmal hemicrania similar to the chronic version, but with periods of remission.

Diagnostic criteria according to the International Headache Society (IHS) are as follows:

- A. At least 20 attacks fulfilling criteria B to E
- B. Attacks of severe unilateral orbital, supraorbital, or temporal pain lasting 2 to 30 minutes
- C. Attack frequency above 5 a day for more than half of the time, although periods with lower frequency may occur
- D. Pain is associated with at least one of the following signs/symptoms on the pain side:
 1. Conjunctival injection and/or lacrimation
 2. Nasal congestion and/or rhinorrhea
 3. Ptosis and/or miosis
 4. Restlessness or agitation
- E. Headache is stopped completely by indomethacin
- F. Not attributed to another disorder

Indomethacin should be used at least in a daily dose of 150 mg orally, or 100 mg by injection, but smaller maintenance doses are often employed.

The pain duration in this patient was seconds to minutes and was not associated with autonomic changes. Classically, CPH lasts longer than seconds to minutes, as described in the case history. CPH is also not a triggered pain condition. The age and gender are certainly similar.

SHORT-LASTING UNILATERAL NEURALGIFORM HEADACHE WITH CONJUNCTIVAL INJECTION AND TEARING

This syndrome is characterized by short-lasting attacks of unilateral pain that are much briefer than seen in any other trigeminal autonomic cephalalgia and most often associated with a prominent lacrimation and redness of the ipsilateral eye. There is no substantial evidence in the literature for episodic SUNCT. The literature suggests that the most common mimic of SUNCT would be a lesion in the posterior fossa, or TIC.

IHS diagnostic criteria for SUNCT are as follows:

- A. At least 20 attacks fulfilling criteria B to E
- B. Attacks of unilateral orbital, temporal stabbing, or throbbing pain lasting from 10 to 120 seconds
- C. Attack frequency from 3 to 200 per day

- D. Pain is associated with conjunctival injection and lacrimation
 E. Not attributed to another disorder

Pharmacologic interventions that may be helpful include the following:

Lidocaine 4 mg/min intravenously
 Carbamazepine 1,200 mg
 Lamotrigine 200 mg
 Topiramate 200 mg
 Gabapentin 2,400 mg

Surgical interventions that may be helpful include microvascular decompression.

Both CPH and SUNCT have been reported to coexist with TIC.

SUNCT is an unlikely diagnosis in this patient as she did not report pain in her eye, nor did she have autonomic features with the pain. It is important however to ask patients about the presence of autonomic features, as this may provide further information to better define the diagnosis.

Neuropathic Pain

Neuropathic pain suggests there has been some tissue or nerve injury. With injury, there is a permanent peripheral nerve and/or central nervous system change. Clinically, neuropathic pain can be divided into continuous and intermittent and may present simultaneously or independently. Table 40-2 is a clinical classification for neuropathic facial pain.

Neuropathic pain presents clinically as an intermittent bright, stimulating, electrical, sharp or burning pain. This is typically seen in TIC, glossopharyngeal neuralgia, nervus intermedius neuralgia, and occipital neuralgia. These intermittent neuralgias are triggerable, usually by non-noxious stimuli. Vascular nerve compression is the proposed etiology. Compression may also be secondary to other structures, including posterior cranial fossa tumors and bony growths (eg, Eagle’s syndrome).

Overview of Trigeminal Neuralgia

According to the 1988 report of the Headache Classification Committee of the IHS, TIC is described as “a painful unilateral affliction of the face, characterized by brief electric shock-like (lancinating) pain 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 brushing the teeth, but may also occur spontaneously. The pain is abrupt in onset and termination may remit for varying periods.” Symptomatic TIC is described as “pain indistinguishable from TIC, caused by a demonstrable structural lesion.”

This lesion is usually a tumor, such as an acoustic neuroma, or may be due to demyelination, as seen in multiple sclerosis. If there is tissue or nerve injury, there may be an ensuing continuous TIC, which is usually referred to as traumatic TIC or trigeminal dysesthesia (TD) or deafferentation.

TIC is usually unilateral and only occurs bilaterally in 4% of subjects. There is no genetic link to the disorder. The average age at onset is between the sixth and seventh decades, with women slightly more affected than men in a ratio of 3:2. The bright, stimulating, electrical shock-like pain perceived is short-lived, lasting seconds to minutes. If not questioned carefully, the patient may report that the pain is continuous, as intermittent attacks may last all day. Additionally, there is often a dull pain associated with TIC, or the sharp volleys come and go continuously. The author believes the persistent aching pain may be secondary to a reflex muscle splinting, and can be controlled with stretching exercise and a vapocoolant spray (myofascial pain). Mechanical maneuvering of the trigeminal sensory system usually triggers TIC pain. The area from which the pain is activated has been described as a trigger zone. Characteristically, trigger zones occur around the supra-orbital, infraorbital foramina, the inner canthus of the eye, lateral to the ala, and over the mental foramen. Trigger zones are also common intraorally. Pain is not elicited from the trigger zone if deep pressure is used, or during a latency period between paroxysms. Anesthetizing the trigger zone with topical or injected local anesthetic agents may terminate the pain for the duration of anesthesia.

The second and third trigeminal nerve divisions are most commonly affected. The first-division cases occur less frequently than 5%. Often, there is ipsilateral reflex facial spasm, hence the term “tic douloureux,” which has been used synonymously with TIC. When present, compression of the trigeminal and facial nerves may be implicated.

It is postulated that TIC may be due to a trigeminal nerve focal demyelination at any point along its course. Exploring the posterior cranial fossa reveals that between 60 and 88% of cases have trigeminal nerve root vascular compression. The compression is present in the posterior cranial fossa as the trigeminal nerve exits the pons. This has

Table 40-2. Neuropathic Orofacial Pain

Intermittent	Trigeminal neuralgia
	Glossopharyngeal neuralgia
	Nervus intermedius neuralgia
	Occipital neuralgia
Continuous	Trigeminal dysesthesia / deafferentation
	Trigeminal dysesthesia / deafferentation – sympathetically maintained

been postulated to set up a centrally mediated disinhibition of pain modulation and/or peripheral repetitive ectopic action potentials. Once there is sensitization, there may be increased afferent fiber activity and enhanced response to tactile stimulation, resulting in trigeminal nucleus interneuron discharge and heightened trigeminothalamic neuron discharge, producing pain. Tumor has been implicated in up to 6% of cases. These include acoustic neurinomas, cholesteatomas, meningiomas, osteomas, and angiomas. Aneurysms and adhesions have also been implicated. Although the pain may be typical of TIC, there are usually additional symptoms or cranial deficits present depending on the tumor size and location.

When patients are in the 20- to 40-year age range and present with TIC, multiple sclerosis (MS) should be ruled out. This patient is relatively young for TIC, and although there are no other signs associated with MS, further work-up should be considered. The weight loss described characteristically occurs in TIC patients as they cannot eat secondary to pain triggered by chewing. However, tumor is also associated with weight loss, and once again should trigger further investigation. It is standard of care to obtain brain imaging, with specific attention to the posterior cranial fossa. Magnetic resonance imaging (MRI) scanning will show the posterior fossa better than computed tomography (CT) scanning.

Ratner and Roberts have proposed that bony cavities found in the alveolar bone may be the cause of TIC, and

that repetitive curettage of these cavities is curative. The presence of the bony cavities is not disputed, but their role as a cause of TIC is highly controversial. The author does not believe they are a cause of TIC.

Diagnostic Studies for Trigeminal Neuralgia

Although there are no definitive studies that confirm the diagnosis of TIC, MRI is considered the study of choice for patients presenting with symptoms that may be caused by TIC. With current equipment, resolution is sufficient to identify a vascular loop in contact with the trigeminal nerve as it exits the pons. Although these loops may be present in 30% of asymptomatic people, when seen in the presence of TIC, it helps to confirm the diagnosis and may help in deciding whether surgical microvascular decompression is an option. The MRI will also confirm the presence or absence of tumor, aneurysm, or MS.

In this case, the MRI showed the vascular loop present only on the side of the pain. This helped explain that there was no dental pathology, rather that the dental procedure may have been the peripheral stimulus that was needed to trigger the system, which was sensitized by the presence of the compressed nerve. It is described that up to 80% of patients report that their TIC symptoms started following a routine dental therapy. In no way should this be construed as the cause. See Figure 40-1, which shows the presence of the blood vessel in contact with the trigeminal nerve.

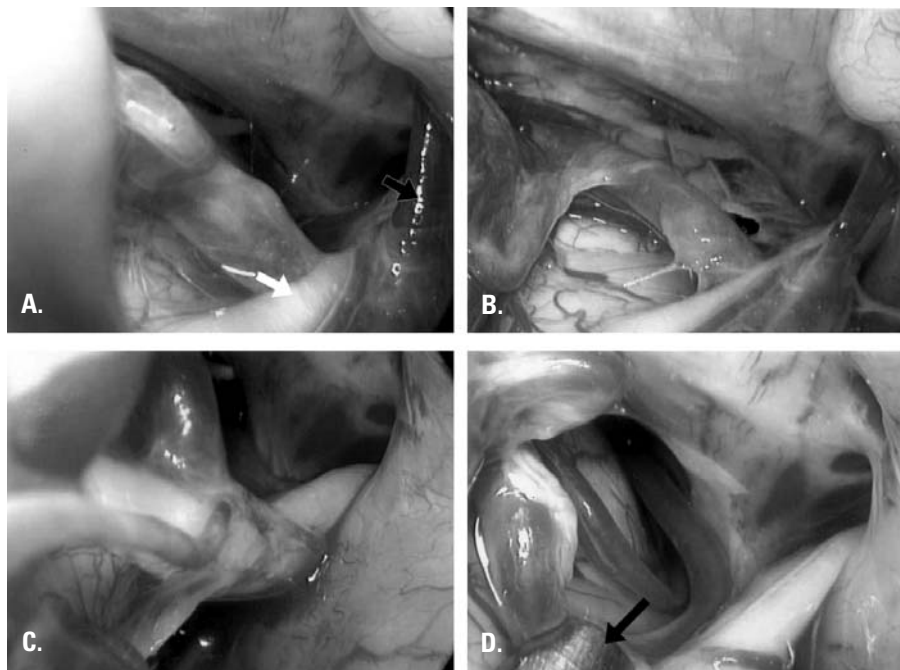


Figure 40-1 A, Blood vessel (*black arrow*) in contact with the trigeminal nerve (*white arrow*). B–D, Removal of this vessel and placing a Teflon cushion (*black arrow*) between the vessel and nerve relieves the pain.

Therapy for Trigeminal Neuralgia

Therapy for TIC is divided into pharmacologic and surgical. Table 40-3 summarizes the pharmacologic agents that may be used, and Table 40-4 presents the surgical therapies.

The anticonvulsant action in pain management is not well understood. Some, like carbamazepine, block use of dependent sodium channels, inhibiting sustained repetitive firing. There is also an effect in the spinal cord, reducing posttetanic synaptic transmission potentiation. There is also decreased synaptic transmission in the trigeminal nucleus, which may explain their effectiveness in facial pain. Valproic acid increases brain concentrations of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter in the central nervous system as well as affecting sodium channels. The action of phenobarbital is not at the trigeminal nucleus but rather in the cortex. It is therefore not effective for TIC. Gabapentin is structurally related to GABA, but does not interact with GABA recep-

Table 40-3. Common Pharmacotherapies Used in Trigeminal Neuralgia Therapy

Generic	Dosage (mg/day)	Blood Level (µg/mL)	Serum Half-Life (h)
Carbamazepine	100–1,200	4–12	12–17
Gabapentin	100–3,500	–	–
Klonopin	0.5–8	–	22–33
Lamotrigine	50–500	2–5	14–59
Lioresal	10–80	–	–
Orap	2–12	–	55–154
Oxcarbazepine	150–2,400	–	8–11
Phenytoin	200–500	10–20	18–24
Topiramate	50–500	–	21
Valproic acid	125–1,500	50–100	6–16

tors or with many of the other receptors involved with pain such as *N*-methyl-D-aspartate, glutamate, dopamine, serotonin, sodium channels, or calcium channels. Topiramate may also relieve pain through blockage of

Table 40-4. Surgical Management of Trigeminal Neuralgia

Procedure	Comment
Alcohol block	Relief is typically 8–16 months
Alcohol gangliolysis	Paresthesia or dysesthesia occurs in 48% Corneal anesthesia occurs in 15% Neuroparalytic keratitis occurs in 4–7% Postoperative paresthesia occurs in 55% Paresthesia occurs in 38% Herpetic outbreak occurs in 26%
Neurectomy	Transient masticatory muscle weakness occurs in 45% Relief is typically 26–38 months
Glycerol gangliolysis	Anesthesia dolorosa and corneal anesthesia are rare 7–10% have early recurrence 7–21% develop recurrence over extended follow-up Facial hyperesthesia occurs in 24–80% Corneal anesthesia occurs in 9% Facial dysesthesia occurs in 8–29%
Radiofrequency gangliolysis	1–17% have early recurrence 4–32% develop recurrence over extended follow-up Masseter weakness occurs in 7–23% Trigeminal dysesthesia occurs in 11–42% Corneal hyperesthesia occurs in 3–27% Neuroparalytic keratitis occurs in 1–5% Anesthesia dolorosa occurs in 1–4%
Microvascular decompression	16–29% develop recurrence over extended follow-up Mortality occurs in 1% Morbidity occurs in 10–23%
Rhizotomy	15% develop recurrence over extended follow-up Mortality occurs in 0.5–1.6% Facial weakness occurs in 7–8% Paresthesia occurs as a minor complaint in 56% Paresthesia occurs as a major complaint in 5% Neuroparalytic keratitis occurs in 15%
Trigeminal tractotomy	Ipsilateral limb ataxia occurs in 10% Contralateral limb sensory loss occurs in 14%
Gamma knife	Onset may be 6 weeks or longer Facial numbness Trigeminal dysesthesia

sodium channels, enhancing GABA activity, and blocking glutamate activity. Lamotrigine may inhibit release of glutamate, and inhibits voltage-sensitive sodium channels.

All drugs used in TIC have side effects and great care must be used in their administration. Baclofen has perhaps the least side effects, but may also be less effective than carbamazepine or oxcarbazepine. Baclofen is initiated at 10 mg per day and increased every 2 days to a maximum of 80 mg per day, usually in four doses. Drowsiness and confusion are the major side effects and many patients will not tolerate it for this reason. Carbamazepine is without doubt the most effective drug. It is suggested that one begin at 100 mg per day and increase by 100 mg every 2 days to a maximum of 1,200 mg. The side effect of aplastic anemia, although rare, needs to be monitored carefully with routine blood tests. Should there be no relief, a carbamazepine blood level should be obtained to ensure a therapeutic blood concentration. A trough level should be obtained and is usually in the range of 5 to 10 µg/mL. The sustained preparations have improved compliance and reduced the sedating side effects. Oxcarbazepine is as effective and has less side effects, requiring less frequent blood monitoring. Gabapentin, although not formally studied, has been useful in doses from 300 mg per day to 3,000 mg per day. Topiramate is also useful and doses are initiated at 15 mg at bedtime, and increased to between 100 and 300 mg per day. Initially, the author uses up to 100 mg only at bedtime, which may be effective. Phenytoin is a good alternative in doses ranging from 100 to 400 mg per day. Using drug combinations may be necessary to either maximize effect or minimize side effects. Pimozide has been described as being more effective than carbamazepine in a double-blind, crossover design using 48 subjects with TIC refractory to medical treatment. Side effects were reported in 83% of subjects who received pimozide, and included physical and mental retardation, hand tremors, memory impairment, involuntary movements during sleep, and slight Parkinson's disease manifestations. None of the patients stopped treatment because of side effects. Therefore, it is felt by the study authors that it is effective, but because of the adverse effects, it should be reserved for severe and intractable TIC.

One of the biggest problems seen in the use of pharmacotherapy is inadequate dose used. The patient was prescribed carbamazepine at a dose of 400 mg per day. She had some relief, but was never instructed to increase the dose, nor was she informed that it should be dosed three times per day because of the relatively short half-life of the drug. She was not given the option of using a longer acting brand, which also may have been beneficial.

It is suggested that pharmacologic intervention be exhausted prior to embarking on a surgical therapy. It has been reported however that over a 10-year period, patients

with TIC are likely to obtain surgery because they do not like the medication side effects, they are no longer controlled, or they want a "cure." Patients must be made aware that the pharmacotherapies only serve to control the pain, but do not eliminate the cause. The primary purpose is to try and initiate a remission period. Once pain free for a month, the author usually reduces the medication, with instructions to resume therapy if the pain returns.

Surgical Therapies for Trigeminal Neuralgia

The surgical treatments for TIC are summarized in Table 40-4. Less traditional treatments reported for TIC include the curettage of the bony cavities described above. Ratner and Roberts report a long-term success of 80%. This therapy is controversial and not recommended by the author. Sokolovic and colleagues have described the use of peripheral streptomycin (1 g) and lidocaine (2%) injections around the peripheral nerve. Bittar and Graff-Radford completed a double-blind, placebo-controlled, cross-over study using streptomycin, and the results were not favorable.

The most effective surgical approach remains microvascular decompression. Advances in microvascular decompression include the use of an endoscopic approach. This allows clearer observation and is less traumatic. Gamma knife radiosurgery is a recent advance for TIC. This technique offers a relatively noninvasive means for lesioning the trigeminal nerve adjacent to the pons using a 4 mm collimator helmet. Complications are rare, and to date the author has seen one case of TD attributed to the procedure.

Pre-trigeminal Neuralgia

Sir Charles Symonds first described pre-trigeminal neuralgia (pre-TIC). Fromm and Sessel have described a further 16 cases in which patients initially present with a dull continuous aching toothache in the upper or lower jaw, and in whom the pain changed to classic TIC. Further, they describe 7 cases in which the continuous pain was successfully treated with traditional TIC therapies. The diagnosis of pre-TIC is based on the following criteria: 1) description of pain as dull toothache, 2) normal neurologic and dental examinations, and 3) normal CT or MRI scan of the head.

Conclusion

This patient presented with facial pain that was classic of TIC. She had not received an adequate work-up or therapy. By taking a history with specific attention to the pain location, clinical character, triggerability, lack of autonomic features, along with the examination findings of a normal cranial nerve evaluation and no local dental pathology,

TIC could be diagnosed. This did not however exclude the potential for an intracranial lesion or MS. Because of her age, imaging was necessary. The MRI demonstrated the presence of a compressing vessel in contiguity with the trigeminal nerve on the affected side, further confirming the diagnosis.

Treatment was initiated with carbamazepine as she was already using this agent with some success. It was slowly titrated to 1,000 mg per day, which was administered in three doses. A slightly higher dose was given prior to bedtime to help sustain the effect through the night. Classically, patients with TIC do not wake out of their sleep, as being in a recumbent position is beneficial. However, they often have severe pain in the morning, requiring loading the dose at night or using a long-acting preparation. At 1,000 mg of carbamazepine per day, she was pain free. She was initially overjoyed and reported, “I have my life back.” This was relatively short-lived, as after a few months, she felt that the cognitive effects of carbamazepine were interfering with her teaching abilities. She was feeling fatigued and requested an alternative. We discussed at length other medication trials, but she ultimately chose surgery.

After evaluation by the surgeon, she underwent a microvascular decompression via an endoscopic approach. A dime-size bone window was made behind her ear, and via the endoscope the offending vessel was located and a Teflon pledget was placed between the vessel and nerve (see Figure 40-1). The patient spent one night in the intensive-care unit and then 1 day in the hospital, and was discharged pain free. She did complain of dizziness postoperatively and was placed on steroid for 4 weeks; this resolved. Immediately postoperatively, the carbamazepine was cut to 50% and slowly she was weaned off it, and 1 year later remains pain free and off medication.

TIC is a very well-treated disorder, and once appropriately diagnosed, aggressive therapy pharmacologically and/or surgically should be offered. For patients suffering continuous facial pain following neural trauma, TD or deafferentation should be diagnosed. The therapy in this group is different and discussed elsewhere by Graff-Radford, and surgical interventions should be avoided.

Selected Readings

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Editorial Comments

The history of treatment of TIC is a most interesting one, and one that has resulted in highly effective therapies for the majority of patients. Experience with the Janetta procedure has made surgical intervention a definitive therapy in selected patients, in which neuroimaging has demonstrated a cross compression of the trigeminal nerve with vascular loops. Nevertheless, a lot of patients can be adequately managed with medication alone or other interventions, as outlined. This is a very detailed and erudite overview of the subject, but again, it is history and physical examination of the patient that ultimately leads to the correct diagnosis and treatment. There are many useful clinical clues in this case and its discussion, and it is hoped that this chapter will be of benefit to all readers of this volume.

FINAL DIAGNOSIS:

Trigeminal neuralgia

